Beyond Static Geometries: A Novel Dynamic 3D Hemodynamic Model of Left Atrium for Personalized Cardiovascular Risk Assessment

João Lameu[1], João G B Torro[1], Rodrigo L Amaral[1]

[1]Federal University of ABC, São Bernardo do Campo, Brazil

Abstract

Left ventricular diastolic dysfunction (DD) is a progressive disease that deprecates the left atrial (LA) mechanical function and ventricular filling. DD is present in 25–30% of older adults, increases cardiovascular and thrombotic risk by increasing blood stasis in LA. We propose a computational approach using clinical LA phasic volume to dynamically model LA mechanical performance and high-resolution 3D hemodynamics. A single LA geometry captured at end-systole was used to generate volume-varying meshes. These meshes deform according to reservoir, conduit, and booster pump phases, incorporating mitral valve closure. Three cases were modeled: (1) healthy dynamic LA, (2) pseudonormal DD dynamic LA, and (3) pseudonormal DD static LA. Healthy simulations showed organized vortical flow and protective Time-Averaged Wall Shear Stress (TAWSS > 1 Pa). In pseudonormal DD, flow stasis increased, particularly in the left atrial appendage (LAA), where TAWSS dropped below 0.1 Pa, indicating elevated thrombogenic potential. The dynamic model predicted superior LAA washout than the static model, especially in the healthy case, due to preserved atrial ejection. The framework enables personalized thrombotic risk assessment directly from standard clinical imaging, eliminating the need for costly dynamic reconstructions being more accessible in clinical practice.

1. Introduction

Left ventricular diastolic dysfunction (DD) affects 25-30% of community-based populations, with higher prevalence in elderly patients and those with hypertension, diabetes, or chronic kidney disease [1]. This progressive condition impairs ventricular filling, elevates intracardiac pressures [2,3], and disrupts left atrial mechanics and hemodynamics. DD progresses through three stages: altered relaxation (Type I), pseudonormal (Type II), and restrictive (Type III) patterns [3,4], each progressively compromising atrial volumes and ejection fractions while heightening thrombogenic risk [3-6].

The left atrial volume index (LAVI) serves as a key biomarker, with values >34 mL/m² indicating significant dilation [7]. Phasic LAVI enable calculation of passive, active, and total ejection fractions of LA, crucial for risk stratification in arrhythmias and DD [5,6,8,9]. Clinical observations reveal progressive LAVI increases and LA ejection fraction reductions with DD severity, particularly affecting active ejection fraction and correlating with atrial fibrillation onset [5].

Despite advances in understanding LA mechanics and their relationship with disease progression, a high-resolution hemodynamic assessment under DD conditions remains underexplored, particularly in terms of 3D volume dynamics and thrombotic risk quantification. While Computational Fluid Dynamics (CFD) enables detailed LA flow analysis, most models neglect dynamic volume changes and mitral valve effects [10-12]. This study introduces a clinically accessible CFD approach incorporating dynamic volume variations based on phasic volumes (maximum, at end-systole, V_{max}; pre-atrial contraction, $V_{pre\text{-}A}$; minimum, V_{min}), closed mitral valve effects, and pulsatile flow profiles to improve thrombotic risk stratification in DD patients.

2. Mathematical Modeling

The proposed model uses a 3D LA geometry, obtainted from public dataset [9], acquired at end-systole to generate the base mesh. During preprocessing, N meshes with varying volumes were generated, by isotropic scaling of the base mesh to represent the cardiac cycle phases: reservoir, conduit, and active contraction (booster pump, BP) [10]. The meshes were dynamically deformed during simulation according to the cycle phase, using Events functionality of Ansys Fluent Solver, as illustrated in Figure 1-a. The approach incorporated closed mitral valve effects during reservoir phase. Cardiac cycle meshes were dynamically adjusted at 1 mL volume intervals, though the method accommodates arbitrary resolution based on required detail levels.

The phasic volumes - V_{min}, V_{max} and $V_{pre\text{-}A}$ (Figure 1-a) - obtained from clinical data ware summarized in Table 1. These volumes quantified LA volume rate change per atrial cycle phase, defining the dynamic mesh model (Figure 1-b, green lines). In Table 1, *p-LAEF* (%) = $100\cdot[(V_{max} - V_{pre,A})/V_{max}]$, *a-LAEF* (%) =$100\cdot[(V_{pre,A} - V_{min})/V_{min}]$ and *LAEF* (%) = $100\cdot[(V_{max} - V_{min})/V_{max}]$ are the passive, active and total left atrium ejection fraction.

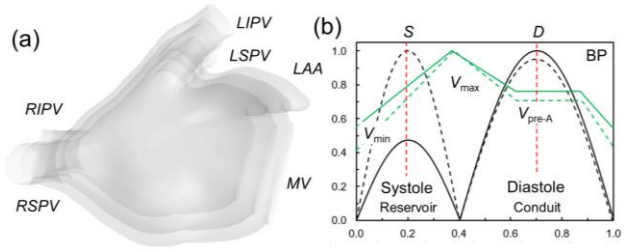


Figure 1. (a) Superposition of three surfaces of LA at phasic volumes with anatomical markers (R, L – right, left; I, S – inferior, superior, PV – pulmonary vein, MV – mitral valve, LAA – left atrial appendage; (b) Normalized profiles of flow rate (black lines) with *S* and *D* peaks, along the LA phases (BP is the booster pump), and LA volume (green lines) with details of phasic volumes.

Table 1. Clinical parameters for LA phasic volume and ejection fractions for dynamic model [5,7,8,10]. The static model used only V_{max}.

Clinical parameter	Healthy	Pseudonormal (DDII)
$LAVI_{max}$ (mL/m^2)	29	48
V_{min} (mL)	24	53
V_{max} (mL)	58	96
V_{pre-A} (mL)	41	73
p-LAEF (%)	29	24
a-LAEF (%)	42	27
LAEF (%)	59	45

Phasic volumes were obtained from clinical data of LAVI considering a Body Surface Area of 2.0 m² [7].

The Ansys CFD package was employed in all steps, from 3D geometry preparation (Ansys SpaceClaim), mesh generation (Ansys Meshing) to the numerical solution (Ansys Fluent) and post-processing (Ansys CFD-Post). The base geometry was used to generate hybrid-element meshes with about 1.8 million cells. In wall-zones, 10 inflation layers were generated (first layer thickness of 0.01 mm, growth rate of 1.2) for accurate velocity gradient estimations. The main characteristics of the model were: laminar, isothermal, incompressible, transient/pulsatile flow, with non-Newtonian blood behaviour (Carreau-Yasuda model) [11].

Pulmonary vein (PV) inflow was modelled using clinically-derived pulsatile profiles [4], featuring systolic (*S*) and diastolic (*D*) peaks. Healthy cases used $S/D = 1.0$, while DDII used $S/D \approx 0.5$ [4]. Cardiac output was set to ~5 L/min at 75 bpm (cycle duration T = 0.8 s). In dynamic models, the mitral valve closed during reservoir phase (open otherwise) with 8 mmHg outlet pressure [13]. The static model (rigid mesh) maintained an open valve throughout the cardiac cycle at constant 8 mmHg [13]. Three scenarios were evaluated: (1) healthy case with dynamic model, (2) pseudonormal DD (Type II) with dynamic model, and (3) pseudonormal DD with a rigid mesh (static approach).

For the numerical solution, a residual target (RMS) of 1×10^{-4} was employed. Two complete cardiac cycles were simulated using a time step of 1×10^{-3} s, and time-averaged values were obtained for the last cycle. The thrombus-prone regions were assessed by blood stasis zones, i.e., low blood velocities, usually below 10 cm/s [14] and the Time-Averaged Wall Shear Stress (*TAWSS*), with the thromboembolic risk classified as: high risk for *TAWSS* < 0.1 Pa, low risk for *TAWSS* > 0.4 Pa [13,15]. For the dynamic models, the time-averaged values were obtained for the mean LA volume along the cardiac cycle.

3. Results and Discussion

Figure 2(i-iii) presents the instantaneous results for velocity fields at systolic peak, diastolic peak and end-diastole (onset of atrial contraction), alongside time-averaged velocity (Figure 2-iv). Figure 2(v-vi) demonstrates two perspective views of the *TAWSS* field on LA surface. In the healthy case with a dynamic mesh (Figure 2-a), ventricular systole shows blood inflow from the PVs filling the entire atrial chamber, including the LAA, accompanied by a prominent vortical flow in the LA. This organized pattern mitigates stasis and optimizes LA filling [16,17]. The dynamic model accounted for LA expansion and gradual pressure rise during filling, yielding a more realistic hemodynamic pattern [18]. In pseudonormal DD (Type II) elevated LA pressure compensates for impaired ventricular relaxation, significantly altering hemodynamics (Figure 2-b). Compared to the healthy case (Figure 2-a), blood stagnation predominated throughout the LA chamber, attributed to LA dilation and absence of vortical flow patterns (Figure 2-b). PV inflow jets also exhibited lower velocities, consistent with larger cross section areas**,** consistent with clinical observations [19]. For the static DDII model (Figure 2-c), continuous mitral outflow produced near-uniform velocity field with pronounced LAA stagnation. Rigid-wall simulations overestimate thrombotic risk by neglecting atrial deformation's impact on stasis [12,15].

The conduit phase (open mitral valve) enables passive PV-to-ventricle flow during early diastole. The LA acts as a conduit, directing flow efficiently to the mitral annulus [16,17], as seen in both dynamic models (Healthy, Figure 2-a; DDII, Figure 2-b). The healthy case maintained the vortical pattern initiated during systole, driven by left PV inflow, while right PV flow traversed the vortex periphery. This mechanism prevents stasis and enhances LAA washout – markedly diminished in DDII [16,17]. Declining LA pressure during ventricular filling maintained streamlined flow with minimal stasis [16,17].

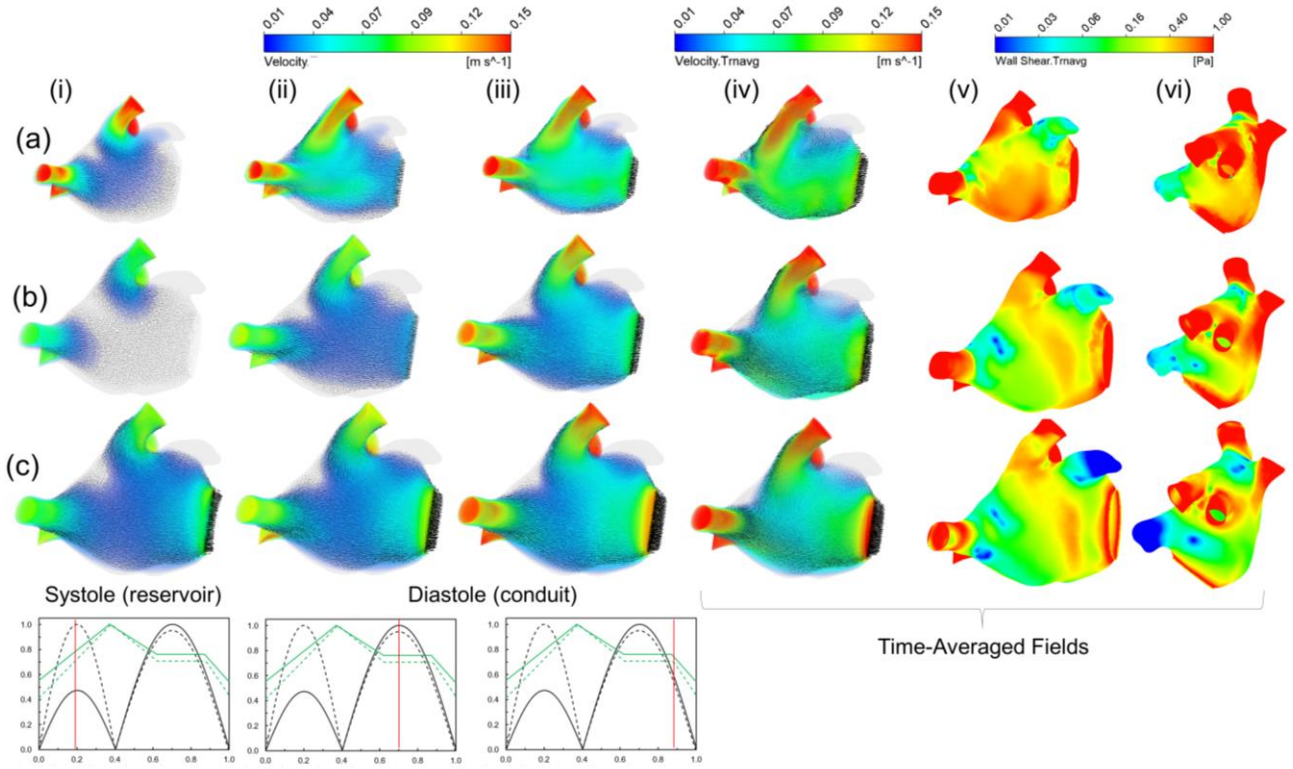


Figure 2. (a) Healthy dynamic model, (b) DDII dynamic model; (c) DDII static model. (i)-(iii) Instantaneous velocity fields at (i) systolic peak, (ii) diastolic peak and (iii) diastolic end; (iv-vi) time-averaged fields for (iv) velocity and (v-vi) *TAWSS* for two perspective view of LA surface.

Healthy cases exhibited higher velocities owing to smaller volumes versus DDII [16,17]. Although dynamic and rigid models shared similar conduit-phase patterns in the main chamber, DDII cases showed LAA stagnation from direct mitral flow, exacerbated by chamber enlargement. The superior active ejection fraction in healthy dynamic models reduced LAA stagnation, validated by *TAWSS* distributions (Figure 2-v-vi). Smaller LA volumes promote distinct intra-atrial vortices, whereas larger volumes disrupt them (Figure 2-b,c) [20,21]. Vortices improve PV-to-MV transit efficiency [21], their disruption in DDII elevates stasis and thrombogenic risk – particularly in the LAA – due to impaired washout from reduced active/total ejection fractions [20,21] (Figures 2-b,c). At cycle end, the healthy dynamic case achieved higher global time-averaged velocity, attributed to smaller atrial volume [20,21], whereas static DDII showed higher transmitral velocity but lower global velocity, producing larger stasis zones consistent with *TAWSS* analysis (Figure 3).

The *TAWSS* distribution at LA surface (Figure 3) revealed distinct patterns. The healthy dynamic model demonstrated higher, physiological protective *TAWSS* (> 1.0 Pa) throughout the atrial chamber, reflecting lower thromboembolic risk due to higher blood velocities and ejection fractions and smaller LA volume. For DDII cases, low-shear regions appeared in the upper atrium zone between opposite PVs, which can be attributed to flow jet interactions and chamber dilation. Very low *TAWSS* (< 0.1 Pa) were also noticed in LAA, particularly at its apex in dynamic models - a known thrombogenic site in arrhythmias and cardiac dysfunction [11,22]. The static DDII model exhibited extensive low-*TAWSS* areas (< 0.1 Pa) covering over half the appendage length (from apex to ostium) and significantly larger stasis zones compared to dynamic models. This can be attributed to lower LAA velocities inherent from the absent active ejection.

4. Conclusion

This study demonstrated that our clinically accessible CFD framework incorporating dynamic volume variations parameterized by phasic volumes, mitral valve closure effects, and pulsatile flow profiles, enabling effective hemodynamic discrimination of thrombotic risk in DD patients. In healthy case, dynamic modelling demonstrated efficient LA filling with organized vortical flow. minimizing stasis, maintaining physiological protective *TAWSS*. Pseudonormal DD (Type II) cases exhibited larger volumes disrupting vortex formation, with extended low-velocity zones and elevated LAA stagnation. The dynamic model quantified active ejection effects, revealing reduced LAA stasis and volume-dependent *TAWSS* distributions

corresponding to clinical thrombotic sites, along with conduit-phase efficiency impairment in DDII. By integrating routine clinical parameters with hemodynamic simulation, this framework provides a practical tool for risk stratification by *TAWSS* mapping and disease monitoring by accounting for the LA remodelling progression. Future work should evaluate and validate the results in larger cohort of patient-specific geometries while maintaining the computational efficiency of the proposed dynamic approach.

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Conflict of Interest

The authors declare do not have conflict of interest

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João Lameu da Silva Jr.
Federal University of ABC (UFABC)
Alameda da Universidade, s/n, São Bernardo do Campo, Brazil
joao.lameu@ufabc.edu.br