Design, Development and Test of Different Cardiac Contraction Models in Atrial Fibrillation

Michela Colonnelli[1], Alessandro Masci[1], Corrado Tomasi[2], Cristiana Corsi[1]

[1] DEI, University of Bologna, Campus of Cesena, Bologna, Italy
[2] Deparment of Cardiology, Ospedale Santa Maria delle Croci, Ravenna, Italy

Abstract

Computational fluid dynamics represents a valuable non-invasive approach to determine and assess physically meaningful parameters in a complex fluid dynamics system represented by AF. The aim of this study was the design, development and test of different LA motion fields in AF on a patient-specific 3D anatomical model to clarify the influence of contraction models on LA hemodynamics. Since LA motion field is not available from clinical data during AF, three displacement models were designed to simulate the irregular, disorganized, very rapid and strongly reduced LA contraction, a random model, a discrete random model and a con-tinuous sinusoidal model. Blood velocity fields, kinetic energy, vortex structures and blood stasis were analyzed in both SR and AF conditions in the LA. Velocities in SR were higher than in AF, particular-ly during atrial systole. The three AF models resulted in different wash-out velocities both at the mitral valve and at the ostium of the LA appendage. Vortices were also differently distributed inside the LA showing a more organized flow in the sinusoidal model which was also characterized by the lowest blood stasis (8.6%) inside the LA appendage. Overall, different LA de-formation models in AF affect LA hemodynamics and additional studies should be performed to develop a realistic contraction model to simulate AF episodes.

1. Introduction

Atrial Fibrillation (AF) is the most common arrhythmia. It was demonstrated that the lifetime risk of developing AF after 40 years of age is 26% for men and 23% for women, for European descend [1]. Over 6 million Europeans suffer from this arrhythmia and its prevalence is estimated to at least double in the next 50 years, as the population ages. Moreover, AF increases five-fold the risk of stroke [2]. In particular, left atrial appendage (LAA) is the left atrial site with the highest blood stasis risk, increasing thrombus formation and stroke. In fact, 90 % of the intracardiac thrombi in patients with cardioembolic stroke/TIA originally develop in the LAA [3]. To this purpose, several clinical studies suggested that stroke risk stratification could be improved by using hemodynamic information on the left atrium (LA) and left atrial appendage (LAA). These studies affirms that AF modifies the intra-atrial blood flow dynamics, and this could imply an increase of blood stasis risk and, therefore, clot formation and embolism. One factor is the anatomical remodeling, which consists in the progressive LA enlargement and increase in the LAA elongation [4]. A second factor is given by the altered mechanical function. Atrial contraction becomes completely chaotic and therefore during the AF episodes its efficacy is scarse. Moreover, there is a progressive loss of atrial function even in sinus rhythm. Howewer, the exact way these complex mechanisms interplay cannot be assessed experimentally and remains largely unknown.

Computational fluid dynamics (CFD) represents a valuable non-invasive approach to determine and assess physically meaningful parameters in a complex fluid dynamics system such as velocity, the cardiac blood flowrates, vorticity, turbulent kinetic energy, etc. Morevoer, CFD modeling of the left atrium (LA) in atrial fibrillation (AF) has not been faced exhaustively considering the relevance of its potential clinical impact. Yet, in view of a personalized approach for the CFD simulations of the LA, the necessity of a realistic motion model of the atrial cavity during an AF episode is a crucial point for a creation of a complete patient-specific atrial fluid dynamics model. Therefore, the aim of this study was the design and development and testing of different contraction model of the LA. These motion models are used as input for the computational fluid dynamics model of the left atrium, proposed in [5, 6], in order to enhance the differences in haemodynamics parameters for each LA motion model used for the simulations. The developed model was tested in patients in both sinus rhythm (SR) and AF for a comprehensive evaluation of hemodynamic implications of AF episodes in both LA and left atrial appendage (LAA).

 ISSN: 2325-887X DOI: 10.22489/CinC.2019.173

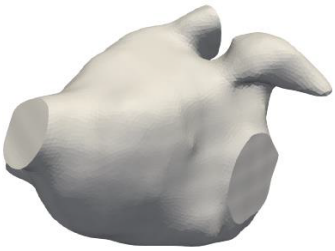

Figure 1. LA anatomical model.

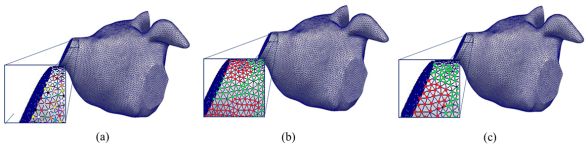

Figure 2. Graphical representation of the contraction models of the LA: (a) random motion model; (b) discrete motion model; (c) sinusoidal motion model.

2. Methods

Data-set consisted in a LA 3D anatomical model and its relative motion model in SR developed in [5, 6], extracted from CT data with specifically image segmentation algorithms (see Figure 1). Non-rigid data registration of the CT dynamic acquisitions allowed us to derive the 3D LA motion field in SR. Since the LA motion field is not available from clinical data during an AF episode, three LA contraction models were designed to simulate the irregular, disorganized, very rapid and strongly reduced LA contraction. In the following paragraphs, we detail these motion models for simulating AF.

2.1. Random Motion Model

To simulate conditions preceding chronic AF (paroxysmal and persistent AF conditions) we chose first to model atrial contraction by employing a random displacement applied independently to each vertex of the anatomical LA model and consisting in a sinusoidal function at a frequency of 4 Hz multiplied by a random factor from an uniform probability density function from 0 to 1. The contraction frequency was defined considering the typical frequency of atrial fibrillatory episodes [7]. Moreover, the sinusoidal wave was modulated by a small amplitude (0.1 mm) in order to avoid numerical issues arising from an excessive worsening of the mesh quality. In figure 2(a), we show a schematic description of this first motion model chosen for simulating contraction during AF episodes.

2.2. Discrete Motion Model

In this model the idea was to divide the LA mesh in neighbouring regions and apply on each one of them a different displacement field. More specifically, LA mesh was divided in 16 regions and we imposed to each region a random contraction/expansion motion model following the radial direction with respect to the midpoint of the region. In figure 2(b), we observe the application of this method on a specific part of the LA mesh.

2.3. Sinusoidal Motion Model

The most critical point of the previous model was to manage the mesh points at the boundary between two adjacent regions that could create great degeneration of the mesh quality. For this reason, to simulate the simultaneous contraction/expansion of LA adjacent anatomical regions in a continuous way and not with a discrete approach, as we have seen in the second method, we applied this particular sinusoidal function:

$$\mathbf{d}(\mathbf{\Theta}) = \mathbf{r}(\mathbf{\Theta}) \cdot \sin(\frac{\mathbf{2\pi\Theta}}{\mathbf{L}}) \cdot \sin(\omega \mathbf{t});$$

where r represents the maximum displacement amplitude, Θ the position reffered to the center of mass of the atrial cavity, and L the spatial period. Amplitude and frequency of the sinusoidal wave were chosen by following the work of [8]. L was setted to 20, representing the number of periodic repetitions of the sinusoidal wave through the spatial mesh domain. Amplitude and frequency were setted to 1 mm and 4 Hz respectively. Figure 2(c) shows the description of the aforementioned displacement model applied to the LA mesh nodes.

2.4. Numerical Simulations

The CFD model described in [6] was used in order to apply the developed three motion models to simulate the LA motion in AF. Moreover, the motion field of the LA extracted by the dynamic CT sequence was used in order to perform the CFD simulation of the LA in SR. Five cardiac cycles were simulated.
Blood velocity, kinetic energy, vortex structures and blood stasis in the LA were analyzed in both SR and AF conditions.

3. Results and Discussion

From the analysis of the CFD simulation results, we found that the most important differences in the four motion models were during the atrial diastole phase. Indeed, we found an increment of the velocity up to 15-20 cm/s in proximity of the pulmonary veins (PVs) only for the SR

simulation. For the three motion models of AF we found a slightly increment of the PVs velocity only for the sinusoidal model and not for the other two motion fields, as shown in Figure 3. The sinusoidal model leads to a higher expansion of the atrial chamber and consequently to an higher flowrate through the PVs with respect to the discrete and random models. Obviously, another relevant difference in the cardiac cycle between the four simulated conditions is during the atrial systole: despite its reduction caused by the persistence of the AF, we found an increase of the velocity at the mitral valve (MV) for the SR simulation with respect to the other three and this is probably related to a contractile activity of the LA that pushes blood through the MV in the LV. However, in three motion models simulating AF this increment of the velocity at the MV was strongly reduced.

In general, velocities in SR were higher than in AF, particularly during atrial systole. Focusing on the LAA fluid dynamics, the three AF models resulted in different velocities both at the tip and at the ostium of the LA appendage (see Figure 4). The SR condition, as expected, shows higher velocities inside the LAA and consequently a better washout of the blood flow expecially in the LAA distal part, as observed in Figure 4. Regarding the three AF simulations, the one in which we applied the sinusoidal motion model showed higher velocities inside the LAA with respect to the other two displacement fields. Moreover the blood flow could reach also the LAA tip with this particular motion model, thus favouring a lower risk of blood stasis and thrombi formation. Focusing on the vorticity, most of the vortex structures were located near to the LAA ostium. In SR, we found a higher number of vortex structures overall the LAA throughout the cardiac cycle with respect to the AF simulations. The AF sinusoidal motion model allowed the vortex structures to reach with a higher probability the LAA tip expecially with respect to the random model simulation where the vortex structures were concentrated near the LAA ostium. From velocity and vortex structures analysis, as expected, blood washout was more effective in SR with respect to the three AF simulations. Considering the three contraction models, the sinusoidal model was the one showing an adequate blood washout, despite less than SR condition, thus implying a lower thrombi formation risk and consequently stroke risk. In order to confirm the aforementioned findings, we performed a specific study to try to quantify the LAA blood stasis. Indeed, we populated the LAA with 500 fluid particles at the beginning of the simulation and counted how many remained inside the LAA after each cardiac cycle. After 5 cycles, 3.8% of the particles remained in the LAA in SR, while 8.6% remained in AF with the sinusoidal motion model. 12.6% remained in AF with the discrete motion model and 15% in AF with the random model. These results confirmed our considerations based on the velocity and vorticity analysis. We found an expected reduced washout expecially for the AF random model and discrete model AF which in the long term might be indicative of the generation of blood clots.

4. Conclusions

In this work we described in detail an automatic framework which enabled to apply three different displacement fields in order to simulate the LA motion throughout the cardiac cycle during AF episodes. These three different displacement fields were the input for the CFD model and haemodynamics parameters were computed in order to quantify the differences between the three approaches. Moreover, the SR condition was simulated, having available the SR motion field extracted from the CT dynamic sequence. Results enhanced the SR condition, as expected, showed higher velocities and number of vortex structures in the LA and in the LAA. Regarding the three motion models simulating AF, the one with the results nearer to the SR condition was the sinusoidal displacement field, where we observed a lower blood stasis risk, expecially in the LAA, with respect to the random and discrete motion models. The CFD simulation could improve the knowledge on intra-atrial blood stasis and on the probability of clot formation with a completely personalized approach and potentially enables optimized patient risk stratification and therapy. Unfortunately, to simulate AF conditions, different LA deformation models affect simulation results, therefore additional studies should be performed to develop a realistic contraction model to simulate AF episodes.

References

[1] Zakeri R, Van Wagoner DR, Calkins H, Wong T, Ross HM, Heist EK, Meyer TE, Kowey PR, Mentz RJ, Cleland JG, et al. The burden of proof: the current state of atrial fibrillation prevention and treatment trials. Heart Rhythm 2017; 14(5):763–782.

[2] With the special contribution of the European Heart Rhythm Association (EHRA) D, by the European Association for Cardio-Thoracic Surgery (EACTS) E, Members AF, Camm AJ, Kirchhof P, Lip GY, Schotten U, Savelieva I, Ernst S, Van Gelder IC, et al. Guidelines for the management of atrial fibrillation: the task force for the management of atrial fibrillation of the european society of cardiology (esc). European heart journal 2010;31(19):2369–2429.

[3] Yaghi S, Song C, Gray WA, Furie KL, Elkind MS, Kamel H. Left atrial appendage function and stroke risk. Stroke 2015; 46(12):3554–3559.

[4] Gupta DK, Shah AM, Giugliano RP, Ruff CT, et al. Left atrial structure and function in atrial fibrillation: Engage af-

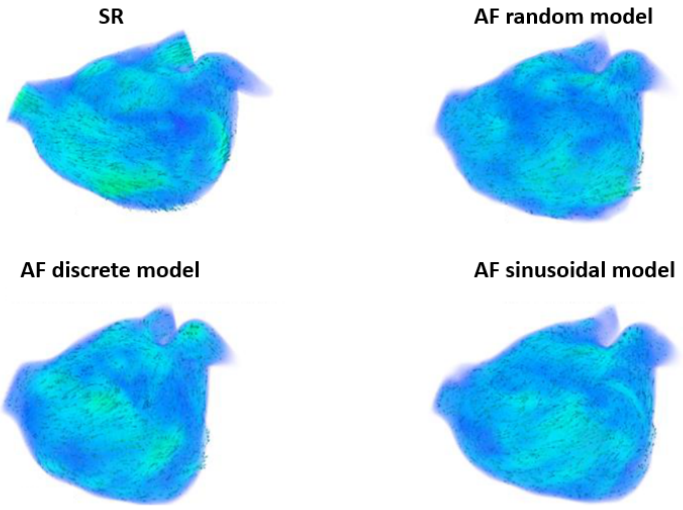

Figure 3. LA velocity field computed by the CFD model for SR condition and for the three motion models used for simulating AF.

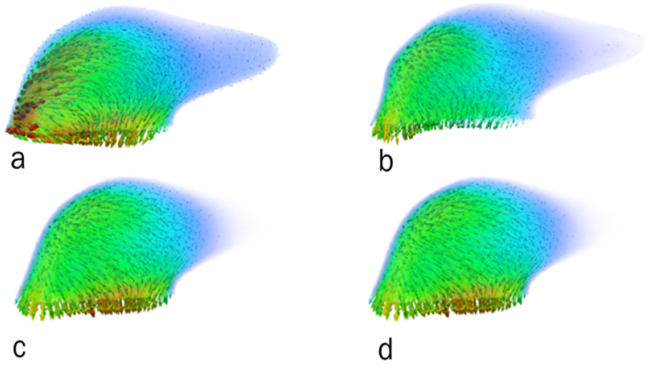

Figure 4. Simulated blood velocities inside the LA appendage during the atrial diastole in SR (a), applying the continuous sinusoidal model (b), the discrete random model (c) and the random model (d).

timi 48. European Heart Journal 2014;35(22):1457–1465. ISSN 0195-668X.

[5] Masci A, Alessandrini M, Forti D, Menghini F, Dedé L, Tommasi C, Quarteroni A, Corsi C. A patient-specific computational fluid dynamics model of the left atrium in atrial fibrillation: Development and initial evaluation. In International Conference on Functional Imaging and Modeling of the Heart. Springer, 2017; 392–400.

[6] Masci A, Barone L, Dedè L, Tomasi C, Fedele M, Quarteroni AM, Corsi C. The impact of left atrial appendage morphology on stroke risk assessment in atrial fibrillation: a computational fluid dynamics study. Frontiers in physiology 2018; 9:1938.

[7] Langley P, Bourke J, Murray A. Frequency analysis of atrial fibrillation. In Computers in Cardiology 2000. Vol. 27 (Cat. 00CH37163). IEEE, 2000; 65–68.

[8] Koizumi R, Funamoto K, Hayase T, Kanke Y, Shibata M, Shiraishi Y, Yambe T. Numerical analysis of hemodynamic changes in the left atrium due to atrial fibrillation. Journal of biomechanics 2015;48(3):472–478.

Address for correspondence:

Alessandro Masci
DEI, University of Bologna
Via Risorgimento 2, 40136 Bologna, Italy
E-mail address: alessandro.masci4@unibo.it