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BLOOD FLOW SIMULATION USING SMOOTHED PARTICLE HYDRODYNAMICS

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ABSTRACT

To understand the characteristics of blood flow, it is important to identify the key parameters that influence the flow of blood. The characterisation of blood flow will also enable us to understand the flow parameters associated with physiological conditions such as atherosclerosis. Thrombosis plays a crucial role in atherosclerosis and it also helps to stop bleeding when a blood vessel is injured. This article focuses on using a meshless particle-based Lagrangian numerical technique, named the smoothed particles hydrodynamic (SPH) method, to study the flow behaviour of blood and to explore the flow conditions that induces the formation of thrombus in a blood vessel. Due to its simplicity and effectiveness, the SPH method is employed here to simulate the process of thrombogenesis for various blood flow parameters. In the present SPH simulation, blood is modelled by particles that have the characteristics of plasma and of platelets. To simulate the coagulation of platelets which forms thrombus, the adhesion and aggregation processes of the platelets are modelled by an effective inter-particle attraction force model. With these models, the motion of platelets in flowing blood, and their adhesion and aggregation are effectively coupled with viscous blood flow. In this study, the adhesion and aggregation of blood particles are analysed on a (straight vessel) under various low Reynolds number scenarios. The results are compared with the experimental results, and a good agreement is found between the simulated and experimental results.

Keywords: *smooth particle hydrodynamics (SPH); blood flow; thrombus; arteries*

1.Introduction

A thrombus is considered to be one of the most important causes of many diseases in human body. On the other hand, A blood clot anchored to a damaged vascular wall can stop bleeding or it can prevent atherosclerosis in arteries. The danger is that a thrombus can affect the blood flow in the vessels and this can cause potentially deadly accidents, such as cardiac infarction (or heart attack) or ischemic stroke when the damage occurs in the coronary or the carotid arteries, respectively. The formation of a thrombus depends on platelet flow; for example, the transport to denuded subendothelium, formation of membrane tethers, adhesion to the subendothelium, and aggregation. Many experimental studies have provided information on the biochemical effects of fluid forces on thrombogenesis. In recent years, due to the availability of vast computational power, research on computer simulation of thrombosis has become a field of deep interest. Although fluids can be simulated in either the Eulerian or Lagrangian method, the Lagrangian method is considered to be more suitable for this type of simulation due to their obvious advantages in tracking movement of particles similar to platelets[1]. The purpose of this study is to analyse flow parameters that influence the formation of thrombosis inside arteries. A Lagrangian smoothed particles hydrodynamics (SPH) is used for numerical simulations of the blood flow consisting plasma and platelets.

2. Numerical Methodology

The governing equations for solving incompressible or weakly compressible isothermal fluid flow using SPH are mass and momentum conservation equations given by,

$$\frac{1}{\rho}\frac{D\rho}{Dt} + \nabla.\mathbf{v} = 0; \qquad \frac{D\mathbf{v}}{Dt} = -\frac{1}{\rho}\nabla \mathrm{p} + v\nabla^2.\mathbf{v} + \mathbf{F}. \qquad (1),(2)$$

Where ρ, t, v, $\mathbf{v}$, and p represent the density, time, kinematic viscosity, velocity and pressure of the fluid particles and, **F** represents the external force acting on fluid particles. The fluid pressure for weakly compressible SPH formulation is obtained by an equation of state as presented in [2]**.** The numerical procedure to calculate fluid velocity is derived from the momentum equation (2) as,

$$\mathbf{v}^{n+1} = \mathbf{v}^n + \left(-\frac{1}{\rho}\nabla P + v\nabla^2.\mathbf{v}^n + \mathbf{F}\right)\Delta t \qquad (3)$$

Where superscript n and $n+1$ refer to current and next time steps, respectively, and Δt is the numerical time step. The position and density of the fluid can be updated respectively at every time step by,

$$\mathbf{x}^{n+1} = \mathbf{x} + \mathbf{v}^{n+1}\Delta t, \qquad (4)$$

and (from the continuity equation (1)),

$$\rho^{n+1} = \rho^n - \rho^n(\nabla.\mathbf{v}^{n+1})\Delta t. \qquad (5)$$

The pressure is then estimated from the updated density.

3. Modelling Platelet Motion

The platelets tend to adhere and aggregate when the blood vessel is damaged. This can lead to formation of a primary thrombus. Inside the primary thrombus the neighbour platelets link together, which are then bound by vWF fibrinogen in plasma and collagen in the sub-endothelial tissue [3]. This process takes place by making a link between neighbouring platelets and bound by vWF fibrinogen in plasma and collagen in the sub-endothelial tissue. To numerically model such platelet motion, an algorithm based on a penalty or spring force mechanism [4] is adopted. This model dictates the interactions between platelets and plasma inside the blood vessel. When the platelets are within a distance d_{ad} from the damaged area, the platelets are attracted towards the damaged wall by an adhesive force given by eq. (6). The platelets adhering to the wall are then activated and attract other platelets which are within a distance of d_{ag} from them. This attractive force is called an aggregation force which is given by eq. (7). The aggregation force takes the same form as that of the adhesive force but has a different spring constant.

$$\boldsymbol{F}_{ad} = \begin{cases} K_{ad}(|\boldsymbol{r}_{ij}| - r_o)\boldsymbol{n}_{ij} & (|\boldsymbol{r}_{ij}| \le d_{ad}) \\ 0 & (|\boldsymbol{r}_{ij}| > d_{ad}) \end{cases}; \boldsymbol{F}_{ag} = \begin{cases} K_{ag}(|\boldsymbol{r}_{ij}| - r_o)\boldsymbol{n}_{ij} & (|\boldsymbol{r}_{ij}| \le d_{ag}) \\ 0 & (|\boldsymbol{r}_{ij}| > d_{ag}) \end{cases} \quad (6), (7)$$

In the above equations $\boldsymbol{F}_{ad}, \boldsymbol{F}_{ag}$ are the adhesive and aggregate forces and K_{ad}, K_{ag} are the corresponding spring constants. The $\boldsymbol{r}_{ij}$ here is distance between activated platelet and vessel wall (or other non-activated platelets), r_o is the original or natural length of the spring and $\boldsymbol{n}_{ij}$ is a unit vector linking platelet and damaged wall (or linking activated platelet and other surrounding platelets). These two forces are introduced in equation (2) for platelet particles which are influenced by adhesion and aggregation.

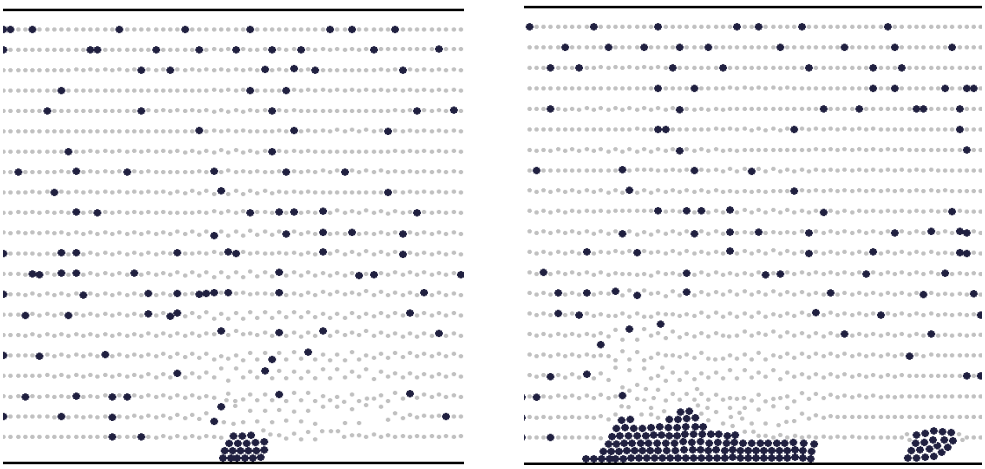
4. Blood Flow Model

In this work, the blood flow simulations were performed inside a straight blood vessel with flow Reynolds numbers 0.01, 0.02, and 0.03, which were defined at the inlet velocity. The total length of

the vessel (L) and the width between two walls (D) are respectively 130μm and 20μm. The dimensions of the damaged wall (L_i) is 30μm (refer to the length of the wall damage) and the distance from the inlet to the damaged wall (L_o) is 40μm (see Fig.1). The total number of particles used in the simulation was 5079. Four layers of boundary dummy particles were also used. The initial distance between particles is 1.0 μm. The density ρ and kinematic viscosity v of the plasma and platelets, were set as $\rho = 1\text{x}10^3 \text{kg/m}^3$ and $v = 1\text{x}10^{-6}\text{m}^2/\text{s}$. The boundary conditions were; a uniform velocity at the inlet, zero pressure at the outlet and, non-slip condition at the walls enforced by dummy boundary particles. The amount of the platelet particles used is approximately 8.8% of the plasma to resemble normal physiological condition. The time step was set to $5\text{x}10^{-7}\text{s}$ to ensure the stability of numerical integration scheme. In the reported numerical simulations, the spring constants K_{ad} and the K_{ag} are $9.0\text{x}10^9\ N/\text{m}$ and $4.5\text{x}10^9\ N/m$ respectively, while d_{ad}= 3.0μm = d_{ag}, and r_o = 2.0μm.

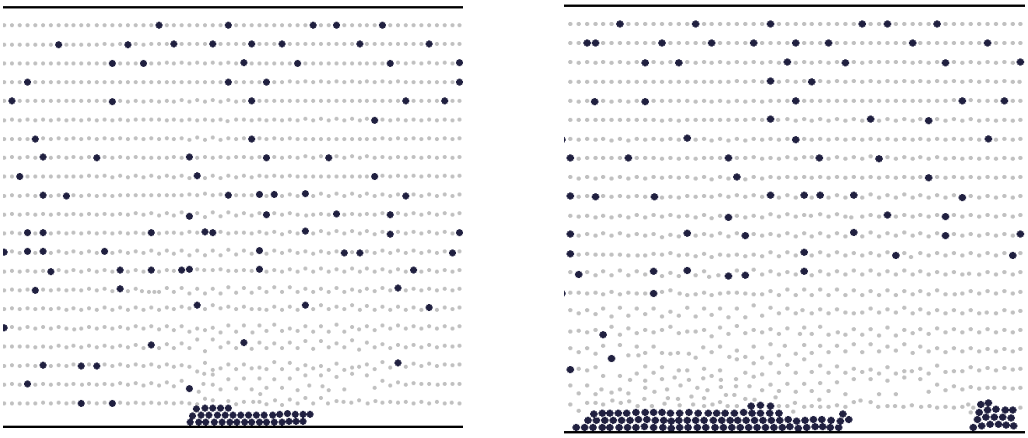
5. Results

The purpose of this study is to demonstrate the formation of thrombus and to investigate the applicability of SPH in modelling such process. The corrected SPH is used to improve the accuracy [5] of the simulation. Normally, a thrombus is formed by adhesion and aggregation of platelets which are transported by the blood flow in different geometries of arteries or vessels, where the growth rate of thrombus formation varies with the stenosis and the flow rate of blood. Figure 1 illustrates the formation of thrombus at two different stages of the flow. In these figures, for clarity, plasma and platelet particles are shown by two different the plasma and platelets are denoted by light and the dark grey respectively. The platelets are activated when they are within d_{ad} distance from the damaged region and form a primary thrombus. During the course of time, a primary thrombus is developed to cover the whole damage area by forming several layers of platelets. When thrombus grows to a certain volume, part of the thrombus is separated and transported downstream by the blood flow. Figures 1 and 2 depict the growth of thrombus at different times for Reynolds numbers 0.01 and 0.03 of the flow. From the figures below, various stages of thrombus growth on the damaged area of the wall are clearly evident. It can be noted from Fig. 1(b) and 2(b) that, part of the thrombus is separated from primary thrombus once the primary thrombus grows to a substantial volume. It is interesting to observe that the volume of the primary thrombus and the time at which separation of the thrombus takes place are affected by the flow rate. From these figures it can be concluded that the higher the flow rate the thinner the thrombus growth would be. Further, it can be noted that with higher flow rates the separation of thrombus takes place quicker.



(a) (b)

Figure 1: The platelet aggregation in the flow Re=0.01 at (a) t=0.2s; (b) t=0.6s



(a) (b)

Figure 2: The platelet aggregation in the flow Re=0.03 at (a) t=0.2s; (b) t=0.6s

6. Discussion

The influence of changing hemodynamics on the platelet transport and thrombus formation was investigated using SPH. The formation and subsequent behaviour of thrombus at various blood flow rates were analysed by numerical simulations. It is evident from the results above that the blood flow rate plays a crucial role in the build-up and separation of thrombus. The results show that the growth rate of the thrombus, its thickness, and formation/separation vary according to the blood flow rate. These results are consistent with the observations reported in [6].

7. Conclusions

This work has focused on the simulation of the thrombogenesis process using the SPH method by considering platelet aggregation and the influence of blood flow rates on thrombus growth. In the numerical simulations, blood inside a straight vessel is discretised by particles which are assumed to have the characteristics of blood constituents, such as plasma and platelets. The potential of SPH method to simulate thrombogenisis process is demonstrated via numerical examples. This study also demonstrates the ability and accuracy of the SPH method in modelling blood flow with low Reynolds numbers.

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