

Numerical Simulations of Blood Flow in the Left Atrial Appendage: Investigating the Influence of Anatomical Shapes

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Abstract

Atrial fibrillation (AF) is a prevalent type of arrhythmia affecting a significant portion of the population and is the leading cause of an ischemic stroke. This study employs advanced computational modeling and simulation techniques to investigate blood flow dynamics within the left atrium and the left atrial appendage (LAA) in AF patients. Patient-specific anatomical model was created using Mimics and 3-matic software, enabling precise segmentation from anonymized CT images. COMSOL Multiphysics was used to simulate blood flow patterns. Our results reveal dynamic flow velocity variations throughout the cardiac cycle, emphasizing the complexity of blood circulation within the left atrium. Notably, regions within the LAA with reduced flow velocities (below 1 cm/s) were identified, promoting formation of clots. These findings have direct clinical implications such as guiding decisions related to anticoagulant therapy and refining interventions for stroke prevention in AF patients. This research aims to provide tools for reduction of the incidence of clot-related complications in AF patients by enhancing our understanding of cardiovascular health risks.

Keywords: CFD simulation; left heart, LAA, blood circulation.

Introduction

Cardiovascular diseases, in particular atrial fibrillation (AF), continue to pose a significant health burden worldwide. Atrial fibrillation (AF) is one of the most common types of human arrhythmias. AF is affecting approximately 3 % of adult population and almost 6 % of people over age of 65 [1]. Atrial fibrillation, characterized by irregular and often rapid heart rhythms, is a leading cause of stroke, responsible for up to 20 % of all ischemic strokes. One of the critical anatomical parts in the heart associated with stroke risk in AF patients is the left atrial appendage (LAA). This small, pouch-like structure within the left atrium has been implicated in the formation of blood clots, which can subsequently lead to embolic events, including strokes.

The development of blood clots within the LAA is closely linked to the blood flow dynamics within this region of the heart. Patients with low blood flow in the LAA may have an elevated risk of blood clot formation. However, the relationship between blood flow patterns and clot risk remains complex and not yet fully understood. This knowledge gap necessitates advanced computational modelling and simulation techniques to gain insights into the hemodynamic behaviour of blood within the LAA.

In our research, we created patient-specific anatomical models of the left atrium (LA) and utilize COMSOL Multiphysics for blood flow modelling. Our primary objective is to simulate and investigate blood flow patterns within these personalized LA model, with the aim of comprehensively assessing and monitoring individualized risk profiles influenced by anatomical differences. These variations in the LA's structure can significantly impact blood flow dynamics, potentially leading to

issues such as blood clot formation and other cardiac complications. Some of the first numerical studies were performed in [1]-[3]. By harnessing the advanced capabilities of COMSOL Multiphysics, we can tailor our simulations to the unique anatomical features of each patient's LA. This personalized approach holds immense promise in enhancing our understanding of how anatomical differences contribute to LA-related health risks, enabling healthcare professionals to make more informed clinical decisions and develop targeted intervention strategies. Ultimately, our research strives to improve patient care by advancing our knowledge of patient-specific cardiac anatomy and its implications for cardiovascular health.

Methods

3D anatomical model preparation

To conduct the simulations, we employed a multi-step approach. First, we utilized the Mimics and 3-matic software (Materialise, Belgium) to perform left heart segmentation from anonymized computed tomography (CT) images, enabling the extraction of accurate anatomical models. Our procedure commenced with thresholding, where we employed pixel intensity values to precisely isolate the LA from the surrounding anatomical structures within the CT scans. Following this, we focused on model refining, ensuring the seamless removal of any irregularities or imperfections in the segmented LA model to enhance its anatomical accuracy. Subsequently, we continued with remeshing and optimizing the mesh quality of the model to prepare it for further computational simulations. Ultimately, our efforts culminated in the export of the segmented LA model to the STL format, providing a high-fidelity representation that serves as the cornerstone

for our advanced investigations into blood flow dynamics and the assessment of individualized cardiac risk profiles. This meticulous segmentation process, spanning thresholding to STL export, significantly improves the precision and reliability of our patient-specific cardiac modelling and simulation endeavours. These models were then prepared for numerical simulations by creating a high-quality surface mesh, which was subsequently imported into the COMSOL Multiphysics software.

COMSOL model import and settings

Within COMSOL Multiphysics, the mesh was initially imported as a mesh part and subjected to meticulous editing to guarantee its integrity and rectify any potential errors. Subsequently, the mesh underwent a transformation into a geometry representation. To establish the input profile, the inlet and outlet openings were further extruded by 4 cm each.

By incorporating turbulent flow physics, specifically the Reynolds-Averaged Navier-Stokes (RANS) equations and the Shear Stress Transport (SST) turbulence model, along with transition modelling, we aim to enhance the accuracy and realism of our simulations. The generalized equation for blood flow in the left atrium using RANS and SST can be defined as following equation:

$$\nabla(\rho u) + \nabla(\rho u \times u) = -\nabla p + \nabla(\mu_{eff} \cdot \nabla u) + \rho \cdot g + S \quad (1)$$

Where ρ is the density of blood, u is the velocity vector of blood, p is the pressure, μ_{eff} is the effective dynamic viscosity, g represents gravitational forces and S represents source terms.

The inlets were set to the three pulmonary veins, which are crucial for blood supply to the LA and subsequently to LAA as can be seen in the Figure 1. The inlets were configured with time-dependent pressure conditions (Figure 2), while the outlet was set to a time-dependent velocity condition (Figure 3), to accurately simulate the physiological behaviour of blood flow throughout one cycle lasting 0.8 seconds.

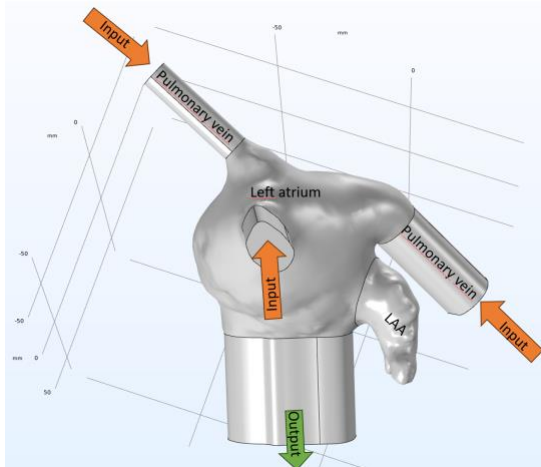


Figure 1. Left atrium patient specific model with marked input pulmonary veins.

The blood pressure and velocity were taken from [1]. The left atrium model comprises a single domain, which represents the blood. The dynamic viscosity was set to 0.035 Pa·s and the blood density was set to 1060 kg·m⁻³.

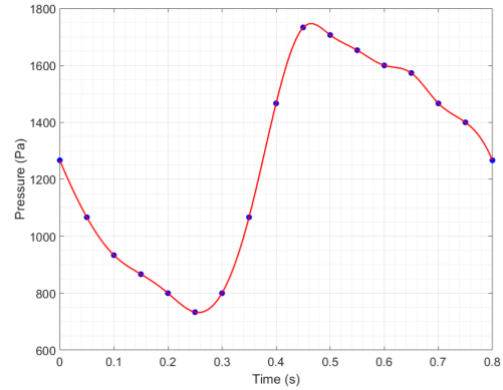


Figure 2. Time-dependent input blood pressure set on the pulmonary veins.

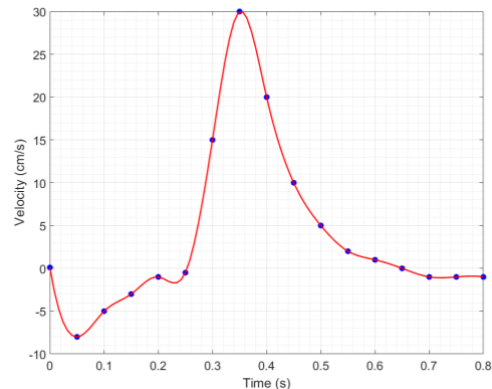


Figure 3. Time-dependent output blood velocity set on the mitral valve.

To evaluate the results of the simulations, we utilized MATLAB's Live Link, enabling data analysis and visualization. We performed an in-depth analysis of the blood flow characteristics, including velocity profiles, pressure distributions, and turbulence patterns within the LAA.

Simulation Results

In the Figure 4, we present a comprehensive analysis of flow velocities over one cardiac cycle using visualizations generated in COMSOL Multiphysics. These results are depicted through streamlines, with flow velocity information indicated by a color gradient at specific locations within the simulated left atrial model. Additionally, flow direction is graphically illustrated using arrows. We have chosen to showcase three representative cases to highlight the dynamic nature of blood flow within the left atrium.

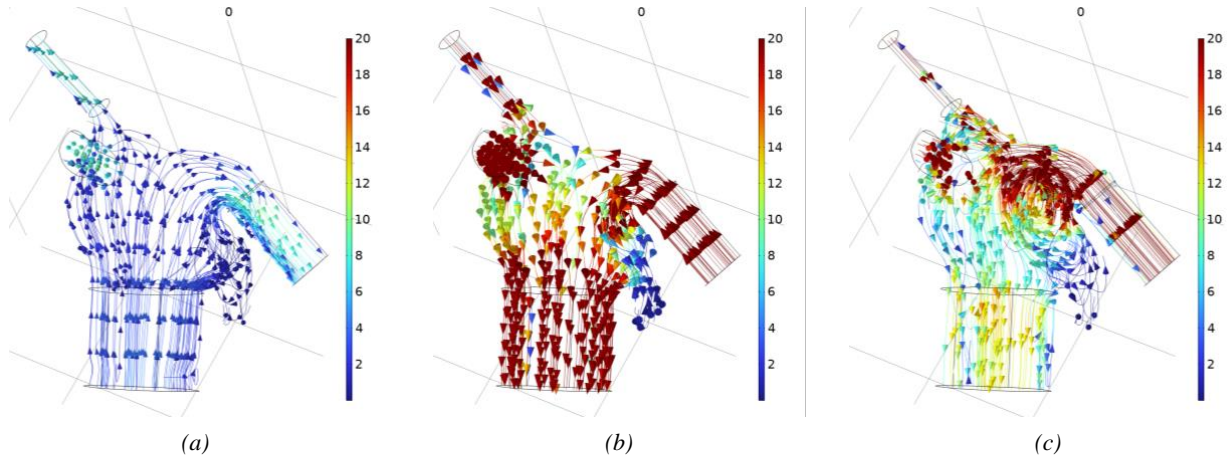


Figure 4. Streamline visualizations of cardiac flow velocities (in cm/s) in the left atrium (a) 0.15 Seconds, (b) 0.35 Seconds (Peak Flow), and (c) 0.45 seconds. Streamlines are color-coded to represent flow velocity in cm/s, while arrows indicate flow direction.

In Figure 4, the visualizations vividly illustrate the intensity and directionality of blood flow both within the left atrium and the left atrial appendage (LAA) during the peak phase of the cardiac cycle. Specifically, Figure 4(a) corresponds to the timestamp of 0.15 seconds, representing the early phase of the cardiac cycle. In Figure 4(b), which corresponds to 0.35 seconds, the moment of peak flow is captured, where the flow velocity reaches its maximum during the cardiac cycle. Finally, Figure 4(c) represents the time point of 0.45 seconds, signifying the rest phase when blood flow decelerates. These visual representations provide an understanding of the complex hemodynamic patterns within the left atrium. By analyzing these cases, we gain a comprehensive understanding of how blood flow velocities change over time, shedding light on critical aspects of cardiac function and potentially contributing to the assessment of individualized risk profiles for conditions such as clot formation.

In the Figure 5, we examine the left atrial appendage (LAA) and its flow velocity distribution in time of the peak flow (0.35 s). The flow exponentially decreases with the distance from LAA entrance. The visualization employs a green isocontour to signify areas within the LAA where the flow velocity drops to 1 cm/s. This specific velocity threshold is crucial because it identifies regions within the LAA with reduced flow speed, which increases the risk of clot formation or precipitation. By focusing solely on the

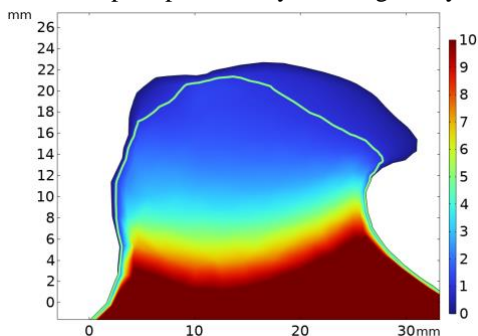


Figure 5. Detailed velocity profile in cm/s within the left atrial appendage (LAA) cross-section.

LAA, this figure provides a targeted view of areas where blood flow is significantly slowed, aiding in the identification of potential clot-prone regions within this critical cardiac structure.

Figure 6 presents the pressure map capturing the peak flow conditions at 0.35 seconds into the cardiac cycle. Notably, the highest relative pressure point is observed within the left atrium's main body, reaching a maximum value of 1100 Pa.

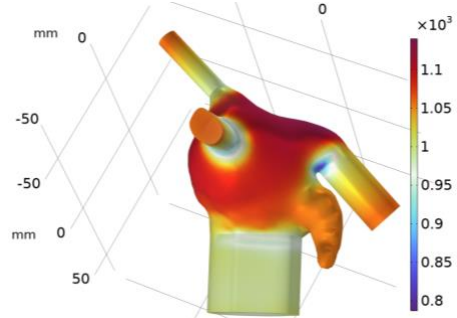


Figure 6. Comprehensive pressure map in pascals (Pa) at the 0.35-second, showcasing the peak flow rate.

Table 1 Maximum blood velocity magnitude at certain times corresponding to the Figure 4.

Time (s)	Maximum Blood velocity (cm/s)
0.15	9.59
0.35	79.14
0.45	48.24

Discussion

Our research provides valuable insights into the complex interplay of blood flow dynamics within the left atrium (LA) and the left atrial appendage (LAA), shedding light on critical factors that contribute to cardiovascular health risks, particularly in individuals with atrial fibrillation (AF).

The blood flow characteristics we observed, as detailed in Table 1, provide a clear representation of how flow velocities vary throughout the cardiac cycle. Notably, the peak flow velocity at 0.35

seconds reached 79.14 cm/s, emphasizing the dynamic nature of blood flow within the LA. Such variations are essential to understanding the hemodynamic forces acting on the atrial structures. Our use of patient-specific anatomical models allowed us to explore the influence of anatomical differences on blood flow patterns. These variations can significantly impact the risk of clot formation within the LAA. The visualizations in Figure 5, highlighting regions with reduced flow velocity (below 1 cm/s) within the LAA, underscore the importance of anatomical considerations in assessing clot-related risks. Clinically, this information could be used as a guidance for treatment decisions and interventions.

The results of our study could have direct clinical implications, particularly in the context of stroke prevention for AF patients. As for the possible limitations of the study, our simulations rely on several assumptions and simplifications, such as steady-state flow and uniform properties of blood. Future research could benefit from incorporating more complex and patient-specific boundary conditions to enhance the accuracy of our simulations. Additionally, validation of our computational findings with clinical data would strengthen the clinical relevance of our study.

Conclusion

In conclusion, our investigation of the blood flow dynamics within the LA and LAA using advanced computational modeling techniques has provided a deeper understanding of cardiovascular health risks, particularly in AF patients. The integration of patient-specific anatomical models, as well as the analysis of flow velocities and pressure distributions, offers a holistic view of cardiac function. This knowledge has the potential to impact clinical practice by guiding treatment strategies, ultimately leading to improved patient outcomes and reduced risks of clot-related complications associated with AF. Our research represents a significant step toward personalized cardiovascular care and shows the significance of interdisciplinary approach in addressing complex cardiac issues.

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