Impact of Rigid Versus Dynamic Boundaries on Computational Fluid Dynamics Predictor of Left Atrial Appendage Thrombus Formation

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Highlights

- One of few rigid vs. dynamic domain comparisons within computational left atrium (LA) literature, using patient-specific movement from a patent with permanent atrial fibrillation (AF)
- Simulations were performed using OasisMove; a newly developed, verified and validated computational fluid dynamics (CFD) solver
- Preliminary results suggest that dynamic walls substantially impact the hemodynamics and predictors of thrombus formation in the left atrial appendage (LAA)
- In contrast, there is negligible difference observed in the LA lumen
- The study suggests that the rigid wall assumption commonly used for patients with AF is not sufficient in LA and LAA modeling

Motivation

Reduced movement of the LA during AF is used to justify rigid wall CFD simulations in over a third of today's computational LA literature. This study aimed to investigate the effects of rigid vs. dynamic wall movement on commonly computed predictors of thrombus formation in the LAA and risk of stroke, including low LAA ostium velocities and relative residence time (RRT).

Methodology

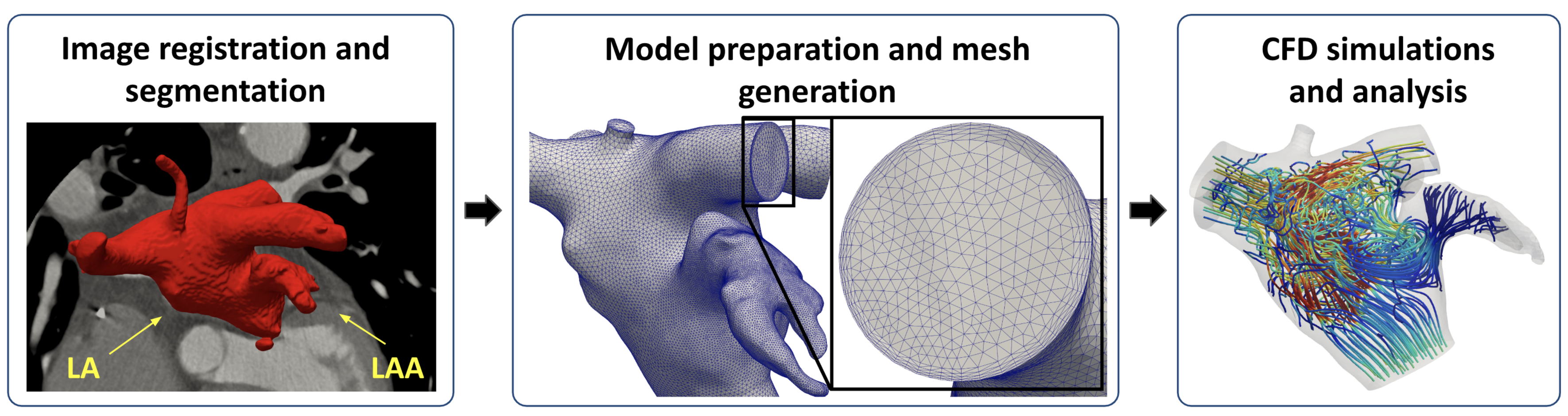

Figure 1: The main steps of the current study, including image registration and segmentation, pre-processing, CFD simulation, and post-processing.

A schematic of the workflow is show in Figure 1. We analyzed 4D computed tomography (CT) acquisitions of the LA in an permanent AF patient using a verified and validated CFD solver, solving the arbitrary Lagrangian-Eulerian formulation of the Navier-Stokes equations as follows:

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

For the dynamic model, we applied patient-specific boundary movement registered from the CT as the wall condition, in contrast to the rigid wall model. At the pulmonary veins, we applied velocity profiles based on LA and left ventricular volume change. The computational model consisted of 3.6M cells, and included four boundary layers to more precisely capture hemodynamic forces related to flow stagnation such as RRT. Simulations were performed with a time step of $\Delta t = 2\cdot 10^{-4}$ s.

Results and Conclusions

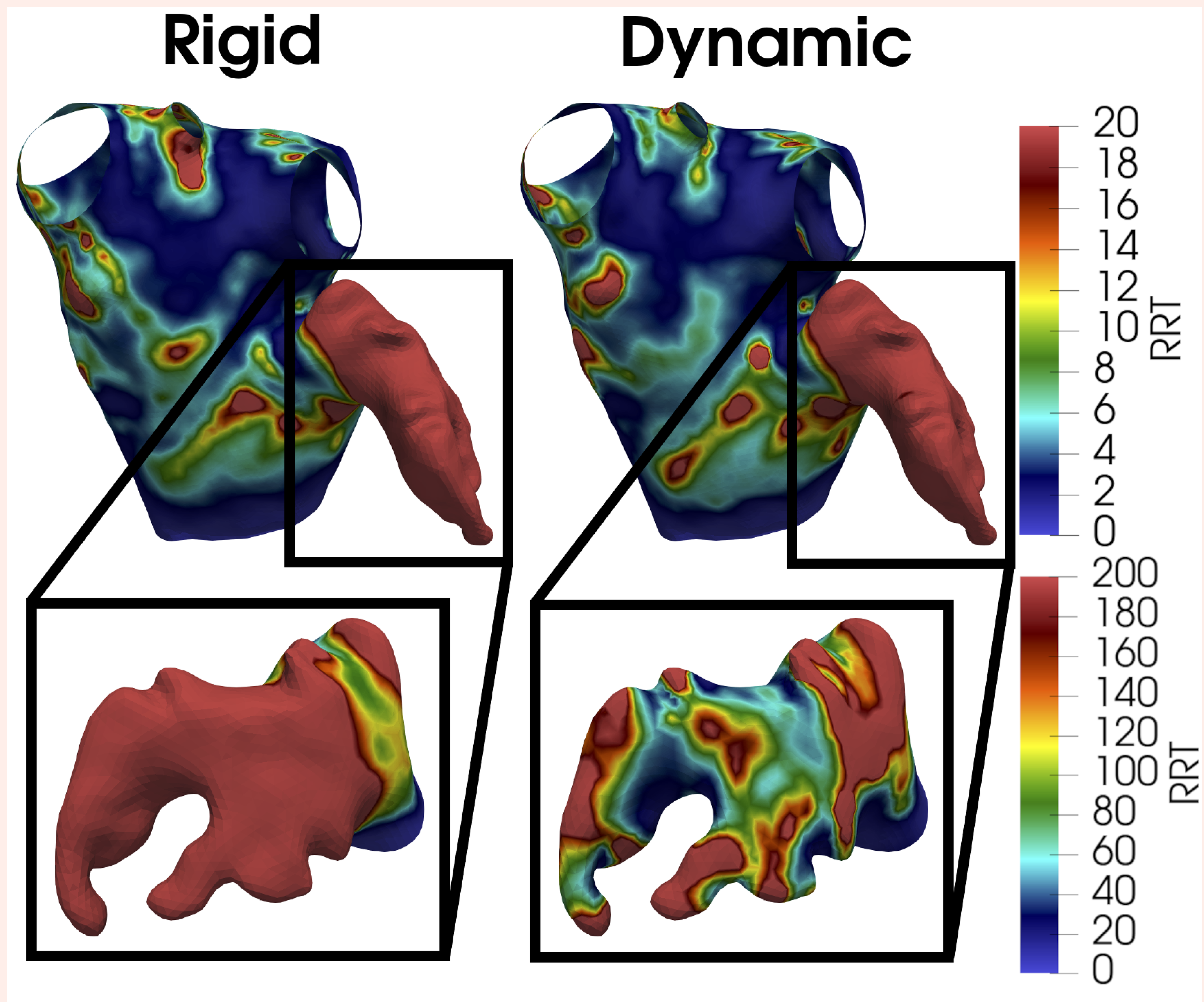

Figure 2: Qualitative results show visible differences in the LAA, but negligible differences in the LA lumen.

Qualitative results displaying RRT in both anatomies are shown in Figure 2, with a zoomed in view on the LAA. First, considering the LA lumen, there is no qualitatively noticeable differences in magnitude, nor location of high RRT, and the RRT is generally low indicating little flow stagnation. Concentrating on the LAA, there are very noticeable qualitative differences, between the rigid and moving case. Interestingly, for the rigid case, the only area with low RRT (< 100) is particularly exposed in the dynamic case. Quantitatively, we observed low LAA ostium velocities averaging at $5.0\cdot 10^{-3}$ m/s during atrial diastole and $6.0\cdot 10^{-2}$ m/s during atrial systole in the rigid model, which was 84% and 7.7% lower than the dynamic model, respectively. Mean LAA RRT values reached close to $1.7\cdot 10^{7}$ 1/Pa in the rigid model, compared to $1.3\cdot 10^{2}$ 1/Pa measured in the dynamic model. For the remaining LA lumen, there was only a $\pm$ 5% difference in RRT.

The results suggest that dynamic walls have a considerable impact on the hemodynamics, consequently on predictors of thrombus formation in the LAA. Although the study was limited to one case, we have demonstrated that risk stratification based on computational models should be performed with caution, and that the rigid wall assumption in patients with AF may lead to limited physiological insight.

Contact

References Software Poster

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