Impact of Wall Boundary Conditions in the Left Atrium on Indicators of Thromboembolism

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Summary

- Moving domain computational fluid dynamics (CFD) simulations in a patient-specific model of the left atrium (LA)
- Comparison of rigid wall simulation versus patient-specific movement in an atrial fibrillation (AF) patient
- Results suggest that moving walls substantially impact the hemodynamics in the left atrial appendage (LAA), and indices measuring endothelial susceptibility and risk of thrombus formation
- Hemodynamics in the LA cavity shows negligible difference between boundary conditions

Motivation

Reduced movement of the LA during AF is used to justify rigid wall CFD simulations in almost half of today's computational LA literature. This study aimed to investigate the effects of rigid versus moving wall movement on commonly used indicators of susceptibility to thromboembolism in the LAA, including low LAA ostium velocities and high values of endothelial cell activation potential (ECAP).

Methodology

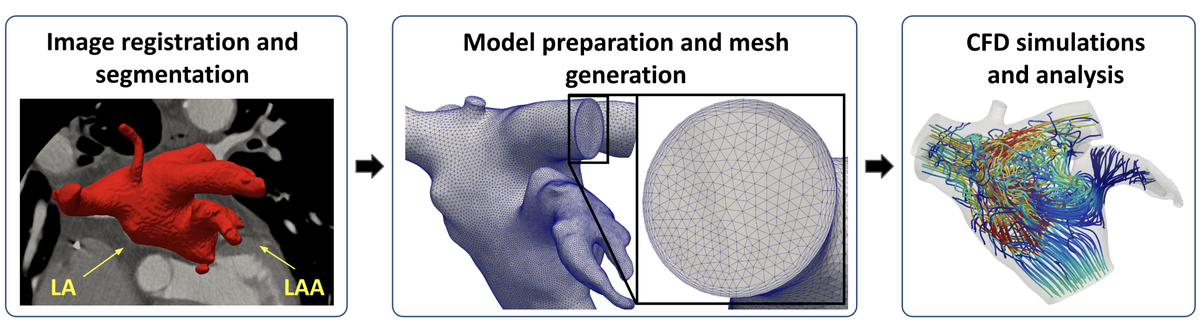

Figure 1: The workflow, including image registration and segmentation, pre-processing, CFD simulations, and analysis.

In Figure 1, we present the workflow used in this study. We acquired a patient-specific model of the LA from dynamic computed tomography (CT), and solved the Navier-Stokes equations in the arbitrary Lagrangian-Eulerian formulation using the verified and validated CFD solver *OasisMove*. The governing equations are:

$$
\begin{aligned}
\left.\frac{\partial \boldsymbol{u}}{\partial t}\right|_{\boldsymbol{x}} + (\boldsymbol{u}-\boldsymbol{w})\cdot\nabla\boldsymbol{u} &= -\frac{1}{\rho}\nabla p + \nu\nabla^2\boldsymbol{u} + \boldsymbol{f} && \text{in } \Omega(t),\\
\nabla\cdot\boldsymbol{u} &= 0 && \text{in } \Omega(t),\\
\boldsymbol{u} &= \boldsymbol{g} && \text{on } \Gamma^{\text{Wall}},\\
\boldsymbol{u} &= \boldsymbol{h} && \text{on } \Gamma^{\text{PV}},\\
\boldsymbol{\sigma}(\boldsymbol{u},p)\boldsymbol{n} &= -p_0\boldsymbol{n} && \text{on } \Gamma^{\text{MV}},
\end{aligned}
$$

where we varied the wall boundary condition $\boldsymbol{g}(\boldsymbol{x},t)$. For the rigid model $\boldsymbol{g}(\boldsymbol{x},t)=0$, while for the moving model we prescribed a non-zero boundary condition registered from the dynamic CT. At the pulmonary veins, we applied a Womersley velocity profile based on LA and left ventricular volume change. The volumetric mesh consisted of 3.5M cells, and included four boundary layers. Simulations were run using $\mathbb{P}_1$ finite elements for velocity and pressure, with a time step of $\Delta t = 2\cdot 10^{-4}$ s for five cardiac cycles.

Results

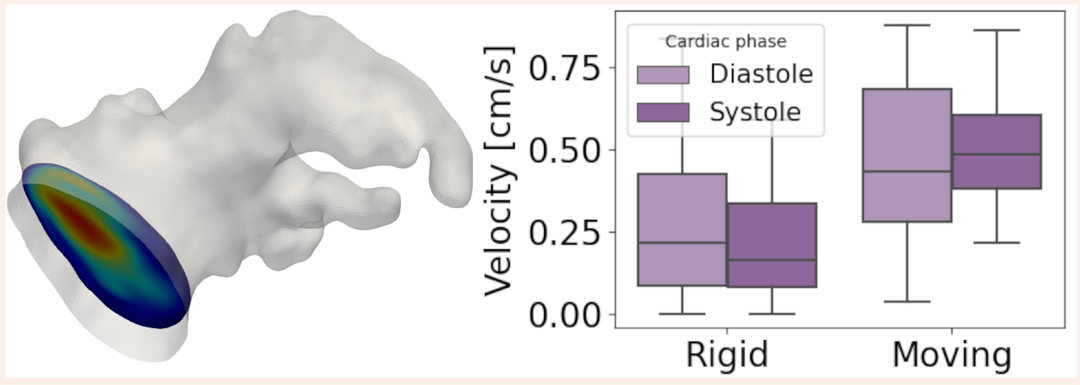

Figure 2: Box-plot of velocity through the LAA orifice at atrial diastole and systole.

In Figure 2 we present the LAA velocity values acquired from a cross-sectional slice at the orifice during atrial diastole and systole. The rigid model exhibits considerably lower velocities, with an average of 0.25 cm/s during diastole and 0.2 cm/s during systole. These velocities are 40% and 60% lower than those observed in the moving model, respectively.

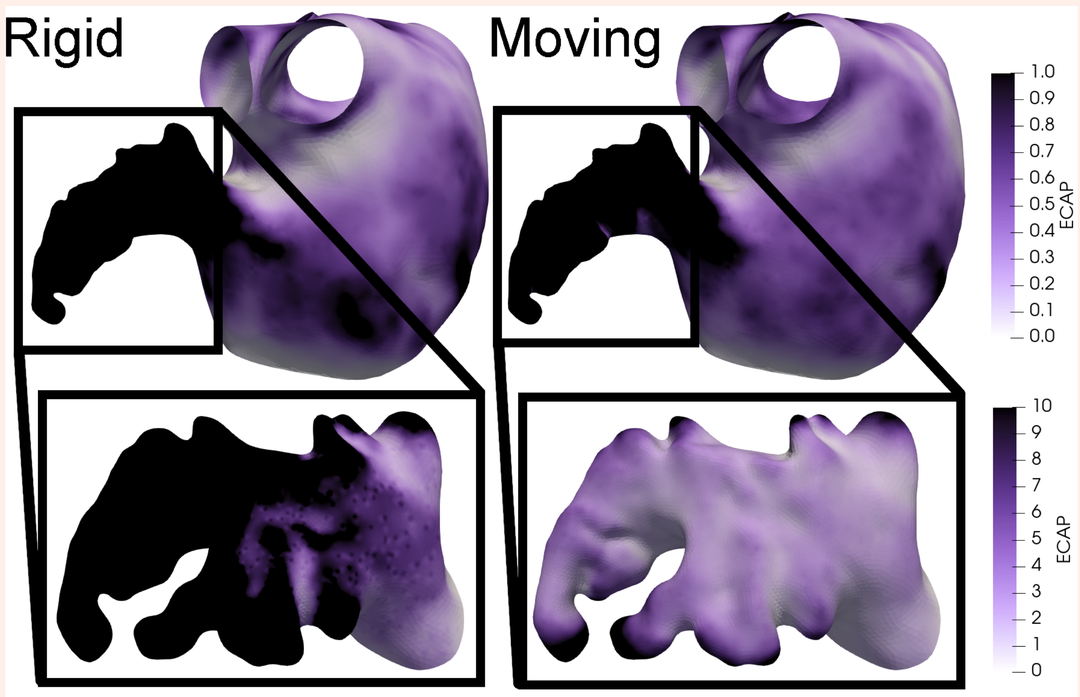

Figure 3: The left atrial model, featuring the ECAP distribution, serving as an indicator of thromboembolic risk. In this model, minimal variations are observed within the left atrial cavity, while the appendage demonstrates significant disparities.

In Figure 3 we present the ECAP distribution, where we observe distinct differences between the LA and LAA. In the LA cavity, the ECAP distribution and magnitude are relatively similar between the rigid and moving model, with a maximal difference of 5%. Contrary, in the LAA, the rigid model demonstrated significantly higher ECAP values, with a mean value of $4.3\cdot 10^5$ Pa^{-1}, compared to 3.4 Pa^{-1} measured in the moving model.

Conclusion

The results indicate that the condition of moving walls has a substantial influence on local hemodynamics and on indicators of thromboembolism in the LAA. The study demonstrates that risk stratification based on computational models should be approached with caution. The assumption of rigid walls in patients with atrial AF may result in limited physiological insights, potentially leading to inaccurate predictions of thrombus formation risk in the LAA.

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