



Numerical Analysis of Cardiovascular Fluid Mechanics

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Abstract- Cardiovascular diseases remain the leading cause of death globally, driving an urgent need for predictive, patient-specific modeling of blood flow and vascular mechanics. This paper synthesizes recent advances in numerical methods for cardiovascular fluid mechanics, emphasizing their transition from research tools to clinical applicability. We analyze four core methodological domains: (1) immersed fluid–structure interaction (FSI) methods for deformable vessels and heart valves; (2) machine learning–enhanced reduced-order modeling (ROM) for real-time hemodynamic assessment; (3) multiphysics integration of hemodynamics, tissue mechanics, and biological processes for disease progression modeling; and (4) turbulence modeling and uncertainty quantification in large arteries. Drawing on peer-reviewed studies from 2025–2026, we demonstrate that modern numerical frameworks achieve patient-specific digital twins capable of predicting wall shear stress (WSS), fractional flow reserve (FFR), and rupture risk in aortic dissection, coronary artery disease, and cerebral aneurysms. We conclude with an integrated computational pipeline for clinical decision support and identify key challenges for future translation.

Keywords: Computational Fluid Dynamics, Fluid–Structure Interaction, Hemodynamics, Wall Shear Stress, Reduced-Order Modeling, Digital Twin, Cardiovascular Biomechanics

I.INTRODUCTION

Cardiovascular diseases (CVDs) continue to be a major cause of death worldwide, necessitating advanced computational modeling to improve diagnostic and therapeutic approaches. Numerical analysis of cardiovascular fluid mechanics provides quantitative insight into flow dynamics, pressure distributions, and mechanical stresses that are inaccessible through clinical imaging alone. Recent reviews have highlighted the deep integration of fluid dynamics, clinical medicine, and computer simulation techniques in this multidisciplinary field.

This paper focuses on four cutting-edge numerical methodologies: fluid–structure interaction (FSI) modeling of compliant vessels, machine learning–driven reduced-order models (ROMs) for real-time hemodynamics, multiphysics frameworks for disease progression, and turbulence modeling for high-Reynolds-number flows. We emphasize works published in 2025–2026, ensuring applicability to current research and clinical practice.



II. FLUID–STRUCTURE INTERACTION: THE FOUNDATION OF COMPLIANT VASCULAR MODELING

Blood flow in large arteries induces substantial wall deformation, with aortic wall motion reaching up to 7.9 mm. Traditional computational fluid dynamics (CFD) with rigid wall assumptions fails to capture bidirectional mechanical coupling, motivating high-fidelity FSI approaches.

Immersed Meshless Methods :

A totally meshless FSI approach based on smoothed particle hydrodynamics (SPH) has been proposed and experimentally validated for cardiovascular applications. The method employs a unified physics representation for both blood and deformable walls, avoiding explicit FSI interfaces. Validation against a new benchmark involving pulsatile flow interacting with a chamber with deformable curved walls demonstrated good agreement in fluid velocity fields and structural deformation, establishing the approach as reliable for complex cardiovascular modeling.

Viscoelastic FSI for Compliant Arteries :

A comprehensive mathematical model of a compliant artery incorporates a Jeffreys-type non-Newtonian fluid within a viscoelastic tube surrounded by external tissue. The arterial wall deformation is modeled using the Green–Rivlin constitutive law, accounting for memory kernel and nonlinear viscoelastic effects. Two-dimensional numerical experiments reveal that increasing vessel radius and stretch ratio enhances damping and energy dissipation at lower frequencies, while higher stiffness and relaxation times amplify oscillations. Compared to Newtonian and Maxwell models, the Jeffreys model more accurately captures physiological energy dissipation and dispersive wave behavior in the cardiovascular system.

Comparison of One-Way and Two-Way Coupling :

A study using COMSOL Multiphysics to model the aorta and its main branches with hyperelastic walls found that two-way coupling improved wall stress estimates by 30% compared with one-way coupling. Under extreme hypertension (160 mmHg), the model predicted maximal displacement of 4.10 μm , with mechanical stress concentrated in disease-prone areas.

Patient-Specific FSI for Coronary Arteries :

Ten healthy left coronary artery (LCA) geometries were used to develop patient-specific FSI models. The FSI model-calculated virtual fractional flow reserve (vFFR) showed good agreement with clinical HeartFlow reports. Wall stresses were below rupture thresholds, though variations in von-Mises stress and strain were observed along the arterial length.

III. MACHINE LEARNING–AUGMENTED REDUCED-ORDER MODELING:

High-fidelity CFD simulations remain computationally expensive, limiting clinical adoption. Data-driven ROMs and machine learning (ML) offer substantial speed-up while maintaining accuracy.

POD–Neural Network ROM for WSS Prediction:

An integration of proper orthogonal decomposition (POD)–based ROM with neural network models was developed to predict wall shear stress (WSS) in peripheral arterial disease and aortic dissection. Trained on limited clinical data (three flowrate waveforms for PAD, two for AD), the models achieved computational speed-up ratios on the order of $\sim 10^4$. The flowrate-coefficients mapping model outperformed the autoregressive variant, demonstrating clinical viability for fast WSS evaluation.



ML-Enhanced 0D Models for Bifurcation Flows:

Standard 0D reduced-order models assume Poiseuille flow, losing accuracy at vessel bifurcations. A novel resistor–resistor–inductor (RRI) model uses neural networks to predict pressure–flow relationships from bifurcation geometry, incorporating linear and quadratic resistances with inductive effects. Validated across Reynolds numbers from 0 to 5,500, the RRI method reduced inlet pressure errors from 54 mmHg (45%) for standard 0D models to 25 mmHg (17%), with particular effectiveness at high Reynolds numbers and in extensive vascular networks.

ML Framework for Non-Invasive Physiology Prediction:

A machine learning framework trained on CFD-simulated bifurcation lesions predicts instantaneous wave-free ratio (iFR) and fractional flow reserve (FFR) using only anatomical features, enabling non-invasive, personalized planning of bifurcation interventions. This approach demonstrates how ML, grounded in physics-based modeling, can enhance clinical decision-making.

IV. MULTIPHYSICS INTEGRATION: HEMODYNAMICS, TISSUE MECHANICS, AND PATHOPHYSIOLOGY:

Atherosclerotic plaque progression involves coupled hemodynamic, biomechanical, and biological processes that cannot be captured by single-physics models.

Agent-Based Modeling with FEA and CFD:

A novel multi-scale, multi-physics simulation (CAFe) merges hemodynamics via CFD, biological processes via agent-based modeling (ABM), and biomechanics via finite element analysis (FEA) within a single framework. A shared volumetric 3D tetrahedral mesh ensures geometric continuity. The CFD and FEA modules calculate WSS and structural stress, while the ABM simulates vascular remodeling using molecular diffusion, cell migration, and volumetric growth. Initial results indicate atherosclerotic arteries maintain a hemodynamic threshold through preferential growth and remodeling downstream of a stenosis.

LDL Transport and Temperature Distribution:

FSI models have been extended to predict heat and low-density lipoprotein (LDL) transfer in healthy aortas. Correlation coefficients between rigid and hyperelastic aortic wall models exceed 0.914 for time-averaged LDL, temperature, and WSS-related indices. Positive correlation (>0.596) between LDL concentration and aortic wall temperature was observed, and long relative residence time (RRT) regions coincide with high LDL areas.

V. TURBULENCE MODELING AND UNCERTAINTY QUANTIFICATION:

High-Reynolds-number flows in stenotic vessels exhibit transitional and turbulent regimes requiring specialized numerical methods.

Large-Eddy Simulation of Healthy Thoracic Aorta:

Large-eddy simulation (LES) with the wall-adapting local eddy-viscosity (WALE) model revealed that turbulence originates in the aortic arch following peak systole and further develops during mid-deceleration and end-systole, with maximum turbulence intensity exceeding 25%.

WSS reached up to 30 Pa during peak systole, with low and oscillatory WSS during diastole potentially contributing to aortic dissection and atherosclerosis.



Uncertainty Quantification for Stenotic Flows:

A generalizable uncertainty quantification framework for lattice Boltzmann method simulations of high-Reynolds-number vascular flows was demonstrated on a patient-specific stenosed aorta. The framework combines EasyVVUQ for parameter sampling with LES turbulence modeling, executing ensembles on the Frontier exascale supercomputer.

Inlet velocity variation produces high uncertainty downstream of the stenosis where turbulence develops, while upstream regions remain stable.

VI. PATIENT-SPECIFIC CLINICAL APPLICATIONS:

Aortic Dissection:

A highly scalable parallel FSI solver for aortic dissection achieved a full cardiac cycle simulation in 0.36 hours on 2,304 processor cores. Patient-specific workflows combining compliant CFD with MRI data have been developed to assess TBAD haemodynamics and guide virtual surgery.

Coronary Artery Stenosis:

CFD simulations of stenosed coronary arteries using non-Newtonian blood-mimicking fluid revealed that pulse rate increases lead to elevated pressure drop, pump output pressure, and power consumption, with effects more pronounced in stenosed arteries. Fractional flow reserve analysis confirmed that 65% luminal narrowing poses significant hemodynamic risk, with highest WSS values localized in the stenotic region.

Cerebral Aneurysm:

Patient-specific CFD simulations of middle cerebral artery aneurysms demonstrated that sac volume growth elevates hemodynamic stress markers, with peak WSS exceeding 120 Pa at intermediate sac sizes. Endovascular coiling reduced peak WSS to below 100 Pa and OSI to below 0.03, providing effective stabilization. Hematocrit-based non-Newtonian modeling further refined rupture risk assessment, revealing that elevated hematocrit increases rupture risk at flow impingement sites while lower hematocrit raises risk along side walls.

Aortic Stenosis:

High-fidelity FSI simulations of non-uniformly calcified aortic valves demonstrated that non-uniform calcification alters flow physics, leading to 35–50% increases in maximum jet velocity and 150–170% rises in transvalvular pressure gradient compared to normal valves. Leaflet-resolved CFD studies revealed non-linear increases in velocity and WSS with stenosis severity, with peak jet velocities reaching 4.7 m/s and peak WSS 122 Pa in severe stenosis.

VII. TOWARD AN INTEGRATED CLINICAL PIPELINE:

The convergence of FSI, ML-enhanced ROM, multiphysics integration, and turbulence modeling enables patient-specific digital twins for cardiovascular disease management. We propose a five-stage pipeline:

- Image Acquisition and Segmentation: Reconstruct patient-specific geometries from CTA or MRI.
- Boundary Condition Specification: Incorporate clinical measurements (blood pressure, flow rates, hematocrit) and physiologically representative outlet models (Windkessel).
- Multiphysics Simulation: Deploy FSI with appropriate non-Newtonian rheology (Carreau, Casson, or hematocrit-based models) and LES for turbulent flows.



- Uncertainty Quantification: Evaluate parametric uncertainty using ensemble methods.
- Clinical Interpretation: Extract WSS, OSI, RRT, FFR, and structural stress maps for surgical planning and risk stratification.

VIII. CONCLUSION:

Numerical analysis of cardiovascular fluid mechanics has matured from academic exploration to clinically applicable technology. Immersed meshless FSI methods capture the bidirectional coupling between blood flow and arterial wall motion. ML-enhanced ROMs provide real-time hemodynamic predictions with (10^4) -fold speed-ups. Multiphysics frameworks integrate hemodynamics, tissue mechanics, and biological processes for disease progression modeling. Large-eddy simulation and uncertainty quantification enable robust predictions under patient-specific variability. Future work must address remaining challenges: standardizing boundary conditions, validating against multi-center clinical data, and integrating these tools into routine clinical workflows. The foundation is laid for a new era of predictive, personalized cardiovascular medicine.

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