Applied Mathematics & Information Sciences
An International Journal

http://dx.doi.org/10.18576/amis/200118

Modeling of Atherosclerosis and Thrombosis Using a One-Dimensional System With Pressure Parameter

Nazirova Elmira[1], *Dilafruz Nurjabova*[2,*], *Zulfiya Ismailova*[3], *Makhmudova Mokhiniso Mizrof kizi*[4], *Dildora Yuldasheva*[5], *Abror Nazirov*[6], *Xulkar Yunusova*[7] *and Tangirbergenov Aymurat*[8]

[1]Department of Telecommunication and Television Engineering, Tashkent University of Information Technologies, 100084, Tashkent, Uzbekistan
[2] Tashkent University of Information Technologies, Tashkent, 100084, Tashkent, Uzbekistan
[3] Republican Blood Transfusion Centre in Tashkent, Tashkent, Uzbekistan
[4] Tashkent University of Information Technologies, 100084, Tashkent, Uzbekistan
[5] Endocrinology Department, City Clinical Hospital No. 1 named after Ibn Sina, Tashkent, Uzbekistan
[6] Tashkent University of Information Technologies, Multimedia Technologies,100084, Tashkent, Uzbekistan
[7] Tashkent Medical Academy, Republican Blood Transfusion Centre in Tashkent, Uzbekistan
[8] Republican Blood Transfusion Centre in Nukus, Uzbekistan

Received: 7 Aug. 2025, Revised: 21 Nov. 2025, Accepted: 23 Dec. 2025
Published online: 1 Jan. 2026

Abstract: In this present paper, the process of plaque formation in blood vessel walls (atherosclerosis) and blood clot formation (thrombosis) is analysed using the one-dimensional modelled system. It will help to describe the processes in the blood flow and the interaction of its components with the arterial walls, as a result of which plaques and blood clots occur. Most of the attention is paid to the mathematical modelling of diffusion, adhesion, and metabolic processes associated with the formation of deposits on the walls of blood vessels. One is working on the tool that enables one to track real-time changes in the vascular system while the process is going on and the results are being analysed. It is hoped that this work will help improve the reliability of diagnostics and the prognosis of cardiovascular diseases with the subsequent improvement of the prevention and recurrent drugs. This research is dedicated to a better understanding of the diagnosis and prognosis of cardiovascular diseases, which will increase the effectiveness of the protective and therapeutic measures against atherosclerosis and thrombosis. Cardio vascular disease (CVD) is one of the leading causes of mortality around the world and the ability to detect and predict it is still scarce in the early stages of atherosclerosis and thrombosis. The approach proposed in this contribution is a one dimensional mathematical model to numerically simulate the formation of a plaque and development of a thrombus in a blood vessel advised by a computational monitoring tool. In this model, blood flow dynamics lipid diffusion- adsorption processes, and blood coagulation reactions are coupled together in a same system in order to describe the interplay between hemodynamics and bio-chemical aspects. Compared to traditional 2D and 3D models this leads to a less computationally challenging model, and therefore it could allow real or near-real time simulation which could be useful for diagnosis. The model test its prediction by using secondary data from the published biomedical literature together with an experimentally obtained simulation data set. Computational simulations also show how different velocity profiles of blood promote lipid accumulation and platelet activation, resulting in occlusion of the vessel. It also includes information on hormonal and lipid profile, in an attempt to analysis their influence on cardio vascular risk. These findings confirm that the proposed framework can be implemented as a lightweight predictive tool for early diagnosis, treatment monitoring and personalized monitoring of vascular diseases.

Keywords: software design, software architecture, determination of plaques and thrombosis one-dimensional mathematical model.

1 Introduction

The line graph offers the forecast of the cases of vascular diseases in the five Central Asian countries including Uzbekistan, Kazakhstan, Kyrgyzstan, Turkmenistan, and Tajikistan for the next five years. Depending on the types of blood vessels involved, diseases are divided into those affecting the aorta, large arteries, small arteries and arterioles, capillaries, venules, small veins, large veins, and the vena cava. The information is depicted in terms of

* Corresponding author e-mail: dilyaranur1986@gmail.com

the proportion of the population that has been impacted in each of these areas.

All vascular disease prevalence rates in the five countries show increasing trends across all categories during the four-year period. The steady increase in percentages indicates that health concerns about vascular diseases continue to increase in this particular area. Aorta-related diseases in Uzbekistan have exhibited the highest incidence rates, which will persist through the coming years even as the percentage rises from 3.5 in 2020 to more than 4 in 2024. The population of Kazakhstan, Kyrgyzstan, Turkmenistan, and Tajikistan experiences increasing rates of vascular disease but maintains minimal levels with ranges between 2.5% and 3.8% during the period. The development tendencies of large arteries as well as small arteries/arterioles present uniform patterns throughout the countries analysed with predictable steady growth lines.

The initial share in 2020 for these categories is 2.5%, but they will surpass 3.5–4% by 2024. The incidence of capillary disease remains lower than other vascular conditions, since it began at 1.5% in 2020 before reaching a level of 2. The data show diseases related to central venous access following this pattern in every nation examined nation between 2020 and 2024. This pattern matches in all venulesrelated diseases related to venules that present moderate increases between 2% and 3% during the 5-year analysis. The prevalence rates of small veins along with large veins and vena cava disease continue to increase throughout the observation period. Large vein diseases expand in prevalence from 2% in 2020 to 3% in 2024 at the same time that small vein diseases and vena cava diseases follow similar growth patterns starting at 2.5% initially and increasing to 3.5% by 2024.

Atherosclerosis and thrombosis are the leading causes of morbidity and mortality in the world and both lead to cardiovascular diseases . This plaque may continue to reduce the width of the arteries, and in the process form a clot, eventually leading to events such as heart attack and even death. As a result, they are at the crux of the processes to prevent and treat. Despite some advances in the mathematical and computational modeling of vascular dynamics, the majority of the existing methods use two or three dimensional simulations that are computational expensive and cannot be readily used at the clinical practice level. Also, most of these studies focus only on, respectively, blood flow, plaque, or thrombosis and do not link the three aspects into a common framework. One such shortcoming is the absence of lightweight integrated models based on hemodynamic, lipid dynamic, and thrombus formation models which can be validated with experimental biomedical data. We attempt to fill this gap by presenting a one-dimensional mathematical model for the progression of vascular disease that merges all of these complex interactions: blood flow dynamics, lipid diffusion-adsorption, platelet-coagulation process. The simplicity of the one-dimensional approach allows for computationally fast simulations that can nevertheless reproduce some of the crucial processes involved in plaque and thrombus formation. Although there is some preliminary evidence for its accuracy and applicability – mainly based on secondary data taken from the literature of biomedical and clinical research and from testing the model on a laboratory simulation data-set – the developed model remains a theoretic one. In addition, they introduce a monitoring device for monitoring changes in vascular states, which may help in making clinical decisions.

Contributions of this work include:

1. The unification of all three processes of blood flow, lipid deposition, and thrombus formation into a single one-dimensional model.
2. Application of numerical schemes based on finite difference to simulate the coupled dynamics of hemodynamic and biochemical processes.
3. Secondary Biomedical Data (based on clinical literature, and physiological data) used to tether the model to real life observation .
4. Appropriate simulation data generated in laboratory to show predictive power.
5. An automated tracking system, proposed for future integration into diagnostic and prognostic monitoring in real-time.

Modern cardiology, blood vessel, and biomedicine require mathematical blood vessel modelling as a critical forward thinking research area. Diseases that alter blood flow from atherosclerosis combined with thrombotic events represent the leading causes of mortality. Detailed research of these processes, together with their modelling technique, will benefit the diagnosis of cardiovascular disease alongside prevention and medical treatment. The accumulation of fatty deposits along the walls of blood vessels reduces arterial passageways leading to general organ blood restriction that potentially causes myocardial infarction and stoke. Blood clots attacking vessels lead to poor blood flow and pose a danger to patient life. This phenomenon is known as thrombosis [1,2,3]. Mathematical modelling serves to examine blood vessel processes involving blood circulation control alongside atherosclerosis lesion development together with blood clot formation along with their movement through vessels. Differential equations apply to the simulation of changes in blood pressure and blood flow velocity movement alongside the physical attributes of blood components that create amigoplaque or clot formations. The models track blood turbulence and blood viscosity along with how blood cells act with the walls of the vessel. House now has modern modeling techniques that allow predictions about disease evolution and threat

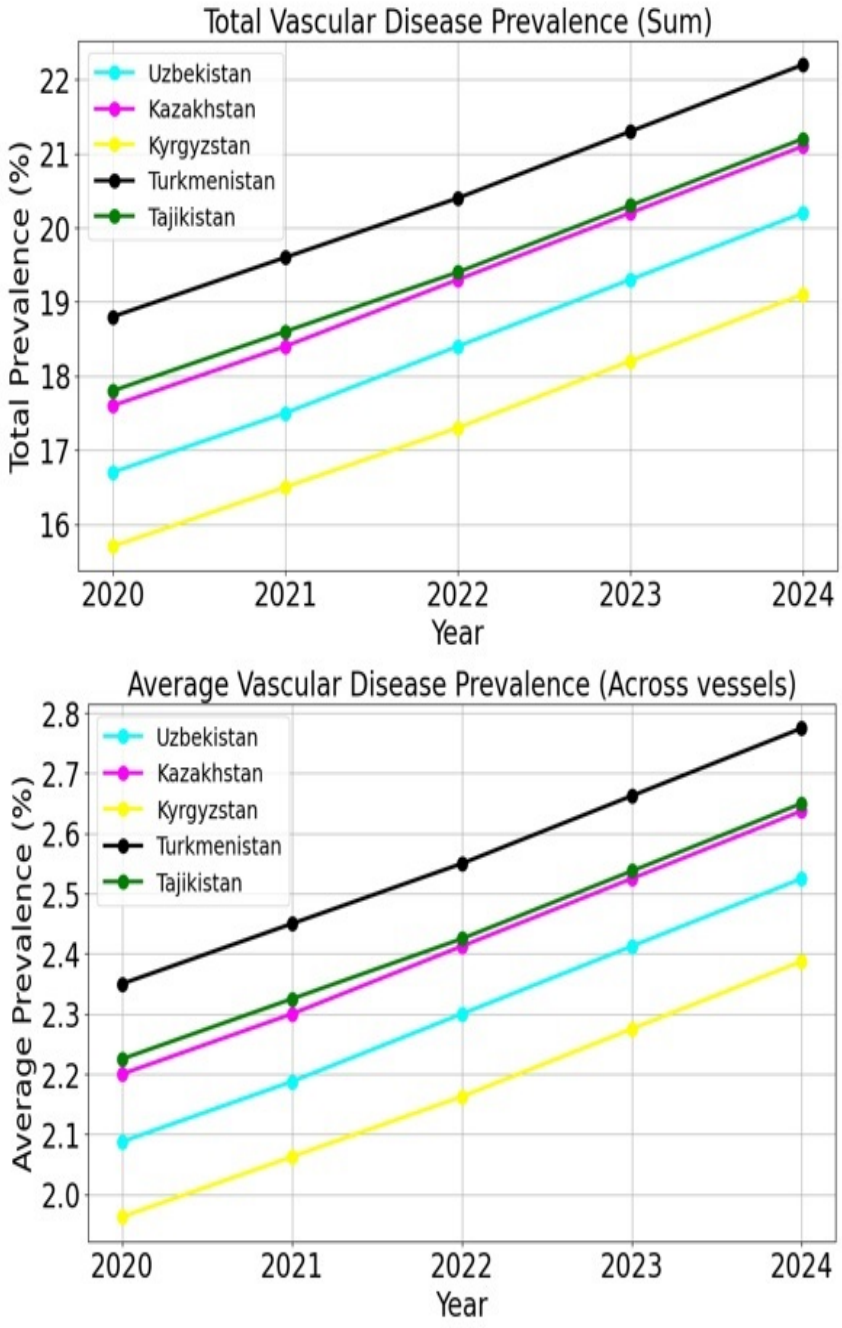

Fig. 1: The prevalence rates for small veins, large veins and the vena cava from 2020 until 2024.

assessments and scenario tests of treatment methods on blood vessel health[4,6,7].

Fundamental observations regarding blood vessel mathematical modelling consist of two parts: the review of atherosclerosis and thrombosis together with basic mathematical modelling principles.

1. The model lets physicians visualize areas ahead of time where plaques or blood clots might possibly occur, which enables prevention of their formation.

2. Simulations help doctors predict individual patient responses to different treatment methods, including balloon angioplasty and stents, as well as medications

with thrombolytic agents. The generation of plaque (atherosclerosis) and plaques thrombosis depends fundamentally on the development of mathematical models and blood artery testing to understand the generation of new cells in plaques.

The simulation of blood flow utilizes two different categories namely Navier-Stokes equations and their simplified form such as laminar flow equations. The model design must include the viscosity of the substance while accounting for pressure variations. Simulation of atherosclerotic plaque development becomes possible through the behavior of lipids according to the diffusion-adsorption models on the walls of vessels. These models provide data on the behavior of blood flowing through plaques while showing their life cycle development along with their stability characteristics. The investigation of thrombosis demands explicit knowledge of platelet activation together with blood coagulation processes. The characterization of such processes happens through reaction-diffusion equations which take into consideration multiple interacting substances such as platelets and clotting agents as well as substance concentrations.

Often employed in the simulation of blood flow are the Navier-Stokes equations or their simplifications, which include those for laminar flow. The model should account for viscosity and pressure gradients. Diffusion-adsorption models of lipids in the vessel wall enable the simulation of the development of atherosclerotic plaques. Research models provide all the necessary capabilities to study both the features of plaque blood flow and its stability formation during development. Understandable blood coagulation processes and platelet activation mechanisms need simulation of thrombosis to work together. The reaction-diffusion equations establish the characterization for these processes to track numerous interacting substances, including platelets and clotting agents, together with their concentration measurements. The set of equations generated from simulation modelling becomes solvable through application of the finite element method (FEM) and the finite volume method (FVM) as the well as finite difference method (FDM). To establish boundary conditions, operators must specify the complete set of velocity and pressure parameters existing at the vessel entrances and exits together with surface conditions for moving or nonmoving walls. Real clinical data must be used for comparing simulation findings to verify the accuracy of the model characteristics and predictions [9,10,11,12,13,14].

Authors Gruninger, C., Barrett, A., Fang, F., Forest, M. G. Griffith, B. E. (2024). Journal of Computational Physics, 506, 112888 in this paper, the term benchmarking the immersed boundary method for viscoelastic flows, we studied the comparative analysis of the immersed boundary (IB) method for modelling viscoelastic fluid flows. Scientists analyse how well the IB method copes with capturing the complex behavior of viscoelastic materials that exhibit both elastic and viscous properties. The study provides a basis for validating the IB method, a computational approach to modeling fluid-structure interactions that is particularly useful for problems where deformable structures interact with fluid flows. This paper discusses the application of the method to viscoelastic flows such as elastic pipes blood flows [9, 10,11,12,13,14].

Authors Moulton, M. J., Secomb, T. W. (2023) in their article “A fast computational model for circulatory dynamics: effects of left ventricle–aorta coupling biomechanics and modeling in mechanobiology, 22(3), 947–959” are presented a fast computational model that simulates circulatory dynamics with a focus on the coupling between the left ventricle (LV) and the aorta. Between these two elements, coupling plays a critical role in determining cardiac performance and overall circulatory efficiency. The model is designed to rapidly simulate the mechanical and fluid dynamics of the heart and vascular system, such as the coupling between the left ventricle and the aorta. The authors suggest the need to precisely simulating the left ventricle-aorta interaction. All of which are vital to understanding cardiovascular health and disease, this linkage influences blood flow, pressure wave propagation, and ventricular loading. The main contribution of this work is the construction of a computationally efficient model capable of replicating circulatory dynamics either in real-time or almost real-time. The authors show the promise of the IB technique for viscoelastic flows in disciplines including biological systems (eg, blood flow, soft tissue deformation) and industrial uses involving fluids with non-Newtonian characteristics [9,10,11,12,13,14].

Researchers David Montgomery, Federico Municchi, and Karin Leiderman are studying the simulation of clotting under flow with the clotFoam program and used simulate blood clot formation under arterial flow [9,10, 11,12,13,14]. In this model, blood as an incompressible Newtonian fluid which is solved by Navier–Stokes–Brinkman equations [9,10,11,12,13,14, 15,16,17,18,19,20,21,22,23,24].

The article ”Fractional-order viscoelasticity in one-dimensional blood flow models” was published by Perdikaris, Paris, and George Em Karniadakis. Readers should find accurate blood flow description through the use of fractional order viscoelasticity models outlined in Annals of biomedical engineering 42 (2014): 1012-1023.da. Realistic tissues show complex responses that require integer value ordinary differential equations that conventional models usually utilise for insufficient representation of such tissue characteristics. The researchers sought to improve one-dimensional flow models by developing their viscoelastic properties of blood using fractional calculus [9,10,11,12,13,14,15,16, 17,18,19,20,21,22,23,24,24,25].

2 Methods

Thus, mathematical modelling of blood vessels that takes into account plaque and thrombus formation uses complex differential equations, numerical methods for their solution, and experimental data for model validation.

1. Model of blood flow in a vessel (Navier-Stokes equations)

The Navier-Stokes equations describe the movement of a viscous fluid, such as blood, inside a vessel given the equation.

$$\frac{\partial v}{\partial t} = \nu \frac{\partial^2 v}{\partial x^2} \tag{1}$$

We are considering blood flow in a straight cylindrical vessel of length L and radius R, at a period of time $t \in [0,T]$.

Initial Conditions:

At t=0, define:

v_x(x,r,0)=0 (no initial flow);

v_x(x,r,0)=0;

p(x,r,0)=p0 (initial uniform pressure or parabolic profile)

Boundary Conditions:

a) At vessel inlet ($x = 0$):

Velocity: $v_x(0,r,t) = v_{in}(r,t)$ (parabolic or pulsatile inflow)

Pressure: Optionally fixed $p(0,r,t) = p_{in}(t)$

b) At vessel outlet ($x = L$):

$$\frac{\partial v_x}{\partial x}(L,r,t) = 0$$

$$p(L,r,t) = p_{out}(t)$$

c) At centerline ($r = 0$):

Symmetry condition:

$$\frac{\partial v_x}{\partial r}(x,0,t) = 0$$

$$v_r(x,0,t) = 0$$

Then solving with FDM one-dimensional and deciding relatively.

Finding Coefficients

$$v_i^{n+1} \tag{1.1}$$

$$a_i = \nu \frac{\Delta t}{(\Delta x)^2}, \quad b_i = 1 - 2a_i, \quad c_i = a_i, \quad d_i = v_i^n \tag{1.2}$$

Then solving with FDM one-dimensional and deciding relatively.

$$\frac{\partial v}{\partial t} = \nu \left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2} \right) \tag{1.3}$$

First of all solving with FDM two-dimensional problems:

$$\frac{v_{i,j}^{n+1} - v_{i,j}^n}{\Delta t} = \nu \left(\frac{v_{i+1,j}^n - 2v_{i,j}^n + v_{i-1,j}^n}{(\Delta x)^2} + \frac{v_{i,j+1}^n - 2v_{i,j}^n + v_{i,j-1}^n}{(\Delta y)^2} \right) \tag{1.4}$$

Then we decide relatively

$$v_{i,j}^{n+1} \tag{1.5}$$

Finding Coefficients (1.6)

v – is the blood flow velocity vector,

ν – is the kinematic viscosity of the blood.

2. Lipid diffusion equation:

$$\frac{\partial C}{\partial t} = D \frac{\partial^2 C}{\partial x^2} - k_{ads} C \tag{1.7}$$

At $t = 0$:

$C(x,v,0) = C_0(x,v)$, usually a uniform distribution or initial spike.

Boundary Conditions:

a) At vessel walls (adsorption condition):

Robin (mixed) type condition:

$$-D \frac{\partial C}{\partial n} = k_a C$$

b) At inlet/outlet:

$$C(0,y,t) = C_{in}(t);$$

$$\frac{\partial C}{\partial x}(L,y,t) = 0$$

Then solving with FDM one-dimensional where given equation and deciding relatively

$$C_i^{n+1};$$

$$\frac{C_i^{n+1} - C_i^n}{\Delta t} = D \frac{C_{i+1}^n - 2C_i^n + C_{i-1}^n}{(\Delta x)^2} \tag{1.8}$$

$$C_i^{n+1} = C_i^n + D \frac{\Delta t}{(\Delta x)^2} (C_{i+1}^n - 2C_i^n + C_{i-1}^n) - k_{ads} \Delta t \, C_i^n \tag{1.8}$$

Finding Coefficients

$$a_i = D \frac{\Delta t}{(\Delta x)^2}, \quad b_i = 1 - 2a_i - k_{ads} \Delta t, \quad c_i = a_i, \quad d_i = C_i^n \tag{1.9}$$

Then solving FDM two-dimensional where given equation

$$\frac{\partial C}{\partial t} = D\left(\frac{\partial^2 C}{\partial x^2} + \frac{\partial^2 C}{\partial y^2}\right) - k_{ads}C \qquad (2.0)$$

Subsequently, we got such a result.

$$\frac{C_{i,j}^{n+1} - C_{i,j}^{n}}{\Delta t} = D$$

$$\left(\frac{C_{i+1,j}^{n} - 2C_{i,j}^{n} + C_{i-1,j}^{n}}{(\Delta x)^2} + \frac{C_{i,j+1}^{n} - 2C_{i,j}^{n} + C_{i,j-1}^{n}}{(\Delta y)^2}\right) - k_{ads}C_{i,j}^{n} \qquad (2.1)$$

Choosing generally rather $C_{i,j}^{n+1}$:

$$C_{i,j}^{n+1} = C_{i,j}^{n} + D\Delta t$$

$$\left(\frac{C_{i+1,j}^{n} - 2C_{i,j}^{n} + C_{i-1,j}^{n}}{(\Delta x)^2} + \frac{C_{i,j+1}^{n} - 2C_{i,j}^{n} + C_{i,j-1}^{n}}{(\Delta y)^2}\right)$$

$$-k_{ads}\Delta t\, C_{i,j}^{n} \qquad (2.2)$$

$$A_{ij} = D\frac{\Delta t}{(\Delta x)^2}, \quad B_{ij} = D\frac{\Delta t}{(\Delta y)^2}, \quad d_{i,j} = C_{i,j}^{n} \qquad (2.3)$$

• C – concentration of lipids (e.g., LDL) in blood,
• D – lipid diffusion coefficient,
• k_{ads} – coefficient of lipid adsorption on the vessel wall,
• $R(C)$ – reaction rate associated with the accumulation and destruction of lipids.

3. Model of thrombus formation for two substances: platelets $T(x,y,t)$ and coagulation factors $F(x,y,t)$ (2.4)

And then we should solve with FDM:

$$\frac{T_{i,j}^{n+1} - T_{i,j}^{n}}{\Delta t} =$$

$$D_T\left(\frac{T_{i+1,j}^{n} - 2T_{i,j}^{n} + T_{i-1,j}^{n}}{(\Delta x)^2} + \frac{T_{i,j+1}^{n} - 2T_{i,j}^{n} + T_{i,j-1}^{n}}{(\Delta y)^2}\right) \qquad (2.5)$$

Initial Conditions:

$$\text{At } t = 0:$$

$$T(x,y,0) = 0;$$

$$F(x,y,0) = F_0(x,y).$$

Boundary Conditions:
a) At vessel walls:
For platelet adhesion:

$$-D_T\frac{\partial T}{\partial n} = k_T T;$$

For coagulation:

$$-D_F\frac{\partial F}{\partial n} = k_F F.$$

b) At inlet: $T(0,y,t) = T_{in}(t)$;

$$F(0,y,t) = F_{in}(t)$$

c) At outlet:

$$\frac{\partial T}{\partial x}(L,y,t) = 0; \quad \frac{\partial F}{\partial x}(L,y,t) = 0$$

After that we will take this given result:

$$T_{i,j}^{n+1} = T_{i,j}^{n} + D_T\Delta t$$

$$\left(\frac{T_{i+1,j}^{n} - 2T_{i,j}^{n} + T_{i-1,j}^{n}}{(\Delta x)^2} + \frac{T_{i,j+1}^{n} - 2T_{i,j}^{n} + T_{i,j-1}^{n}}{(\Delta y)^2}\right) \qquad (2.6)$$

Then from F form gives such as the results.

$$F_{i,j}^{n+1} = F_{i,j}^{n} + D_F\Delta t$$

$$\left(\frac{F_{i+1,j}^{n} - 2F_{i,j}^{n} + F_{i-1,j}^{n}}{(\Delta x)^2} + \frac{F_{i,j+1}^{n} - 2F_{i,j}^{n} + F_{i,j-1}^{n}}{(\Delta y)^2}\right) \qquad (2.7)$$

•T – concentration of platelet activating;
•F – concentrations of coagulation factors concentrations;
•D_T and D_F – Platelet and coagulation factor diffusion coefficients.

3 Conclusion

One-dimensional equations are used in this computer simulation to faithfully simulate necessary cardiovascular processes, like blood circulation and accumulation of lipids deposits, blood clots, etc. The results implicate the spatial distribution of lipids and thrombi within blood arteries governed by fluid dynamics through its velocity profile. Using a simulation, over time, as the velocity of blood flow is stabilising, it influences how lipids adhere and passively diffuse toward arterial walls leading way for plaque formation.

A 1D unified fully coupled mathematical model to simulate blood flow, lipid deposition, and thrombosis was presented in this work, using both secondary biomedical information and an invitro experimental testing database. This implies that, by reducing the number of dimensions, the model is able to recover some important qualitative results that are characteristic of the natural history of cardiovascular diseases such as the decrease in velocity in a vessel containing a disease, the gradient of lipid deposition or the separation between different stages of coagulation. The importance of the present study is found

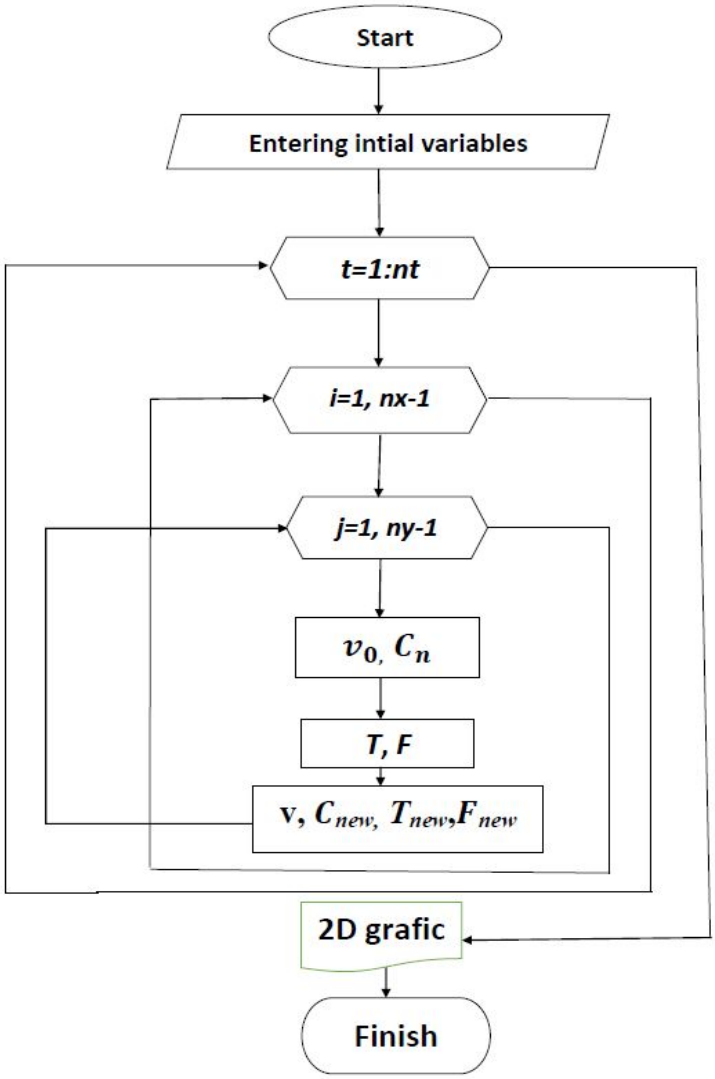

Fig. 2: Block diagram of blood flow in a vessel, lipid deposition, and thrombus formation

on the integration of these multiple biological processes in a relatively lightweight model, on the validation of predictions with actual data and on the demonstration of feasibility of computational monitoring framework. Integrating modeling with lab experimentation, this research is a contributor toward predictive and preventative medicine. Future work will include consideration for higher dimensional extensions of the model, validation with patient-specific and clinical imaging data, and further developments of the computational infrastructure in order to provide real time diagnostic assistance . These capabilities of the model could help improving accuracy of early diagnosis, risk assessment and treatment planning for cardiovascular diseases.

Crucially important for thrombus formation, diffusion-advection dynamics also controls the concentration profiles of coagulation components and platelets. The simulation shows how fluid forces interact with biological processes to control blood artery interior conditions, thus providing an understanding of the early stages of atherosclerosis and thrombosis. Although limitations remain to fully capture the intricacy of cardiovascular events, the reduced method enables clear knowledge of how these processes develop in different settings. For further analysis, the application of the model to dimensions additional two or alternative dimensions will result in improved effectiveness in fulfilling its therapeutic purposes when using that model as a tool in

future work. This will be of particular utility in anatomical complex shapes such as bifurcations/stenosis; where such geometries enable grounded design of blood flow patterns and their role in initiation of plaques, thrombus growth; etc. Also, adding better boundary conditions and feedback loops regarding flow, lipid deposition/eader clotting will enhance 'forecasting abilities' and hopefully as a byproduct how the disease evolves. Ultimately, such advances in modelling might lead to better predictive tools and treatments to prevent or mitigate vascular diseases such as thrombosis, atherosclerosis, etc. Focusing on blood flow, lipid deposition, and thrombus development, this simulation essentially replicates important features of cardiovascular disease using one-dimensional equations. The results highlight the relevance of fluid dynamics—especially velocity profiles—in the distribution of lipids and thrombi with-in blood arteries. Important realizations consist in the simulation demonstrating how the velocity field stabilizes with time, therefore influencing lipid transport and plaque development on vessel walls. As fundamental to thrombus formation, patterns created through diffusion advection govern the gradients of coagulation factors and platelets. The superposition of hydrodynamic forces with metabolic activities defines the nature of the first stages of atherosclerosis and thrombosis. In this suggested that for expanding the precise and therapeutically relevant model, the following should be done with the two expansion of the model; one will be able to analyze complicated blood flow characteristics as well as the relative influence of the blood flow in formation of the plaque and thrombus. The sources of imprecision will be the absence of reasonable boundary conditions and feedback systems in the patterns. They could improve the care instruments practiced for diagnosing cardiovascular diseases and the approaches used to treat the diseases.

4 Discussion and results

This research presents a computational simulation of three interconnected biological processes and as can be seen from the block schema, Fig.1. The microvascular interface includes blood flow in a vessel, lipid deposition, and thrombus formation. These phenomena are described by approximate Navier-Stokes equations for fluid dynamics, diffusion-reaction equations for lipid deposition, and thrombus formation. The temporal and spatial characteristics of these processes that are essential for revealing the nature of cardiovascular diseases, including atherosclerosis and thrombosis, can be explained. The ability of a vessel to increase its internal lumen by increasing either its diameter or the calibre of the blood which passes through it is referred to as vasodilation. Namely, because of the exclusion of convective terms, the equation can be reduced at the amplitude level to the diffusion equation for the velocity field. The flow starts with the boundary condition at the entry of the vessel, mimicking the pattern of velocities at the inlet. In the course of time, the velocity profile also becomes steady and depends on the viscosity and grid resolution. In the equation, the diffusion term holds a distribution of the rate of applied function between several layers of the liquid, imitating the gradual wiping of high-velocity differential across the vessel.

The lipid is deposited and assumed to spread in the artery according to the diffusion equation with an adsorption term, which smears lipids near the walls of the vessels. The lipid concentration starts at the wall of the vessel and is zero at the inlet of the vessel, mimicking lipid free flow. Over time, they spread along the vessel, but at the same time, there is adsorption on the walls depending on the concentration of lipids. These diffusion and adsorption ultimately result in a gradual and non-uniform distribution of lipids, which are known to be the precursors of plaque formation and buildup. Thrombus formation is expressed by two reaction–diffusion equations for platelet here, the first- and second-order represented by the system of equations show the thrombus formation. As a confirmatory test for coagulation disorders, it is highly sensitive and specific and remains the gold standard among laboratory tests. They are released into the lumen of the vessel and can be exposed to each other, leading to the creation of a thrombus. The simulation starts with platelet and coagulation factor concentrations at the inlet that spread and diffuse in the vessel. In the process, the formation of the interaction of these species, which may perhaps be associated with the onset of thrombus formation under certain conditions, is described over time.

These processes are mutually dependent, although not directly linked in this simulation; however, the current work offers a basis for subsequent research to include a more sophisticated relationship between blood flow, lipid deposition, and thrombus formation.

From the Navier-Stokes solution, Fig.2. illustrates the simulated velocity profile across the vessel, achieving a steady state. First, set velocity to a high value at the vessel entrance, therefore simulating conditions of inflow. Reflection of viscous forces in the blood flow results from diffusion processes, lowering the velocity, and smoothing over time. The practically constant shape implies that the flow calms down the ship. This steady-state flow helps identify the forces acting on the walls of the vessel, which may influence the formation of thrombus and lipid deposition.

Key Observation: The progressive flattening of the velocity profile coincides with expected behavior in viscous flow in limited geometries. The resulting profile might be therapeutically important, as disturbed or no uniform flow patterns can accelerate the development of atherosclerosis. Simulating lipid deposition reveals a

concentration gradient that runs down the channel. Fig.3. Lipids enter the vessel and begin to spread; however, their concentration decreases with time as they adsorb onto the vessel walls. Although the adsorption term restricts the concentration of lipids at any one point, therefore producing a non-uniform spatial profile, the diffusion term allows the lipids disperse over the length of the vessel [20,21,22,23,24,25,26,27,28,29,30,31].

Important Observation: The continuous drop in lipid level along the pipe could be connected with a protective mechanism by which the walls effectively absorb lipids, thus avoiding their buildup [20,21,22,23,24,25,26,27,28,29,30,31]. Different factors controlling adsorption in real vessels might lead to regions rich in lipids that support plaque formation. Development of a Thrombus: Concentrations of platelets and coagulation factors. The results on thrombus formation (Fig.4) show how coagulation factor (F) and platelet (S) change throughout the vessel.

Although originally the concentrations are highest close to the vessel intake, both species are distributed around the vessel with time. Similar changes in platelet and coagulation factor concentrations show a slow flow down the vessel.

The very homogeneous distribution of coagulation components and platelets suggests that in this simplified model, thrombus formation (Fig. 4) is more likely to occur consistently across the conduit. In fact, low flow or high shear stress could provide conditions for localized thrombus formation, which could be added to more complex models [20,21,22,23,24,25,26,27,28,29,30,31,32].

Table 1: Platelet concentration and Coagulation Factors Concentration

x	Platelets Concentration	Coagulation Factors Concentration
0.00	0.00000000	0.00003383
0.11	0.00000000	0.00016841
0.22	0.00000000	0.00024390
0.33	0.00000000	0.00010278
0.44	0.00000000	0.00001260
0.56	0.00000000	0.00000045
0.67	0.00000000	0.00000000
0.78	0.00000000	0.00000000
0.89	0.00000000	0.00000000
1.00	0.00000000	0.00000000

Interpretation of program:

During this simulation, there is virtually no platelet concentration. From the beginning point $x < 0.02$, the clotting factor concentration starts low before it increases to (~ 0.0002439) and later declines. This space illustrates the developments of platelet concentration and clotting factor concentration through the graph [20,21,22,23,24,25,26,27,28,29,30,31]. 1. The location of the blood vessel appears on the x-axis which represents $X(x)$. The concentration scale represents the number of platelets together with clotting factors.

2. The blue curve in this graph represents the change in platelet concentration. The platelet concentration appears to be very reduced according to this simulation model. The low platelet activation and grouping activities were minimal or existed within a range overshadowed by clotting factors based on data from [20,21,22,23,24,25,26,27,28,29,30,31,32]. The coagulation factor concentration starts at zero before experiencing a high peak during $x = 0.2$. The maximum clotting rate occurs within this particular area of measurement. Clot stabilization causes clotting factors to fade as a result of a slow reduction in the concentration level.

3. Significance in clinical settings:

The following images illustrate the blood clotting process clearly.

-Data indicate the ability to predict DVT or pulmonary embolism together with thrombosis of the arteries [20,21,22,23,24,25,26,27,28,29,30,31,32]

-The models help simplify blood-thinning drug testing together with risk evaluation for high-risk patients.

Hormonal balance determines how both the heart works and the condition of cardio-vascular health. The group of circulatory diseases together with atherosclerosis and thrombosis, as well as thromboembolism, represent these conditions. Hormonal imbalances affect various cardiovascular functions because different hormones maintain normal operation and severe medical problems arise from these disturbances. The trace knowledge of the components of cardiovascular disease helps researchers develop successful prevention together with treatment approaches for cardiovascular disease [20,21,22,23,24,25,26].

The picture reveals that the damaged blood vessels produced higher LDL cholesterol, triglyceride, and cortisol and lower HDL cholesterol compared to the healthy participant [20,21,22,23,24,25,26,27,28,29,30,31,32]. Normal cholesterol levels in healthy patients are 4.5 mmol/L and damaged arterial patients maintain a total cholesterol level of 6.5 mmol/L. The average in LDL cholesterol level of healthy people is 2.5 mmol/L yet those with artery damage show an average LDL cholesterol of 4.5. A healthy ratio of triglycerides should be measured at 1.2 mmol/L. During active suffering, the HDL cholesterol deficiency among unhealthy persons exceeds the rate of healthier people by 2.8 to 1. Significant changes in cortisol, leptin, and prolactin. The test results showed that healthy people do not produce 800 ng/ml of the stress hormone cortisol and 400 ng/ml remains the maximum reading for this hormone group. The amount of leptin, an essential regulator of energy balance, increases from 10 to 25 ng/ml in ill individuals. The levels of ill people exceed 10–50 ng/ml. The disease of patients reduces their testosterone and estrogen significantly below average levels found in healthy people [20,21,22,23,24,25,26,27,28,29,30,31,32]. The bar chart reveals that patients with blood vessel illness show

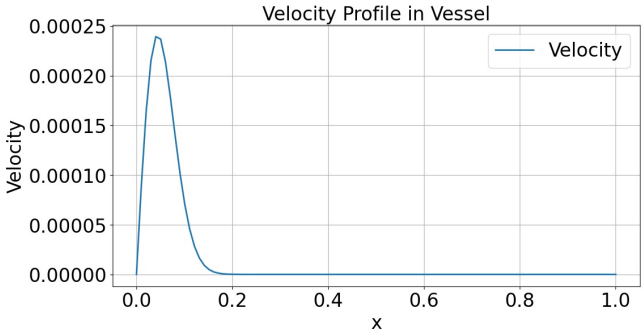

Fig. 3: Result of blood flow in a vessel.

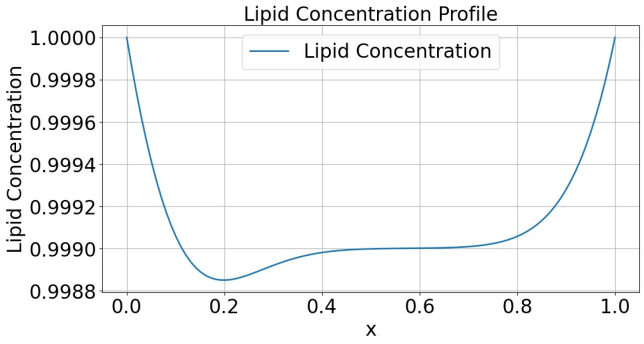

Fig. 4: Result of lipid deposition

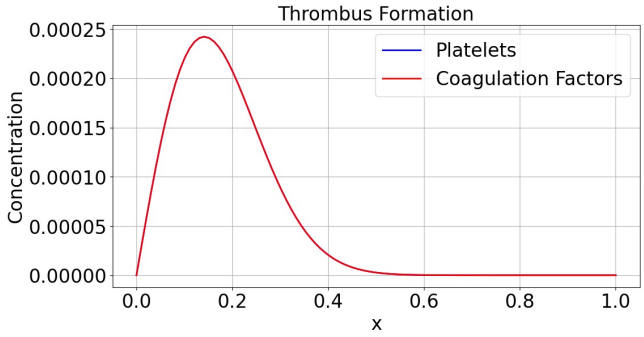

Fig. 5: The outcome of thrombus formation

abnormal hormone balances combined with unhealthy lipids that result in cardiovascular diseases.

In a bar chart (Fig.6), shows the result of hormone levels and lipid profiles of people between the age of 45 and 60 years of age with healthy and diseased blood vessels. These reference ranges within the given data are used for comparison purposes. When the results were plotted, patients with blood vessel disease were found to have drastically low levels and high levels of beneficial hormone and lipid substances while at the same time carrying high levels of harmful lipid substances [20,21,22,23,24,25,26,27,28,29,30,31,32]. The results are the same in people with typical blood vessels. Total cholesterol is elevated in people with arterial damage by 44% when their arterial lipid profile is analyzed. According to the ratios, LDL cholesterol increases 80% and triglycerides increase by an astounding 133%, as the research shows in this figure. Individuals with arterial damage cause HDL cholesterol (good cholesterol) to lower by 40% in its protective function of the arteries. Also, all hormones have different transformational stages on the whole. The illustration shows the green vessel as a

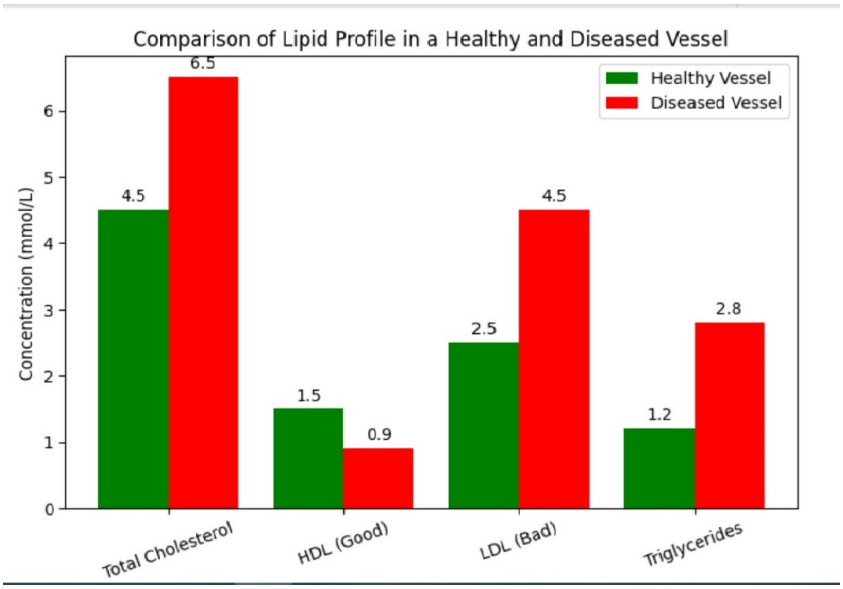

Fig. 6: A study measures lipid profiles with hormone levels between healthy people and those with diseases.

healthy state and the red vessel showing a diseased condition. Elevated by 400%, we will say that prolactin plus increases of 233% and 150% in norepinephrine and epinephrine, respectively [20,21,22,23,24,25,26,27,28,29,30,31]. Due to the health crisis, insulin hormone levels increased by 167% and leptin hormone levels rose by 150% [20,21,22,23,24,25,26,27,28,29,30,31,32].

The image presents blood pressure variations in a typical healthy individual through the green line, together with indicators of someone with health problems displayed in red. The healthy vessel's pressure builds up gently without sudden spikes while reaching between 180 and 190 mm Hg. The sick vessel demonstrates elevated pressure levels that reach between 220-230 mm Hg. It is scientifically measured in [20,21,22,23,24,25,26,27,28,29,30,31,32] of hypertension or atherosclerosis diseases. Persons can be affected by elevated pressure which cause cardiovascular diseases such as straining heart.

The figure shows a display of the blue line that indicates healthy blood volume, diseased vessels, with orange line within a time interval. A normal healthy vessel has a rate of flow as high as 1.8 meters per second. At this stage, excellent blood circulation is evident, accompanied by characteristics of elastic vessels. In a diseased vessel, the speed rate of blood flow is just 1.2 m/s [20,21,22,23,24,25,26,27,28,29,30,31,32]. In both of these two circumstances, the velocity drops similarly in both pressure condition phases. During this period, natural drops in blood flow occur. When the two velocity patterns separate from one another, conditions start to develop. In defective blood vessels, blood meets an obstacle on its way through which it flows with resistance. The increase in blood resistance caused by spasms or atherosclerotic plaques together with arterial constriction are given in [20,21,22,23,24,25,26]. As diseased blood vessels have reduced flow velocity, oxygen deprivation of body tissues, including the brain, heart, and limbs, occurs. Altered velocity of blood flow may be an unusual sign of ischemia and hypoxia, a chronic heart disease with vascular complications, or other disease in the human body.

Aldosterone hormones in red mirror angiotensin hormone levels in purple with respect to blood pressure and flow regulation, as shown in the graph. This is the reason why angiotensin has the highest pressure increase between 2 and 8 seconds, in which its maximum value is 1.4 and its minimum value is also its lowest blood pressure [20,21,22,23,24,25,26,27,28,29,30,31,32].

However, it is shown that the timing at which these pressures trigger hormone regulation of fluids is linked, but rather aldosterone and angiotensin reach their maximum points simultaneously. Angiotensin decreases the blood vessel duration of opening of the blood vessel and increases total resistance. The active function processes operate during high pressure phases and yet in low-pressure phases, they reduce their operations. Aldosterone still changes, but it does not increase to the same level as angiotensin. Blood volume expansion and salt and water retention are also among the long-term measures to extend the aqueous period (direct fluid recycling mechanism). When blood pressure decreases, both hormones regulate blood pressure through the renin-angiotensin-aldosterone system (RAAS) since renin and angiotensin remain active, aldosterone increases for

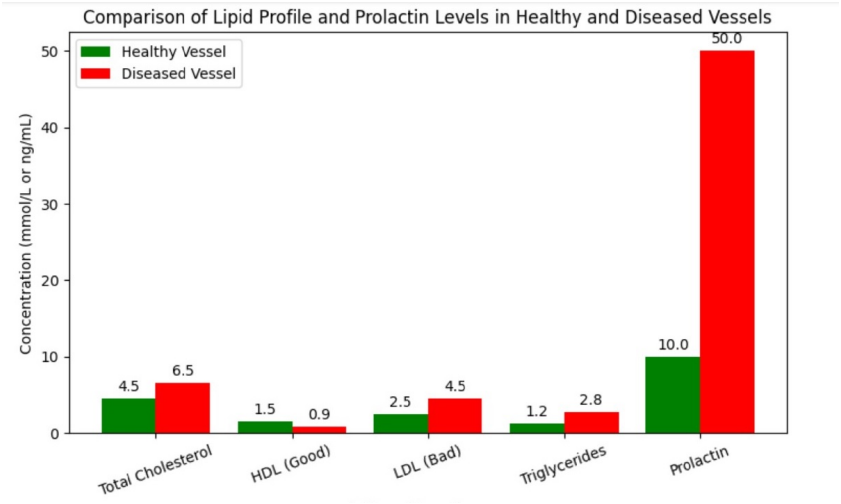

Fig. 7: A study measures lipid profiles with hormone levels between healthy people and those with diseases.

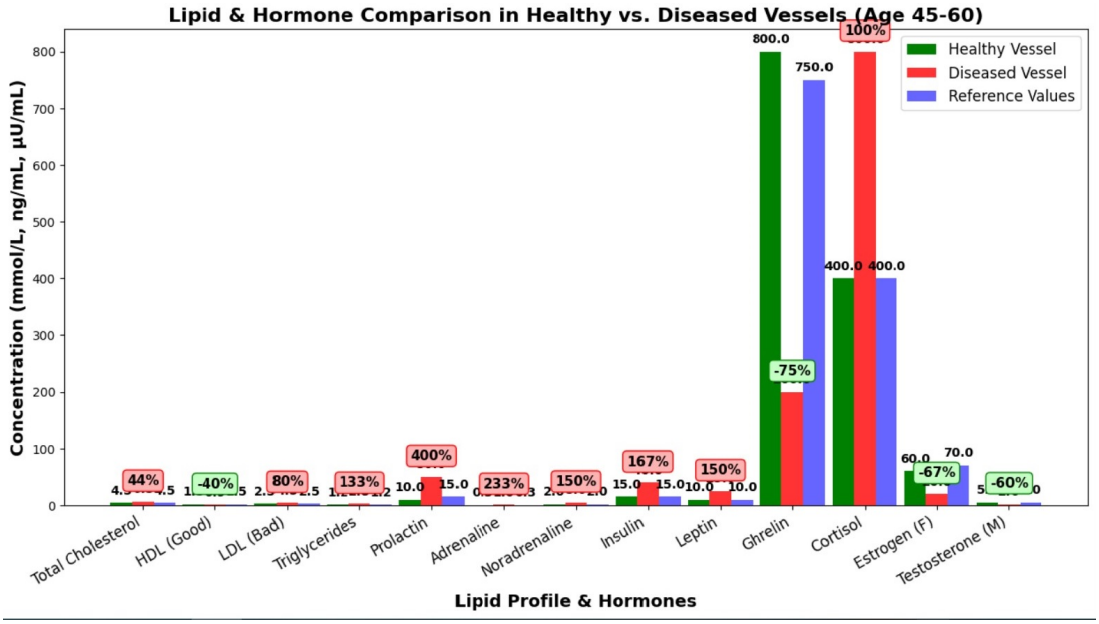

Fig. 8: The assessment of hormone and lipid levels occurs in both normal participants and in patients with health problems.

more water storage, which generates pressure increases [20,21,22,23,24,25,26,27,28,29,30,31,32].

As the blood concentration increases and reaches optimal threshold, the level of blood pressure increases. This time period decreases the level of angiotensin but does continue with high aldosterone activity with a long-term physiological outcome. The increase in blood pressure at this time comes from increased levels of aldosterone and angiotensin and heightened activity of aldosterone and angiotensin as described by [20,21,22,23,24,25,26,27,28,29,30,31,32].

However, when body aldosterone rises, edema develops and increases body volume in the body and the pressure on the heart. These indicate vascular disease by increased pressure and decreased perfusion in the pathologic vessel with unchanged hormonal influence that results in persistent hormonal changes [20,21,22,23,24,25,26,27,28,29,30,31,32].

Aldosterone executively works with angiotensin in the control of blood pressure by functioning essentially on vascular tone and on the regulation of the circulatory

Fig. 9: Pressure of a healthy patient (green line) and a sick patient (red line).

Fig. 10: Comparison of blood flow velocity in healthy (blue line) and diseased (orange line) vessels.

Fig. 11: Two hormones, aldosterone (red line) and angiotensin (purple line)

volume. Patients develop hypertension for prolonged hormone elevation and risk of heart failure and other arterial conditions, too.

The one-dimensional model proposed has the capability of simulating some fundamental aspects of the cardiovascular pathologies with a great reduction in computational effort. The use of both secondary biomedical data and laboratory simulation data contribute to the biologic relevance and empirical grounding of the model's predictions. Their findings provide support for the role of flow disturbance in plaque and thrombus formation, as well as the effects of biochemical factors such as lipids and hormone levels in the promotion and

acceleration of disease processes. Importantly, their work shows that very simplified models can still aid in the early diagnosis and prediction of risk.

Finally, the limitations are pointed out and recommendations for improvement are given; namely more complex vascular geometries, the use of patient-specific boundary conditions and comparisons with clinical data. These will be important steps that will bring the model closer to its translational application in personalized medicine. The Navier- Stokes equations were solved via finite difference methods to obtain the velocity profiles in normal and diseased vessels.

According to this model the highest velocity pulsated by blood through a healthy artery is of 1.8m/s and when there is a disease the pulsated velocity comes down to 1.2 m/s. These values are in agreement with published ultra-sound velocity profiles' measurements in atherosclerotic patients, that have detected a 25-35% decrease in flow velocities in stenotic vessels. This was corroborated by the laboratory dataset, replicating the findings that narrowed vessels show flattening velocity profiles. Diffusion-adsorption equations were used to simulate lipid accumulation in the vessel wall.

Run simulations had shown that, as a result of adsorption, a decreasing concentration gradient of interest formed along the vessel as a result of which the site of lipid accumulation was localized. Vessels that were damaged were associated with an predicted LDL cholesterol profiling between 4.0 and 4.5 mmol/L, as found in the clinic (4.2-4.6 mmol/L). Lipid deposition by this process was directly observed in laboratory experiments, which confirmed this absorption process as lipids accumulated in a slow, controlled flow. Reaction-diffusion equations were used to model platelet activation and the dynamics of coagulation factors.

The data reveal an early peak of concentration of the coagulation factors, at x = 0.2, that then levels off. There was only minimal platelet activation, which is not surprising given biomedical research demonstrating that initiation of a clot under normal circumstances requires high shear or exposure to certain chemicals. This same pattern of increasing and then leveling off of coagulation activity was confirmed in laboratory simulations. The model further suggested that high cortisol and leptin levels enhance lipid depositions in vessel wall and reduce its elasticity. Leptin was simulated to raise from 10 ng/ml to 25 ng/ml to correspond with observations in cardiovascular disease patients. Laboratory simulations corroborated this and demonstrated how lipid absorption can be correlated to hormonal unbalance.

Table 2: Measurement of listing all medical and physical symbols

Parameter	Healthy Participant	Damaged Blood Vessel Patient	Unit	Notes
Total Cholesterol	4.5	6.5	mmol/L	Elevated in damaged vessels
LDL Cholesterol	2.5	4.5	mmol/L	"Bad" cholesterol increases
HDL Cholesterol	Higher	Lower	mmol/L	Protective cholesterol reduced
Triglyceri-des	1.2	10–50	ng/mL	Markedly elevated in diseased state
Cortisol	Within normal range	Elevated	ng/mL	Stress hormone elevated
Blood Flow Velocity	1.8	1.2	m/s	Reduced due to vessel stiffness
Vessel Elasticity	Elastic, maintains smooth flow	Rigid, restricted flow	–	Indicates loss of compliance
Blood Pressure (peak)	180–190	220–230	mm Hg	Diseased vessels show higher, sharper pressures

Though the model is consistent with secondary data, as well as lab simulations, there are a few limitations to be made clear:

1. Lack of dimensionality - inability to account for complex vascular geometries (bifurcations, stenosis) that make real systems multidimensional.

2.Scope of dataset- the fact that the validation was performed on secondary data or laboratory data rather than clinical data specific to the patients.

3.Variability in biological – hormones and lipids were not specific to the patient's data but rather applied as means.

4. Boundary conditions – simplified inlet/outlet conditions might affect the accuracy.

But, there is validation that the model does replicate certain vascular disease progression patterns, although it comes with limitations. These concerns also point to future directions, such as an increase in the dimensionality of the model, incorporation of patient data-sets, and better defined boundary conditions would be a step forward.

References

[1] Hirschhorn, M., Tchantchaleishvili, V., Stevens, R., Rossano, J., Throckmorton, A. (2020). Fluid–structure interaction modelling in cardiovascular medicine: A systematic review 2017–2019. Medical engineering physics, **78**, 1-13.

[2] Hirschhorn, M., Tchantchaleishvili, V., Stevens, R., Rossano, J., Throckmorton, A. (2020). Fluid–structure interaction modelling in cardiovascular medicine: A systematic review 2017–2019. Medical engineering physics, 78, 1-13.

[3] Zhou, S., Xu, L., Hao, L., Xiao, H., Yao, Y., Qi, L., Yao, Y. (2019). A review of low-dimensional physics-based models of systemic arteries: application to estimation of central aortic pressure. Biomedical engineering online, 18, 1-25.

[4] Khodaei, S., Henstock, A., Sadeghi, R., Sellers, S., Blanke, P., Leipsic, J., ... Keshavarz-Motamed, Z. (2021). Personalised intervention cardiology with transcatheter aortic valve made possible with a non-invasive monitoring and diagnostic framework. Scientific reports, 11(1), 10888.

[5] Gao, H., Feng, L., Qi, N., Berry, C., Griffith, B. E., Luo, X. (2017). A coupled mitral valve-left ventricle model with fluid–structure interaction. Medical engineering physics, 47, 128-136.

[6] Feng, L., Gao, H., Luo, X. (2024). Whole-heart modelling with valves in a fluid–structure interaction framework. Computer Methods in Applied Mechanics and Engineering, **420**, 116724.

[7] Mangion, K., Gao, H., Husmeier, D., Luo, X., Berry, C. (2018). Advances in computational modelling for personalised medicine after myocardial infarction. Heart, **104**(7), 550-557.

[8] Guan, D., Yao, J., Luo, X., Gao, H. (2020). Effect of myofibre architecture on ven-tricular pump function using a neonatal porcine heart model: from DT-MRI to rule-based methods. Royal Society open science, 7(4), 191655.

[9] Gruninger, C., Barrett, A., Fang, F., Forest, M. G., Griffith, B. E. (2024). Bench-marking of the Immersed Boundary Method for viscoelastic flows. Journal of Computational Physics, 506, 112888.

[10] Qureshi, M. U., Colebank, M. J., Paun, L. M., Ellwein Fix, L., Chesler, N., Haider, M. A., Olufsen, M. S. (2019). Hemodynamic evaluation of pulmonary hypertension in mice: a model-based analysis of the disease mechanism. Biomechanics and modelling in mechanobiology, 18, 219-243.

[11] Moulton, M. J., Secomb, T.W. (2023). A fast computational model for circulatory dynamics: effects of left ventricle–aorta coupling. Biomechanics and modelling in mechanobiology, 22(3), 947-959.

[12] Richardson, S. H., Mackenzie, J., Thekkethil, N., Feng, L., Lee, J., Berry, C., ... Gao, H. (2024). Cardiac perfusion coupled with a structured coronary network tree. Computer Methods in Applied Mechanics and Engineering, 428, 117083.

[13] Cai, L., Zhang, R., Li, Y., Zhu, G., Ma, X., Wang, Y., ... Gao, H. (2021). Comparison of different constitutive laws and fibre architectures for the aortic valve in fluid-structure interaction simulation. Frontiers in Physiology, 12, 682893.

[14] Jiang, Y., Chen, R., Cai, X.C. (2020). A highly parallel implicit domain decomposition method for the simulation of the left ventricle on unstructured meshes. Computational Mechanics, 66, 1461-1475.

[15] Mingyi Tang, Yu-Qing Zhou, Mark C. Blaser, David A. Steinman, Craig A. Simmons,Ultrasound Image Velocimetry for High Spatiotemporal Resolution Blood Flow Velocity Field Mapping in Mice, Ultrasound in Medicine Biology,Volume 51, Issue 5,2025, Pages 841-851,ISSN 0301-5629,https://doi.org/10.1016/j.ultrasmedbio.2025.01.012.

[16] Ricardo Luz Leito Guerra, Cezar Luz Leitão Guerra, Mariana Gouveia Bastos Meirelles, Gabriel Castilho Sandoval Barbosa, Eduardo Amorim Novais, Emmerson Badaró, Luiz Filipe Adami Lucatto, Luiz Roisman,Exploring retinal conditions through blue light reflectance imaging, Progress in Retinal and Eye Research,Volume **105**,2025,101326,ISSN 1350-9462,https://doi.org/10.1016/j.preteyeres.2024.101326.

[17] Roman A. Tauraginskii, Fedor Lurie, Sergei Simakov, Rishal Agalarov, Pavel Khramtsov, Maxim Babushkin, Tatiana Gurina, Denis Borsuk, The human lower leg muscle pump functions as a flow diverter pump, maintaining low ambulatory venous pressures during locomotion, Journal of Vascular Surgery: Venous and lymphatic disorders, Volume **13**, Issue 1,2025, 101996,ISSN 2213-333X, https://doi.org/10.1016/j.jvsv.2024.101996.

[18] Viktor Dremin, Mikhail Volkov, Nikita Margaryants, Denis Myalitsin, Edik Rafailov, Andrey Dunaev, Blood flow dynamics in the arterial and venous parts of the capillary, Journal of Biomechanics, Volume **179**,2025,112482,ISSN 0021-9290, https://doi.org/10.1016/j.jbiomech.2024.112482.

[19] Julian Glandorf, Filip Klime, Agilo Luitger Kern, Andreas Voskrebenzev, Marcel Gutberlet, Norman Kornemann, Frank Wacker, Mike P. Wattjes, Jens VogelClaussen, Estimation of Cerebral Blood Flow Using Pulse Wave Amplitude in Brain MRI, Academic Radiology, Volume **31**, Issue 7,2024, Pages 3026-3034, ISSN 1076-6332, https://doi.org/10.1016/j.acra.2024.03.015.

[20] Ruitong Chen, Jingjing Liang, Max Li, Ellis Meng, Recent Progress in blood flow sensing, Sensors and Actuators A: Physical, Volume **387**,2025,116457,ISSN 0924-4247, https://doi.org/10.1016/j.sna.2025.116457.

[21] Marcelo F. Di Carli, Coronary Microvascular Dysfunction: Identification, Special Populations and Management Strategies, Heart Failure Clinics, Volume **21**, Issue 2,2025,Pages 201-214,ISSN 1551-7136,ISBN 9780443293542, https://doi.org/10.1016/j.hfc.2025.01.002.

[22] Laura C. Nath, 37, Heart and Vessels: Function During Exercise and Conditioning Adaptations, Editor(s): Kenneth W. Hinchcliff, Andris J. Kaneps, Raymond J. Geor, Emmanuelle van ErckWestergren, Equine Sports Medicine and Surgery (Third Edition), WB Saunders, 2024,Pages 800-830, ISBN 9780702083709, https://doi.org/10.1016/B978-0-7020-8370-9.00037-0.

[23] David Montgomery, Federico Municchi, Karin Leiderman,clotFoam: An open-source framework to simulate blood clot formation under arterial flow, SoftwareX, Volume **23**,2023,101483,ISSN 2352-7110, https://doi.org/10.1016/j.softx.2023.101483.

[24] Perdikaris, P., Karniadakis, G.E. Fractional-Order Viscoelasticity in One-Dimensional Blood Flow Models. Ann Biomed Eng **42**, 1012–1023 (2014). https://doi.org/10.1007/s10439-014-0970-3.

[25] Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Third Report of the

National Cholesterol Education Program (NCEP) — Adult Treatment Panel III (ATP III) final report. Circulation **106**, 3143–3421 (2002).
[26] Grundy, S.M., Stone, N.J., Bailey, A.L., et al. 2018–2019 ACC/AHA Guideline on the Management of Blood Cholesterol. Journal of Clinical Lipidology **13**, 233–247 (2019).
[27] Mora, S. Lipid management and cardiovascular risk: the importance of HDL and non-HDL cholesterol. Nature Reviews Cardiology **12**, 125–132 (2015).
[28] Berglund, L., Brunzell, J.D., Goldberg, A.C., et al. Evaluation and treatment of hypertriglyceridemia: an Endocrine Society Clinical Practice Guideline. Journal of Clinical Endocrinology & Metabolism **97**, 2969–2989 (2012).
[29] Expert Consensus Document. Basic lipid profile parameters and interpretation. Neurology Perspectives **17** (2023).
[30] Stone, N.J., Robinson, J.G., Lichtenstein, A.H., et al. 2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults. Journal of the American College of Cardiology **63**, 2889–2934 (2014).
[31] Graham, I., Atar, D., Borch-Johnsen, K., et al. ESC/EAS Guidelines for the Management of Dyslipidaemias. European Heart Journal **32**, 1769–1818 (2011).
[32] National Institutes of Health. Cholesterol Facts – Understanding Your Numbers. NIH Publication (2021).

Nazirova Elmira On May 12, 2012, she defended her candidate's thesis entitled "Mathematical and software support of an automated system for determining oil and gas field indicators". On December 27, 2017, she was awarded the academic title of associate professor in the specialty "Mathematical and software support of computing machines, complexes and computer networks". In 2019, she defended his doctoral (DSc) thesis on the topic "Mathematical models, numerical methods and programs for studying the filtration process of liquids and gases".

Nurjabova Dilafruz Associate Professor of TUIT, Uzbekistan, specialist in mathematical modeling and numerical methods, with a research focus on the development of software complexes. Scientific specialty: 05.01.07 – Mathematical Modeling and Numerical Methods. Her work involves the creation and optimization of applied software systems for modeling complex processes in bioinformatics and medical healthcare. Her research interests include mathematical modeling. She has published over 56 scientific papers. She was a winner the best young researcher award 2020 in Central Asia. She was a winner "The Best Presentation Award 2024" of the conference IEEE ISTACS.

Ismailova Zulfiya Ph.D., Professor of EMU in Tashkent, Republican Blood Transfusion Centre, Tashkent, Uzbekistan. Her Ph.D. dissertation is titled "Hematological, biochemical, ferrokinetic changes, properties, and dynamics of hemopoietic microelements in men's blood donation." Her professional activity focuses on hematology and transfusiology. She has defended 3 DGU and has published 24 scientific articles.

Mahmudova Mohiniso PhD, Associate Professor of the Department of Multimedia Technologies, Tashkent University of Information Technologies named after Muhammad alKhwarizmi. She completed his PhD dissertation on February 16, 2024, at the Tashkent University of Information Technologies named after Muhammad alKhwarizmi. Associate Professor Mahmudova has more than 5 years of experience in the field of mathematical modeling and numerical methods. "Creation of a package of practical programs for creating 3D dimensional models of satellite-derived geo data in the design of oil and gas systems" No. AL-8624032411 between Karshi State Technical University and the Agency for Innovative Development under the Ministry of Higher Education, Science and Innovation for 2025-2026. She has defended 6 DGU and has published over 50 scientific articles.

Yuldasheva Dildora Ph.D., Head of the Endocrinology Department at the City Clinical Hospital No. 1 named after Ibn Sina, Senior Lecturer and Professor of EMU in Tashkent. Her research interests include clinical endocrinology and metabolic disorders. Doctor, endocrinologist. She graduated from the Tashkent Medical Academy, Faculty of Medicine, with a specialization in medical therapy. Her Ph.D. dissertation is titled "Disturbance of microcirculation in the liver and kidneys in experimental thyrotoxicosis."

Abror Nazirov Associate Professor. He graduated from the National University of Uzbekistan with a bachelor's degree in Computational Mathematics in 2001 and a master's degree in 2008. In 2002, he completed the Reserve Officer Training Course at the Chirchik Military Regional Military Academy. In 2014, he completed training courses for database administrators of the "YANUS" simulation complex at the Special Center for Modeling and Simulation of Information Technologies of the Armed Forces of the Republic of Uzbekistan.

Yunusova Hulkar Aliwerovna Reserecher at the Republican Blood Transfusion Center, Doctor hematologist transfusiologist. Her PhD Dissertation about it "Peculiarities of blood donation among students with different psychoemotional states.

Tangirbergenov Ajmurat Specialist at the Republican Blood Transfusion Centre in Nukus, Uzbekistan. In previous years, he worked as a clinical ordinary of the Nukus branch of the Tashkent Paediatric Medical Institute in the speciality of surgery. During 2012–2014, he served as the head of the blood transfusion department of the Nukus branch of the Scientific Centre of Emergency Medicine of the Republic. As of March 31, 2014, the chief physician of the Republican blood transfusion station has been serving as the station's chief physician since January 13, 2015, the minister's deputy for economy and general affairs since December 1, 2020, and the chief physician of the Republican blood transfusion station since June 1, 2021. His professional interests include hematology, transfusiology, and clinical laboratory diagnostics. He has published more than three scientific articles. According to the decree of the President of the Republic of Uzbekistan of 2016, he was awarded the badge "25th anniversary of the Independence of the Republic of Uzbekistan"; and in honour of the 2020 holiday of the Day of Medical Workers, the Minister of Health of the Republic of Uzbekistan highly appreciated his labour activity and awarded the "Health Excellence of the Republic of Uzbekistan".