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Hemodynamic Analysis of Patient-Specific Abdominal Aortic Aneurysm: A CFD Study

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Abstract

An abdominal aortic aneurysm (AAA) is a localized enlargement of the abdominal aorta that poses significant health risks, particularly if ruptured. The rupture of an AAA can lead to severe internal bleeding and is often fatal if left without an immediate medical intervention. Understanding the hemodynamics within an AAA is essential, as blood flow patterns, pressure distribution, and wall shear stress play a vital role in disease progression and rupture risk. By utilizing patient-specific geometry, this study explores how an elevated level of hematocrit (the proportion of red blood cells (RBC) in the blood) impacts AAAs. We used computational fluid dynamics (CFD) along with elastic wall motion simulations to analyze blood flow, pressure, and wall stress at both normal and above-normal red blood cell measurements. Results are analyzed for a single cardiac cycle and findings indicate the importance of inclusion of RBC counts for more patient-specific analysis. Higher hematocrit levels lead to increased wall shear stress (WSS) and pressure changes within the aneurysm, which could raise the risk of thrombosis and rupture. Although blood flow patterns remained similar across different hematocrit levels, the significant changes in WSS emphasize the role of blood viscosity in aneurysm behavior. Overall, this study highlights the importance of numerical tools in assessing AAA status and provides a basis for better clinical assessments and personalized treatments.

Keywords: FSI, Computational Fluid Dynamics, Hemodynamics, Aneurysm.

1. Introduction

Abdominal aortic aneurysm (AAA) represents a critical health challenge, marked by the gradual weakening and dilation of the abdominal aorta, posing a substantial risk of rupture with potentially fatal consequences. The management of AAA must prioritize the evaluation of rupture risk to prevent severe complications. Computational Fluid Dynamics (CFD) has emerged as a valuable tool for modeling blood flow within AAAs, providing insights into key hemodynamic parameters, such as wall shear stress (WSS) and pressure distribution, which are essential for assessing rupture risk.

In this study, we explore the hemodynamic properties of AAA using patient-specific geometries. By considering pulsatile blood flow, non-Newtonian behavior of blood, this research provides a comprehensive understanding of the biomechanical environment within the aneurysm. Specifically, the study investigates the influence of varying hematocrit levels, which affect blood viscosity, on hemodynamic factors such as WSS, pressure distribution.

Various approaches have been employed to study AAA hemodynamics. Early research relied on physical models, including pressurized rubber tubes [1] and silicone phantoms [2] to simulate aneurysm behavior. However, the advent of CFD has revolutionized the field by offering a non-invasive means to study complex hemodynamics within the aneurysm sac. These models allow for patient-specific simulations based on medical imaging data, improving the accuracy of rupture risk assessment [3].

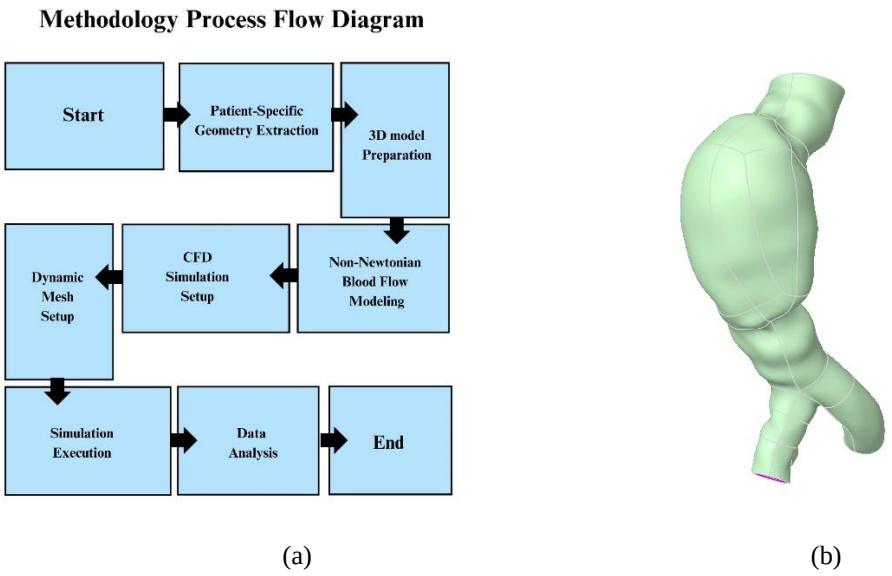
Di Martino et al. [4] conducted one of the early studies on fluid-structure interaction in patient-specific models of aneurysms, using 3D geometries derived from medical images to analyze blood flow and wall dynamics. Accurate boundary conditions, such as pulsatile blood flow profiles, are critical for ensuring the precision of CFD models. Józsa and Paál [5] highlighted the importance of using realistic boundary conditions in CFD simulations to accurately represent blood flow interactions with the aneurysm wall.

The role of WSS in predicting aneurysm rupture has been extensively studied. Fillinger et al. [6] demonstrated that regions of elevated WSS are linked to increased rupture risk, as higher mechanical stress is experienced by the aneurysm wall. Studies also emphasize the significance of incorporating pulsatile blood flow into CFD models. Taylor and Yamaguchi [3] were among the first to compare steady and unsteady flow in aneurysm studies, showing that pulsatile flow provides a more accurate representation of aneurysm hemodynamics and the potential for rupture.

Despite these advancements, challenges remain in modeling arterial wall mechanics and accounting for the non-Newtonian behavior of blood, especially at varying hematocrit levels. Most CFD models treat blood as a Newtonian fluid, but recent studies emphasize the need to incorporate its shear-thinning, non-Newtonian properties [7] [8]. In this study, we explore the hemodynamic properties of AAA using patient-specific geometries and fluid-structure interaction (FSI) simulations. By considering pulsatile blood flow, non-Newtonian behavior of blood, this research provides a comprehensive understanding of the biomechanical environment within the aneurysm. Specifically, the study investigates the influence of varying hematocrit levels, which affect blood viscosity, on hemodynamic factors such as WSS, pressure distribution, and wall deformation.

2. Methodology

The methodology (Fig. 1 (b)) employed in this study integrates CFD simulations to analyze the hemodynamic properties of a patient-specific AAA (Fig. 1 (b)). The process begins with the geometric modeling of the aneurysm, where patient-specific AAA geometry is extracted from medical imaging data. The 3D geometry is processed using segmentation techniques to ensure an accurate representation of the aneurysm sac and arterial walls, followed by further preparation in Blender and ANSYS SpaceClaim. A structured mesh is created to ensure high accuracy in the simulation, particularly in critical regions such as the aneurysm wall and zones of flow recirculation. Mesh independence tests are conducted to confirm that the simulation results are not influenced by the mesh size.



(a) (b)

Fig. 1. (a) Flow diagram of Methodology (b)AAA Geometry

In the CFD model, blood is treated as a non-Newtonian fluid, with the Quemada viscosity model applied to account for the shear-thinning behavior of blood, particularly under different hematocrit levels. The model expressed below,

$$\eta = \eta_p \left(1 - \frac{k.H_{tc}}{2}\right)^{-2} \quad (1)$$

$$k = \frac{k_0 + k_\infty \left(\frac{\gamma}{\gamma_c}\right)^{1/2}}{1 + \left(\frac{\gamma}{\gamma_c}\right)^{1/2}} \quad (2)$$

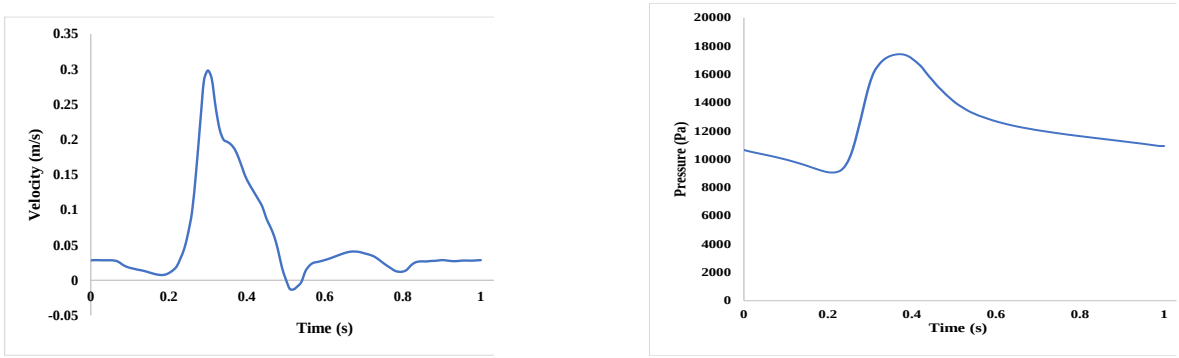
Here, η_p= Plasma viscosity, k_0, k_∞=Lower and upper limit of shear viscosity H_{tc}= Hematocrit number.

The CFD simulations are carried out using ANSYS Fluent, the governing equations to describe the fluid flow within AAA will be the continuity equation and the Navier-Stokes equation (NSE). These equations expressed below,

$$\nabla u = 0 \tag{3}$$

$$\frac{\partial u_i}{\partial t} + \nabla.(u_i u) = \nabla.(v\nabla u_i) - \frac{1}{\rho}\frac{\partial P}{\partial x_i} + b_i \tag{4}$$

where $\nabla \cdot u$ is the divergence of the velocity field u, indicating the net flow of fluid into or out of a specific point, t denotes time, ν is the kinematic viscosity, ρ is the blood density assumed 1060 kg/m3, P is the pressure, and b_i represents any external forces acting on the fluid, such as gravity or body forces assumed 0 N.
The boundary conditions at the inlet are defined by a pulsatile waveform (Fig. 2(a)) that replicates the physiological nature of blood flow, while a time-dependent pressure waveform is applied at the outlet to simulate systolic and diastolic blood pressure variations (Fig. 2(b)), ranging between 80 and 120 mmHg.



(a) (b)

Fig. 2. (a) Inlet velocity profile (b) Outlet velocity profile

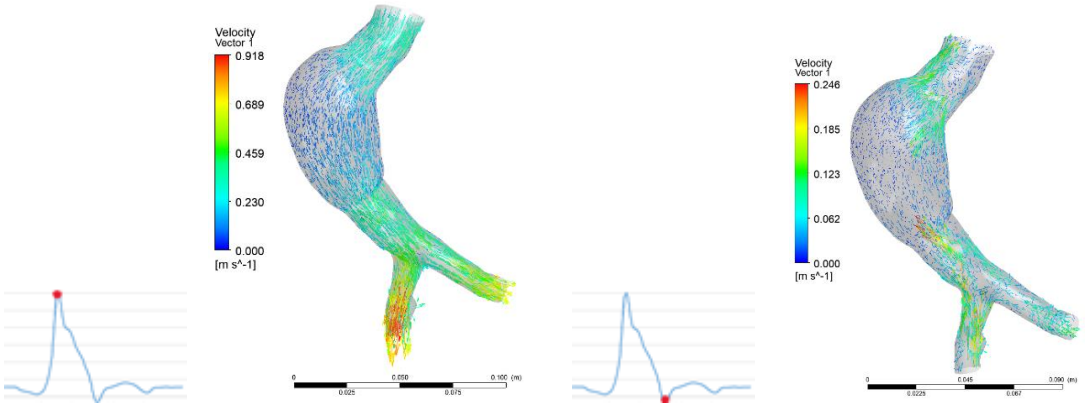
In this study, the CFD simulations were performed using ANSYS Fluent 2023 Academic Version. The aneurysm model was meshed with approximately 6 million elements to ensure high accuracy in capturing the flow dynamics within the abdominal aorta and the aneurysm sac. A dynamic mesh was enabled to account for the deforming nature of the aneurysm wall, ensuring that the mesh adjusts smoothly throughout the simulation. The mesh smoothing algorithm was applied to maintain the quality of the mesh as the simulation progresses. The interior volume of the blood within the aneurysm was treated as a deforming fluid domain, while the aneurysm wall was modeled as a no-slip wall, meaning that the velocity at the wall was set to zero, simulating real-life blood-wall interactions. Additionally, system coupling was applied to the wall boundary to ensure the accurate transfer of forces between the fluid and the arterial structure. The simulation was run for 1 second of real-time, representing one full cardiac cycle, and capturing the pulsatile nature of blood flow.

3. Results and Discussion

3.1 Flow Patterns

The flow patterns within the AAA were analyzed during both systolic and diastolic phases. During systole, which is when the heart contracts and pushes blood through the arteries, the velocity vectors (Fig. 3(a)) indicate a high-velocity jet entering the aneurysm sac. The flow exhibits significant complexity, with large regions of flow recirculation and vortex formation near the walls of the aneurysm. The velocity magnitude reaches as high as 0.945 m/s, with a prominent forward-directed flow exiting the aneurysm. This is characteristic of the high-energy state of blood flow during peak systolic pressure. In diastole, when the heart relaxes and blood pressure drops, the flow dynamics change (Fig. 3(b)). The velocity magnitude significantly reduces, with maximum values only reaching about 0.236 m/s. Recirculation zones become more prominent as the flow stabilizes, and some reverse

flow is observed within the aneurysm sac. These low-velocity regions are important to analyze since they are often linked to thrombus formation and the development of complications within aneurysms.

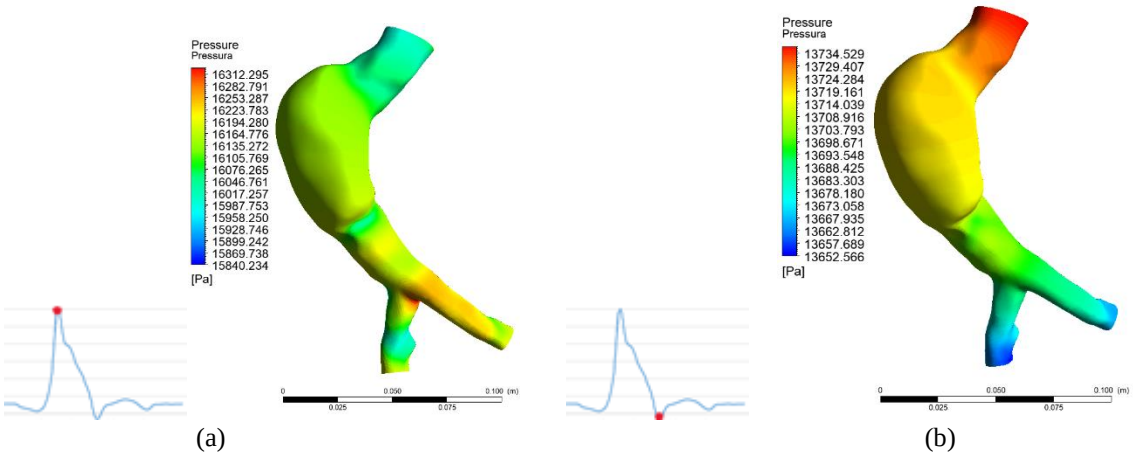


(a) (b)

Fig. 3. (a) Flow pattern at peak systole (b) Flow pattern at early diastole

3.2 Pressure Distribution

The pressure distribution during diastole is illustrated in (Fig. 4 (b)). The values ranged from 13,652 Pa to 13,735 Pa, with the highest pressures occurring at the proximal region of the aneurysm, close to the aortic inflow. Pressure values decreased progressively towards the distal region near the iliac bifurcation. The distribution within the aneurysm was relatively uniform, though some localized areas of increased pressure were observed, particularly in the aneurysm bulge. This pressure gradient aligns with physiological expectations, reflecting reduced hemodynamic forces during diastole when the heart is relaxed and blood fills the ventricles. Conversely, during systole, as shown in (Fig. 4(a)), the pressure range increased substantially, spanning from 15,840 Pa to 16,312 Pa. High pressure values were again concentrated near the proximal neck of the aneurysm, with a more pronounced gradient extending through the aneurysm to the bifurcation. This pressure increase corresponds to the peak forces exerted by the contracting heart, which accelerates blood flow into the aneurysm. The systolic pressure map revealed more pronounced elevated pressure areas within the aneurysm bulge compared to diastole, indicating zones of heightened mechanical stress. These areas are more vulnerable to potential rupture or further dilation over time.



(a) (b)

Fig. 4. Pressure distribution (a) at peak systole (b) at early diastole

3.3 Wall Shear Stress

WSS, the frictional force exerted by blood flow on the arterial wall, emerges as a critical determinant of AAA pathogenesis. WSS is intricately influenced by factors such as blood viscosity, flow velocity, and arterial

geometry. The analysis of WSS across different hematocrit levels (30%, 40%, and 60%) during systole and diastole revealed distinct patterns (Fig. 5). In systole, maximum WSS values were measured at 16.71 Pa, 19.1 Pa, and 25.77 Pa for hematocrit levels of 30%, 40%, and 60%, respectively. During diastole, maximum WSS values were lower, recorded at 2.45 Pa, 2.65 Pa, and 7.4 Pa for the respective hematocrit levels. These findings indicate that higher hematocrit levels are associated with increased WSS, reflecting the influence of blood viscosity. Additionally, lower WSS values were consistently observed in the aneurysm area, attributed to reduced flow velocity and the larger diameter characteristic of aneurysms. This highlights the significance of hematocrit levels in modulating WSS distribution within the AAAand underscores the impact of flow dynamics on aneurysm hemodynamics.

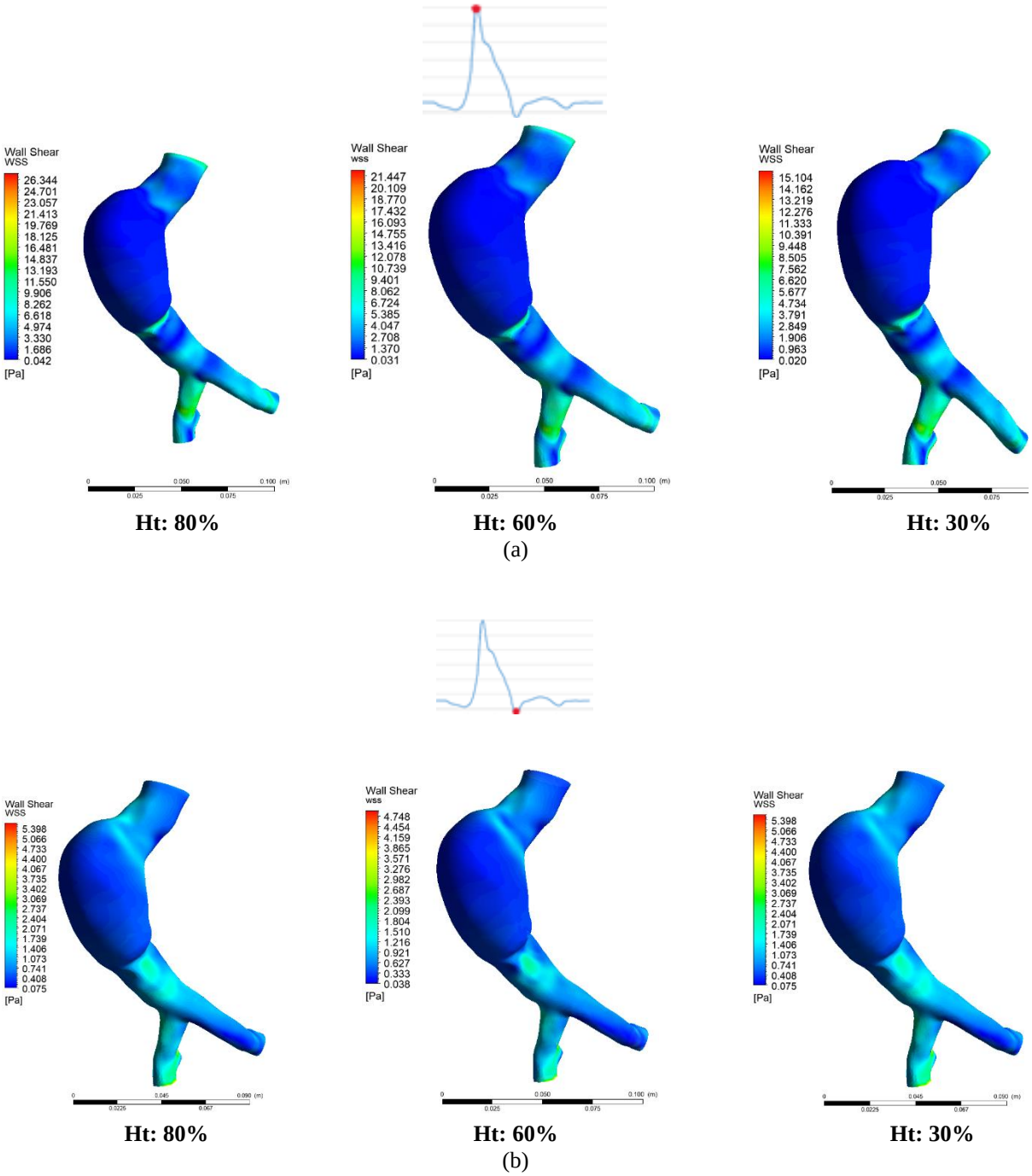


Fig. 5. Wall shear stress (a) at peak systole (b) at early diastole for different hematocrit level

3.4 The area-averaged wall shear stress

The area-averaged wall shear stress (AAWSS) within AAA plays a critical role in understanding the mechanical forces exerted on the aneurysm wall. During systole, the AAWSS values are significantly elevated due to the higher blood flow velocity, with maximum values of 4.68 Pa, 3.53 Pa, and 2.44 Pa for hematocrit levels of 80%, 60%, and 30%, respectively (Fig. 6). These results demonstrate that higher hematocrit levels, which correspond to increased blood viscosity, result in greater wall shear stresses. The higher AAWSS at 80% Ht suggests more intense mechanical forces on the aneurysm wall, which may contribute to the aneurysm's progression. In contrast, during diastole, when blood flow slows, the AAWSS values drop substantially to 0.42 Pa, 0.27 Pa, and 0.19 Pa for the same hematocrit levels. The significant reduction in wall shear stress during diastole highlights the cyclical nature of the hemodynamic forces, with the wall experiencing much lower stress in this phase. However, even in diastole, the higher hematocrit values still result in slightly elevated AAWSS compared to lower hematocrit levels emphasizing the continuous influence of blood viscosity on wall shear stress throughout the cardiac cycle. These findings emphasize the impact of hematocrit on the biomechanical environment in an aneurysm, potentially affecting the risk of aneurysm expansion or rupture.

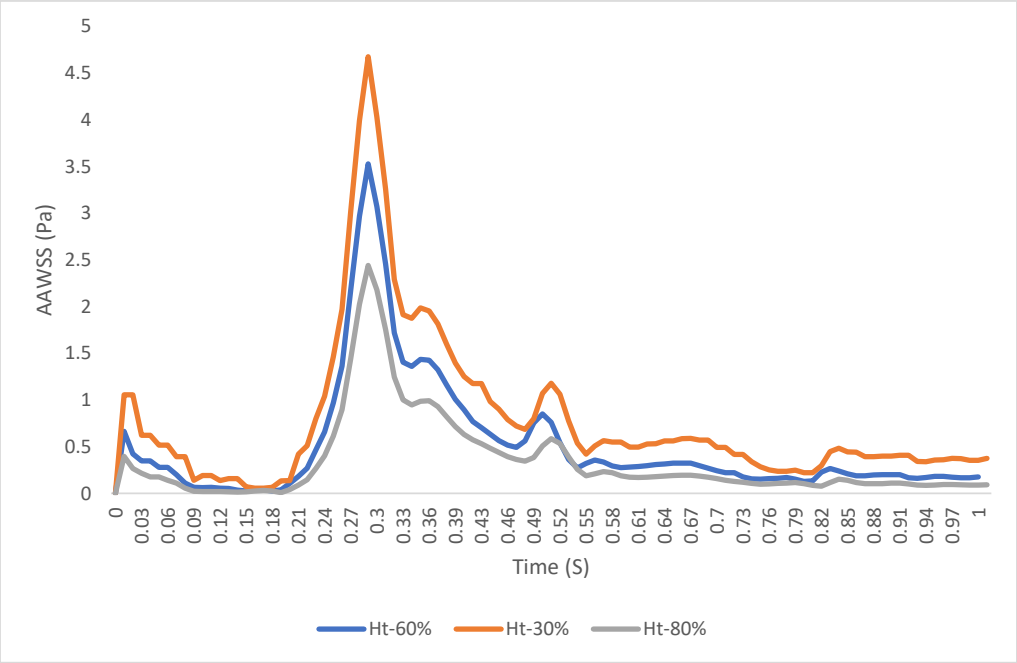


Fig. 6. Area-averaged wall shear stress

4. Conclusion

In conclusion, this study demonstrates that while WSS is significantly influenced by varying hematocrit levels, the overall flow patterns and pressure distribution within the AAA remain largely unaffected. Using patient-specific geometry and CFD simulations, we observed that changes in hematocrit primarily impact WSS, with higher red blood cell concentrations leading to increased mechanical stress on the aneurysm wall. However, the flow dynamics and pressure gradients within the aneurysm showed minimal variation across different hematocrit levels. This suggests that while hematocrit plays a crucial role in affecting shear stress, it has less influence on bulk flow behavior and pressure. These findings underline the importance of considering hematocrit in aneurysm risk assessment, particularly in relation to wall stress, while confirming that flow patterns are more dependent on structural features of the aneurysm than on blood composition. However, this study has several limitations. The simplified modeling of blood as a non-Newtonian fluid does not fully account for the complexities of RBC aggregation and deformation. Additionally, the focus on a single patient-specific model limits the generalizability of the results to a broader population. Future experiments could address these limitations by incorporating more sophisticated blood rheology models and expanding the dataset to include a variety of patient-specific geometries.

11. References

[1] Note on the instability of India-rubber tubes and balloons when distended by fluid pressure. (1891, December 31). Proceedings of the Royal Society of London, 49(296–301), 458–463.

[2] Kyriakides, S., & Yu-Chung, C. (1990). On the inflation of a long elastic tube in the presence of axial load. International Journal of Solids and Structures, 26(9–10), 975–991.

[3] Taylor, T. W., & Yamaguchi, T. (1994, February 1). Three-Dimensional Simulation of Blood Flow in an Abdominal Aortic Aneurysm—Steady and Unsteady Flow Cases. Journal of Biomechanical Engineering, 116(1), 89–97.

[4] Di Martino, E., Guadagni, G., Fumero, A., Ballerini, G., Spirito, R., Biglioli, P., & Redaelli, A. (2001, November). Fluid–structure interaction within realistic three-dimensional models of the aneurysmatic aorta as a guidance to assess the risk of rupture of the aneurysm. Medical Engineering & Physics, 23(9), 647–655.

[5] Józsa, T. I., & Paál, G. (2014, December). Boundary conditions for flow simulations of abdominal aortic aneurysms. International Journal of Heat and Fluid Flow, 50, 342–351.

[6] Fillinger, M. F., Marra, S. P., Raghavan, M., & Kennedy, F. E. (2003, April). Prediction of rupture risk in abdominal aortic aneurysm during observation: Wall stress versus diameter.

[7] Alhayani, A., Giraldo, J., Rodríguez, J., & Merodio, J. (2013, October). Computational modelling of bulging of inflated cylindrical shells applicable to aneurysm formation and propagation in arterial wall tissue. Finite Elements in Analysis and Design, 73, 20–29.

[8] Souche, A., & Valen-Sendstad, K. (2022, December). High-fidelity fluid structure interaction simulations of turbulent-like aneurysm flows reveals high-frequency narrowband wall vibrations: A stimulus of mechanobiological relevance? Journal of Biomechanics, 145, 111369.