Dean&Francis
ISSN 2959-6157

Hydrodynamic Behavior of Blood Components in Elastic Vessels

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Abstract:

The circulatory system's intricate network of elastic vessels is governed by complex hydrodynamic phenomena that are crucial for tissue perfusion and physiological homeostasis. This review synthesizes findings from high-impact clinical observations and advanced computational modeling, focusing on the multi-physics approach of Fluid-Structure Interaction (FSI) to understand cardiovascular health and disease. The article discusses the non-Newtonian rheology of blood, pulsatile flow dynamics, and the impact of arterial stiffness on pulse wave velocity. It also explores the hydrodynamic behaviors of red blood cells, white blood cells, platelets, and plasma proteins, and how computational modeling, particularly Computational Fluid Dynamics (CFD) and FSI, provides insights into clinical applications. The review reveals that altered hemodynamics are fundamental drivers of cardiovascular disease and that computational models, especially FSI, are indispensable for accurate simulation and clinical application, highlighting the need for further research into mechanobiology and personalized medicine.

Keywords: Hemodynamics, Fluid-Structure Interaction (FSI), Computational Modeling, Cardiovascular Disease, Personalized Medicine

1. Introduction

The circulatory system, a marvel of biological engineering, is fundamentally governed by the laws of physics. The flow of blood through the intricate network of elastic arteries, veins, and capillaries is a complex hydrodynamic phenomenon that dictates tissue perfusion, oxygen delivery, and overall physiological homeostasis. The central point is that a multi-physics approach, integrating fluid dynamics, cellular biology, and material science—encapsulated by the concept of Fluid-Structure Interaction (FSI)—is essential for elucidating the mechanisms of cardiovascular health and disease. As evidenced by numerous studies, from epidemiological analyses in The New England Journal of Medicine to detailed mechanistic reviews, the physical forces exerted by blood flow are not passive consequences but active participants in vascular biology and pathology [1].

This review explores the foundational principles of hemodynamics, then delve into the specific behaviors

of red blood cells, white blood cells, platelets, and plasma proteins. Subsequently, it examines the state-of-the-art computational methodologies that are transforming our ability to study these phenomena. This paper provides reference values for conventional mechanical properties and calculates the constitutive parameters of nonlinear elastic and viscoelastic models.

2. Fundamental Principles of Hemodynamics in Elastic Vasculature

Blood is a complex multi-phase system comprising cells suspended in plasma, classified as a non-Newtonian fluid due to its unique flow properties. A critical feature is its shear-thinning behavior (Figure 1), where viscosity decreases with increasing shear rate, influenced by red blood cells (RBCs). In low shear environments, RBCs form rouleaux, increasing viscosity, while in high shear environments, they disperse and align, reducing viscosity. Blood viscosity is determined by factors like hematocrit and RBC properties [2]. Traditional Newtonian models are insufficient in representing blood flow conditions, particularly in pathological states. Advanced non-Newtonian models, like the Williamson and Carreau-Yasuda models, are essential for accurate simulations [3].

Fig. 1 Shear-Thinning Behavior of Blood [4]

Blood flow is pulsatile, driven by heart contractions, which creates pressure and flow waves that affect velocity profiles and induce fluctuations in Wall Shear Stress (WSS). The Womersley number indicates the relationship between flow frequency and viscous effects, influencing velocity shape. In complex flow areas like arterial bifurcations, WSS direction can reverse, measured by the Oscillatory Shear Index (OSI), a marker of flow disturbance linked to endothelial dysfunction and atherosclerosis [5]. Arterial stiffness raises pulse pressure, compromising organ perfusion and increasing cardiovascular risks. Research indicates that pulsatile flow enhances blood viscoelasticity and oxygen delivery compared to non-pulsatile flow [6].

Arteries function as flexible tissues rather than rigid conduits, engaging in Fluid-Structure Interaction (FSI), where blood flow forces interact with the vessel wall's mechanical responses. This elasticity allows arteries to store energy during systole and maintain blood flow during diastole, exemplified by the "Windkessel effect" [7]. Pulse Wave Velocity (PWV) serves as a critical indicator of this interaction; higher PWV reflects arterial stiffness and predicts cardiovascular risks. Computational models factoring in FSI provide more accurate simulations of conditions like atherosclerosis and aneurysms compared to models assuming rigid vessel walls [9].

Blood flow is classified into laminar (ordered) and turbulent (chaotic) regimes, with the Reynolds number (Re) determining their transition. In the human circulatory system, Re ranges from about 1 in small arterioles (laminar)

ISSN 2959-6157

to over 4000 in the ascending aorta during peak systole (transitional/turbulent). While most arteries maintain laminar flow, anatomical features and conditions like stenosis can introduce turbulence, resulting in higher mechanical stress on the endothelium, promoting platelet activation, and increasing thrombosis risk [10]. Recent studies have even challenged traditional views, suggesting that physiological blood flow in major arteries may exhibit key characteristics of turbulence, indicating that turbulence is more prevalent than previously thought [11].

3. Hydrodynamic Behavior of Blood Components

Understanding hemodynamics requires recognizing the unique behaviors of blood components influenced by their physical properties and vessel interactions.

Red blood cells (RBCs), the predominant blood cells, significantly impact blood rheological properties due to their deformability, which allows them to pass through narrow capillaries. Their biconcave shape, viscoelastic membrane, and low cytoplasmic viscosity contribute to this ability, measurable through techniques like ektacytometry. In circulation, RBCs mainly migrate toward the axis of the vessel, forming a cell-free plasma layer that reduces viscous drag. Their motion varies with shear rates, displaying "tank-treading" at high rates and "tumbling" at lower rates, while low shear conditions can lead to aggregate formation, increasing viscosity. Deformability impairment, as seen in sickle cell disease, heightens vascular resistance and contributes to vaso-occlusive crises.

Leukocytes are crucial immune cells that migrate from the bloodstream to inflammation sites through a process called margination, where they are pushed toward the vessel wall by red blood cells. They then undergo rolling adhesion, influenced by shear forces and mediated by selectin proteins, which allows them to detect inflammatory signals [12]. Once activated, leukocytes adhere firmly via integrins and perform diapedesis, migrating into tissue. Computational models have been instrumental in simulating this complex process, accurately depicting the "stop-and-go" motion and "tear-drop" shape of leukocytes in flow [13]. Figure 2 demonstrates the angular displacement of a leukocyte over time, highlighting its "rolling" and "jumping" motions as it interacts with the vascular wall while subjected to flow.

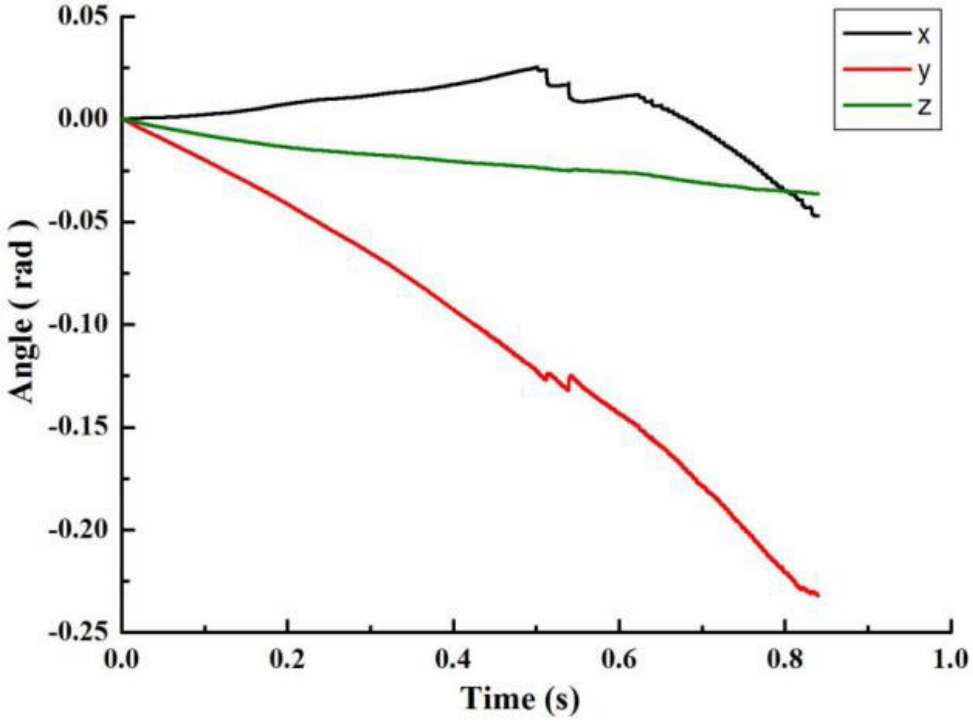

Fig. 2 The dynamics of motion of a single leukocyte due to gravity in flow chamber with fixed time step [13]

Platelets are crucial for stopping bleeding and play a role in thrombosis. Recent research highlights shear-induced activation, where high shear stresses, found in stenosed arteries or mechanical heart valves, can activate platelets directly, promoting aggregation without biochemical agonists. This mechanical activation involves conformational

changes in von Willebrand Factor (vWF), which serves as a shear sensor. This insight is vital for clinical applications since traditional antiplatelet drugs may not effectively manage shear-mediated thrombosis. Computational models utilizing Lagrangian particle tracking have advanced the ability to quantify platelet behavior and predict thrombogenicity, which is beneficial for assessing treatment outcomes for devices like flow-diverting stents [14].

Although often regarded merely as the fluid medium of blood, plasma proteins are active participants in hemodynamics. Proteins—particularly fibrinogen and globulins—are major determinants of plasma viscosity and are the primary drivers of RBC aggregation at low shear rates. Consequently, elevated levels of inflammatory proteins can increase whole-blood viscosity and act as an independent cardiovascular risk factor. Furthermore, a modern concept is the formation of a "protein corona" when proteins in plasma adsorb onto surfaces. This phenomenon is critical for the biocompatibility of medical implants and the behavior of nanomedicines; it also occurs on cell surfaces, modulating their interactions with the vessel wall. Advanced proteomic and modeling studies are now beginning to map the complex dynamics of protein transport and distribution across different biological compartments, revealing their integral role in both physiology and pathophysiology [15].

4. Computational Modeling: From Virtual Vessels to Clinical Insights

The complexity of hemodynamics makes direct in vivo measurement of many key parameters (such as WSS) impossible. Computational modeling has emerged as an indispensable tool, enabling researchers and clinicians to create "virtual vessels" for studying blood flow in unprecedented detail.

4.1 Computational Fluid Dynamics and FSl in Practice

Computational Fluid Dynamics (CFD) is a powerful technique that numerically solves the governing equations of fluid mechanics within a 3D anatomical model. These models are often patient-specific, reconstructed from clinical imaging modalities such as CT or MRI scans. While early models assumed rigid walls, the current state-of-the-art is FSI, which couples the CFD solver with Finite Element Analysis (FEA) of the vessel wall to simulate its realistic elastic or hyperelastic behavior [16]. The clinical applications of this technology are expanding rapidly. For instance, a recent study published in Surgical Endoscopy via PMC demonstrated the feasibility of using a CFD model for preoperative planning in complex pancreatic surgery. The model accurately predicted blood flow redistribution after arterial clamping, achieving 100% accuracy for one scenario and 80% for another—potentially reducing surgical complications [17].

4.2 Multiscale and Agent-Based Modeling

Standard CFD treats blood as a continuum fluid, which is insufficient for modeling discrete cellular events. To address this limitation, more advanced methods have been developed. Multiscale models bridge the vast gap between molecular-level events (e.g., receptor-ligand binding kinetics) and macroscopic cell mechanics and flow. These models can simulate how molecular bond forces translate into cellular adhesion and deformation [18]. Agent-based models, such as the Immersed Boundary-Lattice Boltzmann Method (IB-LBM), treat individual cells as discrete agents with their own properties. This enables high-fidelity simulations of dense cell suspensions, capturing the collective behavior and complex interactions of millions of RBCs and platelets—critical for understanding phenomena such as thrombus formation and microcirculatory flow [19].

5. Challenges and Clinical Implications

Significant challenges in computational modeling persist despite progress in fluid-structure interaction (FSI) and agent-based models. High costs and validation with in vivo data hinder clinical trust, while simplified boundary conditions fail to represent biochemical complexities like coagulation. Future advancements may involve machine learning and AI integration with computational fluid dynamics (CFD) for enhanced simulations. The aspiration is to develop a patient-specific, multiscale "digital twin" model to evaluate treatments and predict disease outcomes before clinical application.

The synthesis of these hydrodynamic principles and component-specific behaviors reveals a profound truth: altered hemodynamics are not merely symptoms but fundamental drivers of cardiovascular disease. Pathological flow patterns—such as low and oscillatory WSS in arterial bends, pathologically high shear in stenoses, and increased pulse wave velocity due to arterial stiffening—directly initiate and perpetuate atherosclerosis, thrombosis, and hypertension. The deformability of red blood cells plays a pivotal role in shaping the near-wall plasma layer, which is the

ISSN 2959-6157

pathway that platelets and leukocytes traverse. Simultaneously, the pulsatility of blood flow has a governing effect on the health of the endothelium, a factor that subsequently controls vascular tone and inflammation. Furthermore, the elasticity of blood vessels dictates the system's comprehensive response to the cardiac pump, as referenced in the literature20. These interconnected factors are essential in understanding the complex dynamics of the circulatory system.

This understanding opens new avenues for diagnosis and treatment. Hemodynamic parameters such as PWV are already being used as surrogate endpoints in clinical trials, and their link to hard outcomes is well-established. Future research must focus on longitudinal studies that combine patient-specific computational modeling with long-term clinical outcomes to validate predictive models. It also needs the specific mechanobiology of endothelial cells, smooth muscle cells, and blood components under dynamic, multi-factorial flow conditions. This could lead to the development of novel "mechanotherapeutics" to normalize pathological flow patterns or modulate cellular responses to mechanical stress.

6. Conclusion

In conclusion, the convergence of fluid mechanics, cell biology, and computational science has transformed our understanding of the cardiovascular system. By viewing blood flow not as a simple plumbing problem but as a complex, interactive biological system, we are moving toward an era of truly personalized medicine. In this era, treatments can be tailored to the unique hemodynamic and biological profile of each individual, ultimately enabling the prevention of disease before it manifests.

However, this study primarily relies on theoretical analysis and computational models to demonstrate how red blood cell deformability, blood flow pulsation, and vascular elasticity influence the circulatory system. However, these models and theories may require additional experimental data to validate their accuracy and applicability. Future research could enhance the reliability of existing computational models and improve their clinical applicability by designing more precise experiments to collect relevant data.

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