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# Mathematical Study of the concentration effect of red cells in blood

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## Abstract

In this paper, a mathematical and numerical study of the effect of red blood cell concentration on a circular cross section tube is presented. The considered PDE system use the Bingham model describing blood as a non-Newtonian fluid. This system consists of Navier-Stokes equations describing the behavior of fluid and advection-reaction-diffusion equations that take into account the influence of chemical reactions on transient flow behavior in the arteries. Finally, numerical tests are presented to treat blood flow in the case of fusiform aneurysms caused by abdominal aortic aneurysm disease.

*Keywords:* Bingham model; advection-diffusion-reaction equation; Abdominal Aortic Aneurysm; Papanastasiou regularization.

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## 1. Introduction

The study of the behavior of fluids according to their viscosity is one of the interesting physical problems. Viscosity is a very important parameter that characterizes the behavior of the fluid since it links the stress of a fluid in motion with the rate of deformation of their constituent elements. For this reason, researchers are increasingly using generalized fluid models to meet the specific needs of modeling, such as the non-Newtonian fluids, which the vis-

cosity cannot be defined as a constant value because of the non-linear relation between the strain rate and the shear stress [Marrero et al. 2014]. If the non-Newtonian character is manifested by the decreasing of the viscosity with increasing shear rate, then it is in the case of rheofluidifiant fluids (or pseudoplastic) [Kim 2002, Chamekh and Elzaki 2018]. These are the fluids most commonly found in nature, where a constraint applied to this type of fluids must exceed a critical value, called yield stress for the flow to begin to occur. Therefore, the yield stress is a free limit between two different behaviors (rigid and viscoplastic) of the material.

Among these fluids, we can characterize the Bingham fluid that is a theoretical model that describes the flow of a rigid viscoplastic and incompressible fluid with a yield stress [Messelmi 2014]. It is of great interest because of its ever-growing industry applications. It has been used in various publications to model the flow of metals, plastics, and various polymers. If the stress applied to the fluid is less than the yield stress, no deformation occurs and the fluid does not flow, then, we obtain a phenomenon of a rigid zone in the flow is transformed into a winder because of the growth of the elastic limit which can contribute to a blockage. This is have been related to the increase of the concentration of the particles constituting this fluid which are in contact with the effect of interaction forces. This behavior is similar to the behavior of the blood, which is considered among the fluids that are characterized by this blocking property since it flows under cardiac activity and its flow can block under the coagulation phenomenon that is quite sensitive to a number of factors such as plasma temperature, viscosity and the concentration of blood cells [ Messelmi 2011]. This blocking situation may help to understand the abdominal aortic aneurysm disease (AAA). This disease can cause fusiform aneurysms and/or saccular aneurysms. In the both cases, the aneurysm is dangerous; they can evolve into rupture, allowing the blood to flow from the vessel. When this situation is reached, blood accumulates and is life-threatening.

In this study, we treat the equations modeling the blood behavior in the abdominal aorta in case of an aneurysm involving the non-Newtonian model

of Bingham. This modeling is based on the biochemical problem of blood flow in AAA diseases and is described by the convection-diffusion-reaction phenomenon, whose blood is considered as an incompressible and rigid viscoplastic fluid with a yield stress of Bingham taking into account the concentration effect of red cells. Then, a result of existence and uniqueness of a maximum principle is proposed for the concentration corresponding to this problem in a three-dimensional delimited domain, also some preliminary and notations found in [Merouani and Messelmi 2015, Messelmi 2014]. In the last part, we simulate the blood flow in AAA regularized by Papanastasiou and some numerical results on velocity field and concentration of red cells in AAA are presented.

## **2. Biological background**

Blood is probably the most important biological fluid and its rheology is interesting from both a theoretical and applied point of view. It is a concentrated suspension of several species of particles in the plasma. It contains almost 45% of figurative elements in volume and 55% plasma [Brujan 2010, Marieb 1993, Toungara 2011, Robertson et al. 2008]. In the 45%, we find 97% of red blood cells or erythrocytes, and the other 3% are distributed between white blood cells or leucocytes and platelets or thrombocytes [Marieb 1993, Toungara 2011, Robertson et al. 2008]. Therefore, the red blood cells give a rheological behavior of blood [Brujan 2010, Robertson et al. 2009]. It is well known that blood is an incompressible Newtonian fluid [Paramasivam et al. 2010, Janela et al. 2010, Marrero et al. 2014]. However, in times and places, we can notice that there are non-Newtonian properties that can be important [Robertson et al. 2009, Khanafar et al. 2006]. For example, in larger blood vessels such as the aorta, the apparent viscosity is constant and the blood was treated as an incompressible Newtonian fluid since the shear rate is high. However, in certain pathological conditions, it is clear to find non-Newtonian effects, because of the presence of circular particles, the concentration of the recirculation zones increases and the particles touch, which induces a low shear rate, the viscos-

ity high due to the aggregation of red blood cells. Since, the change in blood viscosity generally depends on the temperature, the deformation of red blood cells concentration and is related to the change in the percentage of hematocrit. Many studies have been conducted to better understand blood flow in the arterial system, with the idea to find trustworthy answers to these cardiovascular pathologies and complications. By the way, cardiovascular disease, which refers to a group of diseases of the heart and arteries increase, represent a major cause of death. Every year more people die from cardiovascular disease than from any other cause. The AAA disease is one of the most common cardiovascular diseases that usually leads to death in patients. It is a pathology of the aortic wall that affects the main artery of the human body that carries blood from the heart to all other organs. The AAA disease is a dilatation of the wall of the abdominal aorta, mainly caused by inflammation of the endothelium [Toungara 2011, Paramasivam et al. 2010, Marrero et al. 2014]. When the yield strength of the wall is reached, the AAA ruptures causing internal bleeding that is usually fatal in the patient. As a result, the work done on this pathology is a field of investigation of paramount importance in terms of public health since their rupture leads to the death of patients from 80% to 90% cite a52. Due to hemodynamic forces of pressure and shear, the aneurysm continues to expand.

In general, the natural evolution of AAA differs between humans [Mofidi 2006, DeRubertis 2007]. The risk of re-offending is 25% for aneurysms between 4.1cm and 7cm in diameter and 9.5% for aneurysms less than 4cm [Paramasivam et al. 2010, Khanafar et al. 2006 ]. There are many constitutive models that are more complex used to study blood flow as the law of power or Cross [Robertson et al. 2008, Johnstona et al. 2004, Messelmi 2011]. More recently, the Bingham model has been used in the modeling of blood flow to describe its behavior through threshold stress and blocking phenomena that describe blood clotting, formation, and lysis of blood clots. We can find in several works on the phenomena of convection-diffusion-reaction which make the study of the coagulation and the formation of blood clots [Merouani and Messelmi 2015, Messelmi 2011,

Anand et al. 2003, Hansen et al. 2015]. To do this study, we consider a mathematical model that describes the flow of blood involving the non-Newtonian model of Bingham.

### 3. Problem description

The purpose of our analysis is to determine the effect of red blood cell concentration on blood behavior. In order to guarantee this, we consider an incompressible flow inside a domain  $\Omega$ ;  $\Omega \subset \mathbb{R}^n$  ( $n = 2, 3$ ) an open bounded Lipschitzian boundary  $\Gamma$ ,  $\mathbb{S}^n$  is the space of the symmetric tensors of order  $n - 1$  on  $\mathbb{R}^n$ . The blood temperature is assumed constant. We have formulated a non-linear advection-diffusion-reaction problem for the Bingham fluid in a bounded domain of  $\mathbb{R}^3$ . For this problem, viscosity, yield strength, and diffusivity are supposed to depend on the concentration of red blood cells. The modeling is carried out thanks to a system with partial derivatives whose existence and the uniqueness of the solution are studied locally.

Let the space  $\mathbb{R}^3$  and  $\mathbb{S}^3$  are equipped by their the scalar product and the Euclidean norm, respectively. For a velocity field  $u \in H^1(\Omega)^3$ , we consider the deformation rate tensor defined by

$$D(u) = (D_{ij}(u))_{1 \leq i, j \leq n}, \quad D_{ij}(u) = \frac{1}{2}(u_{i,j} + u_{j,i}), \quad \forall u \in H^1(\Omega)^3.$$

The equation of fluid motion is given by

$$u \nabla u = \operatorname{div}(\sigma) + f \quad \text{in } \Omega, \quad (1)$$

$$\operatorname{div}(u) = 0 \quad \text{in } \Omega, \quad (2)$$

with  $\sigma$  is the field of constraints and where the density is assumed to be 1 and  $f$  represents the density by volume forces acting on the fluid, under the condition of incompressibility (3) because the density is constant all along the fluid path.

The model of Bingham is characterized by

$$\begin{cases} \sigma^D &= 2\mu(C)D(u) + g(C) \frac{D(u)}{|D(u)|} & \text{if } |D(u)| \neq 0 \\ |\sigma^D| &\leq g(C) & \text{if } |D(u)| = 0, \end{cases} \quad \text{in } \Omega \quad (3)$$

with  $\sigma^D$  is the deviator of the constraint  $\sigma \mu > 0$  and  $g \geq 0$ , which gives the variation of the velocity with the variation of the shear stress, whose viscosity  $\mu$  and the yield  $g$  depends on the concentration  $C$  of the red cells.

Further, if we assume that the advection convection phenomena of the fluid undergo a chemical reaction under isothermal conditions, then the equation governing the concentration  $C$  is given by

$$u \nabla C - \operatorname{div}(\eta(C) \nabla C) = R(C) \text{ in } \Omega. \quad (4)$$

This equation describes the variation of the concentration of the particles constituting the fluid where  $\eta$  is the diffusivity of the fluid and  $R$  is the reaction function (interaction and collision) between the different particles constituting the fluid. In a particular case, we have  $R(C) = -K C$  of first-order chemical reaction with  $K$  is a constant chemical reaction rate. We assume that the boundary conditions have been taken by:

$$u = 0 \text{ on } \Gamma, C = 0 \text{ on } \Gamma. \quad (5)$$

The nonlinear advection - diffusion - reaction problem for stationary Bingham fluid is modeled by the partial differential system below which consists of to find:

- the velocity field  $u = (u_i) : \Omega \longrightarrow \mathbb{R}^n$ ,
- the field of constraints  $\sigma = (\sigma_{ij}) : \Omega \longrightarrow \mathbb{S}^n$ ,
- the concentration  $C : \Omega \longrightarrow \mathbb{R}$ ,

that verify the equations ((1)-(5)).

#### 4. Variational formulation

To determine the variational formulation that corresponds to the problem ((1)-(5)). We consider the following space  $H$  defined by

$$H = \{v \in H_0^1(\Omega)^3 \mid \operatorname{div} v = 0\},$$

Where  $H$  is a Hilbert space with a scalar product and an induced norm, respectively,

$$(u, v)_H = (u_i, v_i)_{H^1(\Omega)}, \quad \|u\|_H = \|u\|_{H^1(\Omega)} \quad \forall u, v \in H.$$

The set  $H$  is Banach space. We introduce the following operators

$$\begin{aligned} B : H^3 &\rightarrow \mathbb{R}, & B(\mathbf{u}, \mathbf{v}, \mathbf{w}) &= \int_{\Omega} \mathbf{u} \cdot \nabla \mathbf{v} \cdot \mathbf{w} \, dx \\ E : H^1(\Omega)^2 \times H &\rightarrow \mathbb{R}, & E(\theta, \tau, \mathbf{v}) &= \int_{\Omega} \theta \nabla \tau \cdot \mathbf{v} \, dx. \end{aligned}$$

We assume the following hypothesis

$$\begin{aligned} \forall x \in \Omega, \quad \mu(\cdot, x) &\in C^0(\mathbb{R}) \quad \text{and} \\ \exists \mu_1, \mu_2 > 0 : \mu_1 &\leq \mu(y, x) \leq \mu_2 \quad \forall y \in \mathbb{R}, \forall x \in \Omega. \end{aligned} \tag{6}$$

$$\begin{aligned} \forall x \in \Omega, \quad g(\cdot, x) &\in C^0(\mathbb{R}) \quad \text{and} \\ \exists g_0 > 0 : 0 &\leq g(y, x) \leq g_0 \quad \forall y \in \mathbb{R}, \forall x \in \Omega. \end{aligned} \tag{7}$$

$$\eta > \eta_* > 0. \tag{8}$$

$$R(\cdot, x) : \Omega \times \mathbb{R} \rightarrow \mathbb{R} \quad \text{is L Lipschitzian and } R(\cdot, x) \in L^2(\Omega \times \mathbb{R}) \tag{9}$$

Multiplying (1) by a test function  $(v - u) \in H$ , we get

$$\int_{\Omega} u \nabla u (v - u) = \int_{\Omega} \operatorname{div}(\sigma)(v - u) + \int_{\Omega} f(v - u). \tag{10}$$

Using a generalized Green formula, we obtain

$$\int_{\Omega} u \nabla u (v - u) = \int_{\Gamma} \sigma(v - u) - \int_{\Omega} \sigma D(v - u) + \int_{\Omega} f(v - u) \tag{11}$$

Multiplying (1) by  $D(v - u)$ , we obtain

$$\begin{aligned} \sigma^D D(v - u) &= 2\mu(C)D(u)D(v - u) + g(C) \frac{D(u)}{|D(u)|} D(v - u) \\ &= 2\mu(C)D(u)D(v - u) + g(C) \frac{D(u)}{|D(u)|} D(v) - g(C)|D(u)| \\ &\leq 2\mu(C)D(u)D(v - u) + g(C)|D(v)| - g(C)|D(u)| \end{aligned}$$

In addition,

$$\sigma^D D(v - u) = \left(\sigma - \frac{1}{n} \operatorname{Tr}(\sigma) I_D\right) D(v - u).$$

Then,

$$ID(v) = \sum_{i=1}^n nD_{ii}(v_i) = \sum_{i=1}^n \frac{\partial v_i}{\partial x_i} = \operatorname{div} v = 0,$$

$$ID(v - u) = 0 \implies \sigma^D D(v - u) = \sigma D(v - u),$$

$$\sigma D(v - u) \leq 2\mu(C)D(u)D(v - u) + g(C)|D(v)| - g(C)|D(u)|.$$

We replace this result in the equation (11), we obtain the following variational formulation

$$\int_{\Omega} u \nabla u (v - u) + 2 \int_{\Omega} \mu(C)D(u)D(v - u) + g(C)(|D(v)| - |D(u)|) \geq \int_{\Omega} f(v - u),$$

$$\forall v \in H, \tag{12}$$

$$\int_{\Omega} \nabla C \varphi u + \int_{\Omega} \eta(C) \nabla C \nabla \varphi = \int_{\Omega} R(C) \varphi, \quad \forall \varphi \in H_0^1(\Omega). \tag{13}$$

## 5. Existence of the solution

The proof of existence is based on the application of Schauder's fixed point theorem, using in this framework two auxiliary existence results. The first result was obtained in [Kim 1987, Kim 2002], which consists of the existence of a local solution  $u_{\lambda} \in H$  of problem (12) for each  $\lambda \in H_0^1$ , satisfying  $\|u_{\lambda}\|_H \leq c_1$ , where  $c_1$  is a positive constant does not depend on  $\lambda$ . The second is a result which shows that for  $u_{\lambda} \in H$  is a solution of the problem (12), then there exists a unique solution  $C_{\lambda} \in H_0^1$  of the problem (13), satisfying even  $\|C_{\lambda}\|_{H_0^1} \leq c_2$ , where  $c_2$  is a positive constant does not depend on  $\lambda$ .

To begin, for the operator  $B$  and  $E$ , we have the following proprieties

**Lemma 5.1.** *See [Lions 1969]*

- (i) *The operator  $B$  is trilinear, continuous on  $H^3$  and for all  $(\mathbf{u}, \mathbf{v}, \mathbf{w}) \in H^3$  we have*

$$B(\mathbf{u}, \mathbf{v}, \mathbf{w}) = -B(\mathbf{u}, \mathbf{w}, \mathbf{v}). \tag{14}$$

- (ii) *The operator  $E$  is trilinear, continuous on  $H_0^1(\Omega)^3$  and*

$$E(\theta, \tau, \mathbf{v}) = -E(\tau, \theta, \mathbf{v}) \text{ for all } (\theta, \tau, \mathbf{v}). \tag{15}$$

We have a first result of existence

**Lemma 5.2.** *For  $\lambda \in H_0^1(\Omega)$ , it exists a unique solution  $u_\lambda$  of the problem*

$$\begin{aligned} B(u_\lambda, u_\lambda, v) + 2 \int_{\Omega} \mu(\lambda) \varepsilon(u_\lambda) \varepsilon(v - u_\lambda) + \\ g(\lambda) |\varepsilon(v)| - g(\lambda) |\varepsilon(u_\lambda)| \geq \int_{\Omega} f(v - u_\lambda) \quad \forall v \in H, \end{aligned} \quad (16)$$

satisfy

$$\|u_\lambda\|_H \leq c_1, \quad (17)$$

where  $c_1$  is a positive constant does not depend on  $\lambda$ .

**Theorem 5.1.** *Let  $u_\lambda \in H$  is the solution of problem (16). Then, it exists a unique solution  $C_\lambda \in H$  of*

$$\begin{aligned} E(C_\lambda, \phi, u_\lambda) + \int_{\Omega} \eta(\lambda) \nabla C_\lambda \nabla \phi dx \\ = \int_{\Omega} R(C_\lambda) \phi dx \quad \forall \phi \in H_0^1(\Omega). \end{aligned} \quad (18)$$

and satisfy

$$\|C_\lambda\|_{H_0^1(\Omega)} \leq c_2, \quad (19)$$

where  $c_2$  is a positive constant does not depend on  $\lambda$ .

*Proof.* Let  $\mathcal{E}$  the following bilinear form

$$\mathcal{E} : H_0^1(\Omega) \times H_0^1(\Omega) \rightarrow \mathbb{R}, \quad (\psi, \varphi) \mapsto E(\psi, \varphi, u_\lambda) + \int_{\Omega} \eta(\lambda) \nabla \psi \nabla \varphi dx$$

The trilinear operator  $E$  is continuous on  $H_0^1(\Omega) \times H_0^1(\Omega) \times H$ . In fact, using the Hölder inequality and Poincaré theorem, we have

$$\begin{aligned} \left| \int_{\Omega} \nabla \psi \varphi \cdot v dx \right| &\leq \| \nabla \psi \varphi \|_{L^2(\Omega)} \| v \|_{L^2(\Omega)} \\ &\leq \| \nabla \psi \|_{L^2(\Omega)} \| \varphi \|_{L^2(\Omega)} \| v \|_{L^2(\Omega)} \\ &\leq \| \psi \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)} \| v \|_H. \end{aligned}$$

Then, we obtain

$$\left| \int_{\Omega} \nabla \psi \varphi \cdot v dx \right| \leq \| \psi \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)} \| v \|_H. \quad (20)$$

Consider  $r \in L^2(\Omega)$  and the following linear problem

$$\mathcal{E}(C_{\lambda r}, \varphi) = \int_{\Omega} r \varphi dx \quad \forall \varphi \in H_0^1(\Omega). \quad (21)$$

Using the continuity of  $E$ , we can simply show that  $\mathcal{E}$  is continuous.

$$\begin{aligned} |\mathcal{E}(C_{\lambda r}, \varphi)| &= \left| \int_{\Omega} \nabla C_{\lambda r} \varphi \cdot u_{\lambda} + \int_{\Omega} \eta(\lambda) \nabla C_{\lambda r} \cdot \nabla \varphi dx \right| \\ &\leq \left| \int_{\Omega} \nabla C_{\lambda r} \varphi \cdot u_{\lambda} \right| + \left| \int_{\Omega} \eta(\lambda) \nabla C_{\lambda r} \cdot \nabla \varphi dx \right|. \end{aligned}$$

Using Hölder inequality we get

$$\begin{aligned} &\leq \| \nabla C_{\lambda r} \varphi \|_{L^2(\Omega)} \| u_{\lambda} \|_{L^2(\Omega)} + \eta(\lambda) \| \nabla C_{\lambda r} \|_{L^2(\Omega)} \| \nabla \varphi \|_{L^2(\Omega)} \\ &\leq \| \nabla C_{\lambda r} \|_{L^2(\Omega)} \| \varphi \|_{L^2(\Omega)} \| u_{\lambda} \|_{L^2(\Omega)} + \eta(\lambda) \| \nabla C_{\lambda r} \|_{L^2(\Omega)} \| \nabla \varphi \|_{L^2(\Omega)}. \end{aligned}$$

Using Poincaré theorem, then

$$\leq C_p \| C_{\lambda r} \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)} \| u_{\lambda} \|_H + \eta(\lambda) \| C_{\lambda r} \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)}.$$

We have also  $\| u_{\lambda} \|_H \leq c_1$ ,

$$\leq c_1 C_p \| C_{\lambda r} \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)} + \eta(\lambda) \| C_{\lambda r} \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)}.$$

Then, we obtain

$$|\mathcal{E}(C_{\lambda r}, \varphi)| \leq K \| C_{\lambda r} \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)},$$

with  $K = (c_1 C_p + \eta(\lambda))$ .

In addition, by (15), we remark that  $E(u, u, v) = 0$ . Then,

$\mathcal{E}(C_{\lambda r}, C_{\lambda r}) = \int_{\Omega} \eta(\lambda) | \nabla C_{\lambda r} |^2 dx$ , using in addition that (8), we have the coercivity of  $\mathcal{E}$ .

$$\mathcal{E}(C_{\lambda r}, C_{\lambda r}) \geq \eta_* \| C_{\lambda r} \|_{H_0^1(\Omega)}^2.$$

Adding that  $l(\varphi) = \int_{\Omega} r \varphi dx$  is linear continuous by Poincaré formula, we have

$$| l(\varphi) | = \left| \int_{\Omega} r \varphi dx \right| \leq \| r \|_{L^2(\Omega)} \| \varphi \|_{L^2(\Omega)} \leq C_p^2 \| r \|_{H_0^1(\Omega)} \| \varphi \|_{H_0^1(\Omega)}.$$

Using Lax-Milgram theorem on  $H^1(\Omega) \times H^1(\Omega)$ , then, the problem (23) have a unique solution  $C_{\lambda_r} \in H_0^1(\Omega)$ .

To get auxiliary solution of problem (18), we consider the following operator

$$\begin{cases} \mathcal{L} : L^2(\Omega) \rightarrow L^2(\Omega), \\ \mathcal{L}r = R(C_{\lambda_r}). \end{cases} \quad (22)$$

Since  $\eta$  verify (8), for  $r_1, r_2 \in L^2(\Omega)$ , we have

$$|\mathcal{L}r_1 - \mathcal{L}r_2| = |R(C_{\lambda_{r_1}}) - R(C_{\lambda_{r_2}})| \leq L |C_{\lambda_{r_1}} - C_{\lambda_{r_2}}|.$$

We denote  $C_1 = C_{\lambda_{r_1}}$  and  $C_2 = C_{\lambda_{r_2}}$ , respectively, solutions of the following problems

$$\mathcal{E}(C_{\lambda_{r_1}}, \varphi) = \int_{\Omega} r_1 \varphi dx \quad \forall \varphi \in H_0^1(\Omega), \quad (23)$$

$$\mathcal{E}(C_{\lambda_{r_2}}, \varphi) = \int_{\Omega} r_2 \varphi dx \quad \forall \varphi \in H_0^1(\Omega), \quad (24)$$

Subtracting the equations (23) et (24), and using  $\varphi = C_{\lambda_{r_1}} - C_{\lambda_{r_2}}$  as a test function, we get

$$\mathcal{E}(C_1 - C_2, C_1 - C_2) = \int_{\Omega} (r_1 - r_2)(C_1 - C_2) dx,$$

$$\int_{\Omega} \nabla(C_1 - C_2)(C_1 - C_2) \cdot u_{\lambda} + \int_{\Omega} \eta(\lambda) |\nabla(C_1 - C_2)|^2 dx = \int_{\Omega} (r_1 - r_2)(C_1 - C_2) dx,$$

$$\begin{aligned} \text{As } \eta \text{ verify (8), we obtain } & \int_{\Omega} \nabla(C_1 - C_2)(C_1 - C_2) \cdot u_{\lambda} + \int_{\Omega} \eta_*(\lambda) |\nabla(C_1 - C_2)|^2 dx \\ & \leq \int_{\Omega} \nabla(C_1 - C_2)(C_1 - C_2) \cdot u_{\lambda} + \int_{\Omega} \eta(\lambda) |\nabla(C_1 - C_2)|^2 dx \\ & = \int_{\Omega} (r_1 - r_2)(C_1 - C_2) dx. \end{aligned}$$

Using  $E(u, u, v) = 0$ , then,  $\int_{\Omega} \nabla(C_1 - C_2)(C_1 - C_2) \cdot u_{\lambda} > 0$ . Therefore

$$\eta_* \|C_1 - C_2\|_{H_0^1(\Omega)}^2 dx \leq \|r_1 - r_2\|_{L^2(\Omega)} \|C_1 - C_2\|_{L^2(\Omega)},$$

$$\eta_* \|C_1 - C_2\|_{H_0^1(\Omega)}^2 \leq \|r_1 - r_2\|_{L^2(\Omega)} C_p \|C_1 - C_2\|_{H_0^1(\Omega)},$$

$$\|C_1 - C_2\|_{H_0^1(\Omega)} \leq \frac{C_p}{\eta_*} \|r_1 - r_2\|_{L^2(\Omega)}.$$

We have

$$|\mathcal{L}r_1 - \mathcal{L}r_2| \leq L |C_1 - C_2|.$$

Then,

$$\begin{aligned} \|\mathcal{L}r_1 - \mathcal{L}r_2\|_{L^2(\Omega)}^2 &\leq L^2 \|C_1 - C_2\|_{L^2(\Omega)}^2 \\ &\leq L^2 C_p^2 \|C_1 - C_2\|_{H_0^1(\Omega)}^2 \leq \frac{L^2 C_p^4}{\eta_*^2} \|r_1 - r_2\|_{L^2(\Omega)}^2 \\ \|\mathcal{L}r_1 - \mathcal{L}r_2\|_{L^2(\Omega)}^2 &\leq \frac{L^2}{c_1} \|r_1 - r_2\|_{L^2(\Omega)}^2, \text{ with } c_1 = \frac{\eta_*}{C_p^2}. \end{aligned}$$

We can write

$$\begin{aligned} \|\mathcal{L}r_1 - \mathcal{L}r_2\|_{L^2(\Omega)}^2 &\leq \frac{L^2}{c_1} \|r_1 - r_2\|_{L^2(\Omega)}^2, \\ \|\mathcal{L}^2 r_1 - \mathcal{L}^2 r_2\|_{L^2(\Omega)}^2 &\leq \frac{L^2}{c_1} \|r_1 - r_2\|_{L^2(\Omega)}^2, \\ &\vdots \\ \|\mathcal{L}^m r_1 - \mathcal{L}^m r_2\|_{L^2(\Omega)}^2 &\leq \frac{L^2}{c_1} \|r_1 - r_2\|_{L^2(\Omega)}^2. \end{aligned}$$

The operator  $\mathcal{L}$  is contracting in the space  $L^2(\Omega)$ . So, according to Banach's fixed point theorem  $\mathcal{L}^m$  admits a fixed point that we denote  $\tilde{r} \in L^2(\Omega)$  which gives  $\mathcal{L}^m \tilde{r} = \mathcal{L} \tilde{r}$ .

Then, the fixed-point uniqueness gives  $\mathcal{L} \tilde{r} = \tilde{r}$  which also gives  $\mathcal{L} \tilde{r} = R(C_{\tilde{r}}) = \tilde{r}$ , which implies that the equation (18) has a unique solution  $C_\lambda \in H_0^1(\Omega)$ .

Finding now the estimate (19). We choose  $C_\lambda$  as a test function of the equation (18). So we have

$$\int_{\Omega} \nabla C_\lambda C_\lambda \cdot u_\lambda + \int_{\Omega} \eta(\lambda) |\nabla C_\lambda|^2 dx = \int_{\Omega} R(C_\lambda) C_\lambda dx, \forall C_\lambda \in H_0^1(\Omega)$$

Using the Hölder inequality, we obtain

$$\begin{aligned} \int_{\Omega} \nabla C_\lambda C_\lambda \cdot u_\lambda + \eta_*(\lambda) \|C_\lambda\|_{H_0^1(\Omega)}^2 &\leq \|R\|_{L^2(\Omega)} \|C_\lambda\|_{L^2(\Omega)} \\ \eta_*(\lambda) \|C_\lambda\|_{H_0^1(\Omega)}^2 &\leq \|R\|_{L^2(\Omega)} \|C_\lambda\|_{L^2(\Omega)}. \end{aligned}$$

Using the Poincaré inequality, we obtain

$$\eta_*(\lambda) \|C_\lambda\|_{H_0^1(\Omega)}^2 \leq C_p \|R\|_{L^2(\Omega)} \|C_\lambda\|_{H_0^1(\Omega)}.$$

Therefore,

$$\|C_\lambda\|_{H_0^1(\Omega)} \leq \frac{C_P}{\eta_*} \|R\|_{L^2(\Omega)}.$$

So, we obtain

$$\|C_\lambda\|_{H_0^1(\Omega)} \leq c_2.$$

□

**Theorem 5.2.**

*The problem (16)-(18) has a locale solution  $(u, C)$  having the following regularity*

$$u \in H \cap W_0^{1,6}(\Omega)^3 \text{ et } C \in H_0^1(\Omega) \cap L^2(\Omega) \quad (25)$$

*It is easy to check that the regularity (25) using in particular the Theorem of intermediate values and Theorem extensions of Sobolev, that after a possible modification on a set of zero measure, the solutions are:*

$$u \in H \cap C^{0, \frac{1}{2}}(\Omega)^3 \text{ et } C \in L^2(\Omega). \quad (26)$$

*Proof.* We will use the fixed point Theorem of Schauder, by considering the the following closed convex ball:

$$\mathcal{B} = \{\lambda \in H_0^1(\Omega) / \|\lambda\|_{H_0^1(\Omega)} \leq c_2\}$$

Soit

$$\Lambda : \mathcal{B} \rightarrow \mathcal{B}$$

$$\lambda \mapsto \Lambda(\lambda) = C_\lambda \in \mathcal{B},$$

our interest is to check the compactness of the  $\Lambda$  operator. Therefore, we consider  $\lambda_n$  in  $\mathcal{B}$  and consequently this sequence is limited, and since it is a Hilbert space, we can extract a subsequence that we note  $\lambda_n$  and  $C_n = C_{\lambda_n}$  such that

$$\lambda_n \rightharpoonup \lambda \text{ dans } H^1(\Omega)$$

$$\lambda_n \xrightarrow{*} \lambda \text{ dans } L^2(\Omega)$$

$$C_n \rightharpoonup C_\lambda \text{ dans } H^1(\Omega)$$

$$C_n \xrightarrow{*} C_\lambda \text{ dans } L^2(\Omega).$$

As  $C_n$  is a solution of the following problem

$$\begin{aligned} E(C_n, \phi, u_n) + \int_{\Omega} \eta(\lambda_n) \nabla C_n \nabla \phi dx \\ = \int_{\Omega} R(C_n) \phi dx \quad \forall \phi \in H_0^1(\Omega). \end{aligned} \quad (27)$$

with  $u_n$  verifies the following inequality

$$\begin{aligned} B(u_n, u_n, v) + 2 \int_{\Omega} \mu(\lambda_n) \varepsilon(u_n) \varepsilon(v - u_n) + \\ g(\lambda_n) |\varepsilon(v)| - g(\lambda_n) |\varepsilon(u_n)| \geq \int_{\Omega} f(v - u_n) \quad \forall v \in H, \end{aligned} \quad (28)$$

The lemma (5.2) gets a unique strong solution  $u_n \in H$ . Then we can extract a subsuite is also noted by  $u_n$  satisfies

$$u_n \xrightarrow{*} u_\lambda \text{ in } H \cap C^{0, \frac{1}{2}}(\Omega)^3.$$

Then, the injection  $W_0^{1,6}(\Omega)^3 \subset C^0(\bar{\Omega})$  is compact, using the compactness theorem of Aubin, as we can extract suites  $\lambda_n, C_n, u_n$  (see [Brezis 1968, Lions 1969] ) such that

$$\begin{aligned} \lambda_n &\rightarrow \lambda \text{ dans } L^2(\Omega) \\ C_n &\rightarrow C_\lambda \text{ dans } L^2(\Omega) \\ u_n &\rightarrow u_\lambda \text{ dans } C^0(\bar{\Omega})^3 \end{aligned}$$

$$\begin{aligned} B(u_n, u_n, v) + 2 \int_{\Omega} \mu(\lambda_n) \varepsilon(u_n) \cdot \varepsilon(v) dx + g(\lambda_n) |\varepsilon(v)| - \int_{\Omega} f(v) dx \geq \\ 2 \int_{\Omega} \mu(\lambda_n) |\varepsilon(u_n)|^2 dx + \int_{\Omega} g(\lambda_n) |\varepsilon(u_n)| dx - \int_{\Omega} f(u_n) dx \quad \forall v \in H. \end{aligned} \quad (29)$$

Following [Lions 1969], we have  $B$  is continuous, and by passing to the limit

$$B(u_n, u_n, v) \rightarrow B(u_\lambda, u_\lambda, v), \quad \forall v \in H. \quad (30)$$

Using the \* convergence de  $u_n \rightharpoonup u_\lambda$  dans  $H \cap C^{0, \frac{1}{2}}(\Omega)^3$  and and the fact that  $\lambda_n \rightarrow \lambda$  in  $\Omega$ , l'hypothse (6), (7) and according to the dominated convergence

theorem, we obtain for  $v \in H$  the following result as

$$\begin{aligned} \int_{\Omega} \mu(\lambda_n) \varepsilon(u_n) \cdot \varepsilon(v) dx &\rightarrow \int_{\Omega} \mu(\lambda) \varepsilon(u_\lambda) \cdot \varepsilon(v) dx \\ \int_{\Omega} f(u_n) dx &\rightarrow \int_{\Omega} f(u_\lambda) dx. \end{aligned} \quad (31)$$

Adding, we have

$$\begin{aligned} &2 \int_{\Omega} \mu(\lambda_n) |\varepsilon(u_n)|^2 dx + \int_{\Omega} g(\lambda_n) |\varepsilon(u_n)| dx \\ &= 2 \int_{\Omega} \mu(\lambda) |\varepsilon(u_n)|^2 dx + 2 \int_{\Omega} (\mu(\lambda_n) - \mu(\lambda)) |\varepsilon(u_n)|^2 dx \\ &\quad + \int_{\Omega} g(\lambda) |\varepsilon(u_n)| dx + \int_{\Omega} (g(\lambda_n) - g(\lambda)) |\varepsilon(u_n)| dx. \end{aligned}$$

Then,  $\lambda \rightarrow \lambda \in \Omega$ , the functions  $\mu$  et  $g$  are continuous, and due to the low semi-continuity in the space  $H$ , continuity and convexity of functional  $\int_{\Omega} \mu(\lambda) |\varepsilon(v)|^2 dx$  et  $\int_{\Omega} g(\lambda) |\varepsilon(v)| dx$ , we deduct using Fatou lemma that

$$\begin{aligned} &2 \liminf \int_{\Omega} \mu(\lambda_n) |\varepsilon(u_n)|^2 dx + \liminf \int_{\Omega} g(\lambda_n) |\varepsilon(u_n)| dx \\ &\geq 2 \int_{\Omega} \mu(\lambda) |\varepsilon(u_\lambda)|^2 dx + \int_{\Omega} g(\lambda) |\varepsilon(u_\lambda)| dx. \end{aligned} \quad (32)$$

On the other hand, the fact that

$$u_n, u_\lambda \in H$$

and after (29) all lead to the conclusion that  $u$  is a solution of (16). Moreover, it is necessary to pass by the limit in the equation (27). To do this, for every  $\phi \in H^1(\Omega)$ , we have

$$|E(C_n, \phi, u_n) - E(C_\lambda, \phi, u_\lambda)| = \left| \int_{\Omega} (u_n \cdot \nabla(C_n - C_\lambda)\phi + (u_n - u_\lambda) \cdot (\nabla C_\lambda)\phi) dx \right|.$$

By the lemma (5.1), we have

$$\begin{aligned} |E(C_n, \phi, u_n) - E(C_\lambda, \phi, u_\lambda)| &\leq \left| \int_{\Omega} (C_n - C_\lambda) u_n \cdot \nabla \phi dx \right| \\ &\quad + \left| \int_{\Omega} C_\lambda (u_n - u_\lambda) \cdot \nabla \phi dx \right| \end{aligned} \quad (33)$$

Elsewhere, using the regularity (26), we obtain the following inequality

$$\begin{aligned} \int_{\Omega} |u_n \cdot \nabla \phi|^2 dx &\leq \|u_n\|_{C^0(\Omega)}^2 \|\phi\|_{H^1(\Omega)}^2 \\ &\in \phi \in H^1(\Omega). \end{aligned}$$

Then, using the result of the convergence  $C_n \rightarrow C_\lambda$  dans  $H^1(\Omega)$ , que

$$|E(C_n, \phi, u_n) - E(C_\lambda, \phi, u_\lambda)| \rightarrow 0.$$

In addition,  $C_\lambda \in H_0^1(\Omega) \cap L^2(\Omega)$ , gives that  $C_\lambda \in L^4(\Omega)$ . Therefore,

$$C_\lambda \nabla \phi \in L^{\frac{4}{3}}(\Omega)^3, \forall \phi \in H^1(\Omega) \quad (34)$$

Since, de  $u_n \rightarrow \lambda$  in  $C^0(\bar{\Omega})^3$ , we deduce

$$u_n \rightarrow u_\lambda \text{ dans } L^4(\Omega)^3. \quad (35)$$

Combining (35) avec (34). Then,

$$|E(C_n, \phi, u_n) - E(C_\lambda, \phi, u_\lambda)| \rightarrow 0.$$

We obtain

$$|E(C_n, \phi, u_n) - E(C_\lambda, \phi, u_\lambda)| \rightarrow 0 \forall \phi \in H^1(\Omega). \quad (36)$$

Then,  $\lambda_n \rightarrow \lambda$  p. p.  $\Omega$ , the lower limit is easily obtained, using  $C_n \rightarrow C_\lambda$  in  $H^1(\Omega)$ , the assumptions (6), (7) and the use of the dominated convergence theorem

$$\begin{aligned} \int_{\Omega} \eta(\lambda_n) \nabla C_n \cdot \nabla \phi dx &\rightarrow \int_{\Omega} \eta(\lambda) \nabla C_\lambda \cdot \nabla \phi dx \\ &\forall \phi \in H^1(\Omega) \end{aligned} \quad (37)$$

So that the passing to the limit in the equation (29) remains to verify that

$$\int_{\Omega} R(C_n) \phi dx \rightarrow \int_{\Omega} R(C_\lambda) \phi dx, \forall \phi \in H^1(\Omega) \quad (38)$$

This is obtained taking into account that the  $R$  Lipschitzian function and  $C_n \rightarrow C_\lambda$  in  $L^2(\Omega)$  p. p. in  $\Omega$ .

Proving now the following propriety

$$C_n \rightarrow C_\lambda \text{ dans } H_0^1(\Omega) \cap L^2(\Omega) \quad (39)$$

To do this, subtracting the two equations (18) and (27) and using  $\phi = C_n - C_\lambda$ , as a test function in the equation obtained, we obtain

$$\begin{aligned} & \frac{1}{2} \|C_n - C_\lambda\|_{H^1(\Omega)}^2 + E(C_n, C_n - C_\lambda, u_n) - E(C_\lambda, C_n - C_\lambda, u_n) + \\ & \int_{\Omega} (\eta(\lambda_n) \nabla C_n - \eta(\lambda) \nabla C_\lambda) \cdot \nabla (C_n - C_\lambda) dx = \int_{\Omega} (R(C_n) - R(C_\lambda))(C_n - C_\lambda) dx. \end{aligned}$$

Using the hypotheses (6) and (7), we have

$$\begin{aligned} & \frac{1}{2} \|C_n - C_\lambda\|_{H^1(\Omega)}^2 + \int_{\Omega} \eta(\lambda_n) |\nabla (C_n - C_\lambda)|^2 dx \\ & \leq \int_{\Omega} |\eta(\lambda_n) - \eta(\lambda)| |\nabla C_\lambda| |\nabla (C_n - C_\lambda)| dx \\ & \quad + E(C_n, C_n - C_\lambda, u_n) - E(C_\lambda, C_n - C_\lambda, u_n) \\ & = L \int_{\Omega} |C_n - C_\lambda|^2 dx. \end{aligned} \quad (40)$$

In addition, (36) shows that

$$E(C_n, C_n - C_\lambda, u_n) - E(C_\lambda, C_n - C_\lambda, u_n) \rightarrow 0. \quad (41)$$

Therefore, with the equation (40), we get the assumptions (6), (7) and the convergence results

$$\lambda_n \rightarrow \lambda \text{ in } L^2(\Omega),$$

$$C_n \rightarrow C_\lambda \text{ in } L^2(\Omega),$$

and the fact that  $|\nabla C_\lambda| |\nabla (C_n - C_\lambda)|$  belongs to a bounded  $L^1(\Omega)$ , seeing Lebesgue's theorem of dominated convergence.

$$C_n \rightarrow C_\lambda \text{ dans } H^1(\Omega) \cap L^2(\Omega). \quad (42)$$

By subtracting, we deduce

$$\int_{\Omega} |u_n \cdot \nabla C_n - u_\lambda \cdot \nabla C_\lambda|^2 dx \leq c \int_{\Omega} |u_n|^2 |\nabla (C_n - C_\lambda)|^2 dx + c \int_{\Omega} |u_n - u_\lambda|^2 |\nabla C_\lambda|^2 dx.$$

The use of Hölder inequality and (26), we have

$$\int_{\Omega} |u_n \cdot \nabla C_n - u_\lambda \cdot \nabla C_\lambda|^2 dx \leq c \|u_n\|_{C^0(\bar{\Omega})}^2 \|C_n - C_\lambda\|_{H^1(\Omega)} + c \|u_n - u_\lambda\|_{C^0(\bar{\Omega})}^2 \|C_\lambda\|_{H^1(\Omega)}^2 \quad (43)$$

As a result,  $C_n$  converges strongly to  $C_\lambda$  in the space  $H_0^1(\Omega) \cap L^2(\Omega)$ . Then the  $\Lambda$  operator is compact. Using Schauder fixed point theory, we have at least one fixed point  $\bar{\lambda} = C_\lambda$  in  $H_0^1(\Omega) \cap L^2(\Omega)$ .

Finally, we conclude that the function  $(u_{\bar{\lambda}}, C_{\bar{\lambda}}) \in H \cap W_0^{1,6}(\Omega)^3 \times H_0^1(\Omega) \cap L^2(\Omega)$  solve the problem ((16), (18)).  $\square$

## 6. Numerical treatment

### 6.1. Problem regularized

The basic idea is to use one of the regularization methods to approximate the apparent viscosity  $\mu_e$  of the law of the original behavior in order to avoid the discontinuity of this viscosity by  $\mu_{e,\varepsilon}$ , so that we may overcome the instability of calculation. In literature, the Papanastasiou model of regularization has been widely used in numerical simulations of flows of viscoplastic fluids [Glowinski and Ciarlet 2010, Messelmi 2014, Paramasivam et al. 2010].

The Bingham's law (3) is equivalent to the following system

$$\sigma^D = 2\mu_e D(u), \quad (44)$$

$$\mu_e = \mu + \frac{g}{2|D(u)|}, \quad (45)$$

The above system is obviously well suited to the ceded regions, that is to say the regions where the deformation rate tensor  $D(u)$  is non-zero. However, if  $\|D(u)\| \rightarrow 0$ , the exponential model of Papanastasiou is as follows:

$$\mu_{e,\varepsilon} = \mu + \frac{g}{2|D(u)|} (1 - \exp(\frac{-2}{\varepsilon}|D(u)|)).$$

So the Papanastasiou-Bingham model is as follows:

$$\sigma^D = 2\mu_{e,\varepsilon} D(u) = 2\mu D(u) + g \frac{D(u)}{|D(u)|} (1 - \exp(\frac{-2}{\varepsilon}|D(u)|)). \quad (46)$$

We will consider now the problem ((1)-(5) with replacing (3) by (46)). The goal is treated this problem numerically simulation. To do this, here and below, we suppose that fluid is stationary, laminar, viscoplastic and incompressible. We suppose that the volume forces are negligible in front of the forces of viscosity, the blood diffusion coefficient  $\eta$ , the yield stress  $g$  and the blood viscosity  $\mu$  don't depend on the concentration, except the Reaction function that is non linear and generally has an unknown expression. According to the bio-mechanical problem ((1)-(5) with replacing (3) by (46)), the variational formulation is as follows

$$+ \int_{\Omega} \mathbf{u} \nabla \mathbf{u} v + \int_{\Omega} 2\mu D(\mathbf{u}) : D(v) - \int_{\Omega} p \nabla \cdot v - \int_{\Omega} q \nabla \cdot \mathbf{u} + \int_{\Omega} g \frac{D(\mathbf{u}) : D(v)}{(D(\mathbf{u}) : D(\mathbf{u}))^{\frac{1}{2}}} (1 - \exp(\frac{-2}{\varepsilon} (D(\mathbf{u}) : D(\mathbf{u}))^{\frac{1}{2}})) = 0, \forall v \in V_1 \quad (47)$$

$$\int_{\Omega} \mathbf{u} \nabla C \phi + \int_{\Omega} \eta \nabla C \nabla \phi = \int_{\Omega} R(C) \phi, \forall \phi \in H_0^1(\Omega) . \quad (48)$$

In order to solving numerically the non-linear equations ((47),(48)), we will use the Newton algorithm. For this, we need to linearize these equations. To do this, we calculate the differential of each weak forms at point  $\mathbf{u}$ , since the differential in general is a linear application. Then, we solve them in this form:

$$F(u + \delta) = F(u) + DF(u)\delta + o(\delta). \quad (49)$$

For the variational problem, we obtain

$$\left\{ \begin{array}{l} F1(u, p) = \int_{\Omega} u \nabla u v + \int_{\Omega} 2\mu D(u) : D(v) \\ + \int_{\Omega} g \frac{D(u) : D(v)}{(D(u) : D(u))^{\frac{1}{2}}} (1 - \exp(\frac{-2}{\varepsilon} (D(u) : D(u))^{\frac{1}{2}})) \\ - \int_{\Omega} p \nabla \cdot v - \int_{\Omega} q \nabla \cdot u \\ DF1(u, p)(\delta u, \delta p) = \int_{\Omega} (\delta u \nabla u v + u \nabla \delta u v) + \int_{\Omega} 2\mu D(\delta u) : D(v) \\ + g \frac{D(\delta u) : D(v)}{(D(u) : D(u))^{\frac{1}{2}}} (1 - \exp(\frac{-2}{\varepsilon} (D(u) : D(u))^{\frac{1}{2}})) \\ - g \frac{D(u) : D(v) D(u) : D(\delta u)}{(D(u) : D(u))^{\frac{3}{2}}} \left( 1 - \exp(\frac{-2}{\varepsilon} (D(u) : D(u))^{\frac{1}{2}}) \right) \\ + \frac{2}{\varepsilon} g \frac{D(u) : D(v) D(u) : D(\delta u)}{D(u) : D(u)} \exp(\frac{-2}{\varepsilon} (D(u) : D(u))^{\frac{1}{2}}) \\ - \int_{\Omega} \delta p \nabla \cdot v - \int_{\Omega} q \nabla \cdot \delta u \end{array} \right. \quad (50)$$

and

$$\begin{cases} F2(C) = \int_{\Omega} \mathbf{u} \nabla C \phi + \int_{\Omega} \eta \nabla C \nabla \phi - \int_{\Omega} (1 - C) C \phi, \\ DF2(C) \delta C = \int_{\Omega} \mathbf{u} \nabla \delta C \phi + \int_{\Omega} \eta \nabla \delta C \nabla \phi - \int_{\Omega} (1 - \delta C) \delta C \phi \end{cases} \quad (51)$$

## 6.2. Numerical results

This section focuses on the 2D numerical study of the effect of concentration of blood cell in fusiform aneurysms caused by the AAA diseases in 2D model. Blood is considered a non-Newtonian laminar flow, incompressible and stationary, which can present itself with a rigid wall.

### 6.2.1. Two-dimensional geometry construction

We use in this study two types of geometries of an Abdominal Aortic Aneurysms (AAA), one presents a Fusiform aneurysms caused by the AAA diseases without bifurcation. The second is more complex than the other (Fig. 6.2.1). It represents a fusiform AAA with a bifurcation at the level of the abdominal aorta which is split into two iliac arteries. The AAA models have a non dilated aorta diameter  $d = 1,8cm$ , the maximum diameter of the AAA,  $D = 2.75d$ , the aneurysm has a length,  $L = 4d$  and the diameter of each outputs of the right and left branches of the bifurcation is  $d_0 = 0.72d$ , while the order of the average velocity of blood is  $u_0 \approx 0,25 m/s$ , since it does not circulate at the same velocity throughout the body. The two meshes were built with Freefem++ software, using a sinc function as follows [ Khanafar et al. 2006 ]

$$f(x) = \frac{D - d}{4} \left( 1 + \sin\left(\frac{2\pi x}{L} - \frac{\pi}{2}\right) \right) + \frac{d}{2}, 0 \leq x \leq L (= 4d), \quad (52)$$

where  $d$  is a diameter Abdominal Aortic (AA),  $D$  is a maximum aneurysm diameter of the abdominal aortic and  $L$  is the length of the aneurysm.

The most important blood properties are: the Newtonian viscosity  $\mu_{\infty} = 0.00345 Pa.s$  and  $\mu_0 = 0.056 Pa.s$ , at high and low shear rates, respectively. In effect, the erythrocytes rolls are formed at low shear rate. But, if this rate increases, the aggregates of these globules will start to deform without the blood flowing, which implies that there is a critical shear stress (yield stress)  $g \approx$

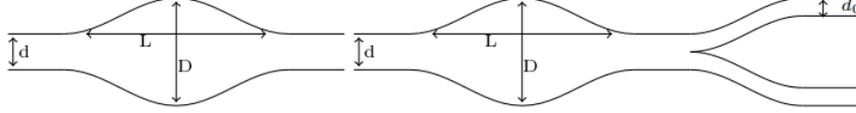


Figure 1: Fusiform aneurysms caused by the AAA diseases without and with bifurcation.

$0.003 Pa$ , below which the flow does not occur. As found in [Picart et al. 1998], that the yield stress varies from  $0.001 Pa$  to  $0.03 Pa$  for a normal blood at hematocrit 40, in general it increases with the rate of hematocrit.

The non-Newtonian blood property has a very significant effect on the velocity profiles, especially on the longitudinal section of the aorta specifically in the aneurysmal sac, without forgetting the concentration and pressure that corresponds to this velocity.

### 6.2.2. Numerical Interpretation

Let  $u_x$  and  $u_y$  the velocity component with  $x$  and  $y$  are horizontal and vertical spatial coordinates. Numerically, we found that the  $u_x$  component of the speed along  $x$  in  $[0, L]$  varies nonlinearly between  $0,015m/s$  and  $0,25m/s$ , while the  $u_y$  component of the speed varies between  $0.0034m/s$  and  $0.18m/s$  for a high rate of shear that due to a very low viscosity  $\mu = 0.00345Pa.s$ . In this state, we can conclude that blood behaves like a Newtonian fluid. This shows that the main flow is relatively dominated by the  $u_x$  component of the velocity. The resolution is done by varying the number of meshes to test the convergence using triangular finite elements. The velocity is computed throughout aneurysmal fusiform at a variable rate of shear which respectively to the deformation of the red blood cell rolls on both models of AAA, shown in Fig. 2.

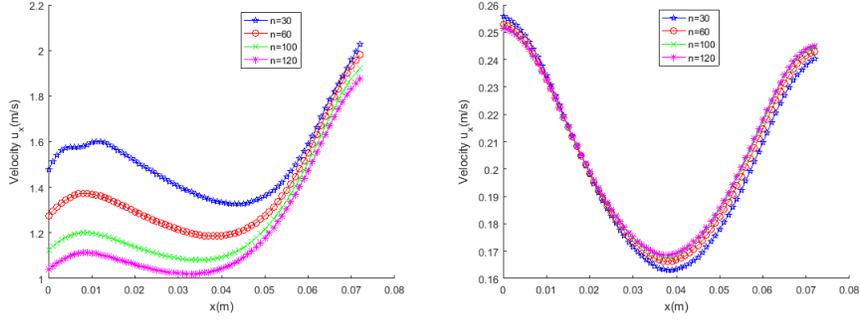


Figure 2: The velocity profiles  $u_x$  on the regime  $x \in [0, L]$  for the fusiform AAA model without bifurcation, the left figure for a low rate ( $\mu = 0.056 Pa.s$ ) and the right figure for a high rate ( $\mu = 0.00345 Pa.s$ ).

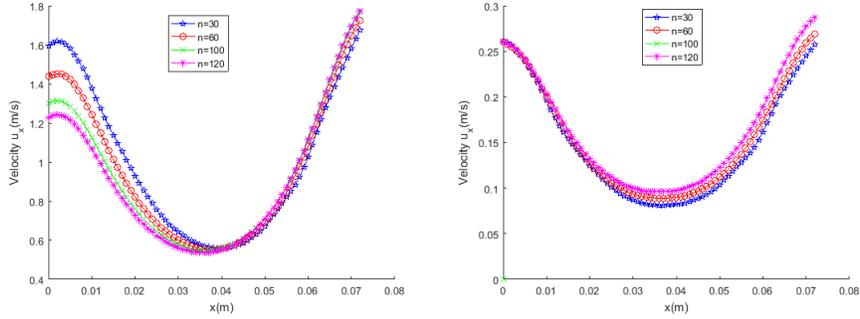


Figure 3: The velocity profiles  $u_x$  on the regime  $x \in [0, L]$  for the fusiform AAA model with bifurcation, the left figure for a low rate ( $\mu = 0.056 Pa.s$ ) and the right figure for a high rate ( $\mu = 0.00345 Pa.s$ ).

The solutions of the velocity for different numbers of meshes, (Fig.. (2) , (3)) are obtained in a compatible way, which makes it possible to conclude to the convergence. For both models with or without bifurcation of AAA disease, a remarkable change in velocity component  $u_x$  with a relatively high viscosity ( $\mu = 0.056 Pa.s$ ) can be observed, than to the case of a shear rate due to a lower viscosity ( $\mu = 0.00345 Pa.s$ ).

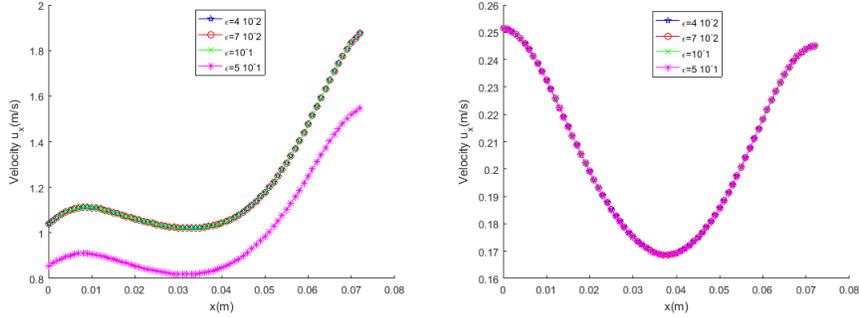


Figure 4: The velocity profiles  $u_x$  on the regime  $x \in [0, L]$  for the fusiform AAA model , the left figure for a low rate ( $\mu = 0.056 Pa.s$ ) and the right figure for a high rate ( $\mu = 0.00345 Pa.s$ ).

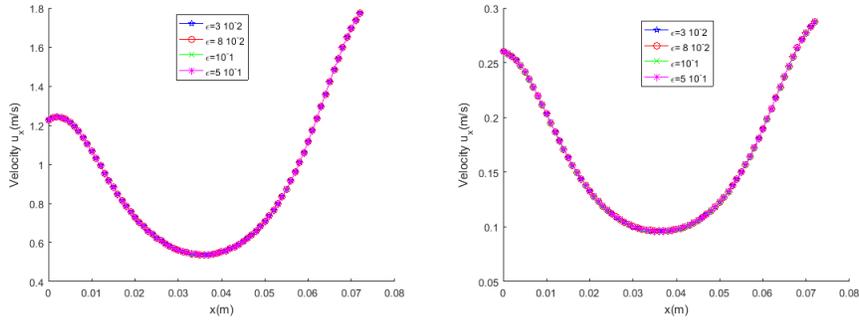


Figure 5: The velocity profiles  $u_x$  on the regime  $x \in [0, L]$  for the fusiform AAA model with bifurcation , the left figure for a low rate ( $\mu = 0.056 Pa.s$ ) and the right figure for a high rate ( $\mu = 0.00345 Pa.s$ ).

To testing some regularization values, we set the number of meshes at ( $n = 100$ ) for. The velocity component  $u_x$  for both models and in both cases low and high shear rates are confounded, see figures eqref ph3 and eqref ph4), which shows the convergents even if we let's change the regularization values. We noted in the case of AAA without bifurcation, when the shear rate is relatively low the value of  $u_x$  is almost constant in the first half of the aneurysm sac ( $x \text{ in } [0, \text{frac}L2]$ ), then this value increases remarkably in the second half. This increase does not appear in the same way when the viscosity is relatively low. On the other hand, for the bifurcated model, there is a remarkably large change

in speed for the case of a low shear rate compared with the case of a high rate.

We have noticed that the smallest regularization value we can take to get convergent solutions for the AAA model without bifurcation is  $4 \cdot 10^{-2}$  and  $3 \cdot 10^{-2}$  for the other. This can influence the reasonableness of the result obtained, since it takes the value of regularization to be small to obtain precise solutions. In what follows,, we are interested in the study of blood flow in the aneurysmal fusiform with velocity distribution analysis in the aorta.

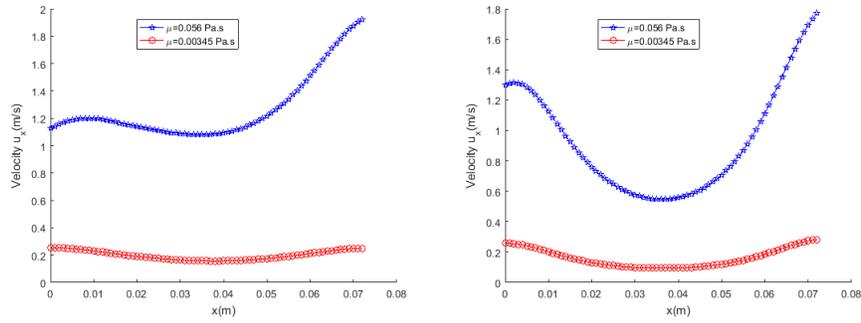


Figure 6: The velocity profiles  $u_x$  on the regime  $x \in [0, L]$  for a low and high rate, the left figure for the fusiform AAA model without bifurcation and the right figure for the fusiform AAA model with bifurcation.

Figure 7: The concentration that corresponds to the velocity profiles  $u_x$  on the regime  $x \in [0, L]$  for a low and high rate, the left figure for the fusiform AAA model without bifurcation and the right figure for the fusiform AAA model with bifurcation.

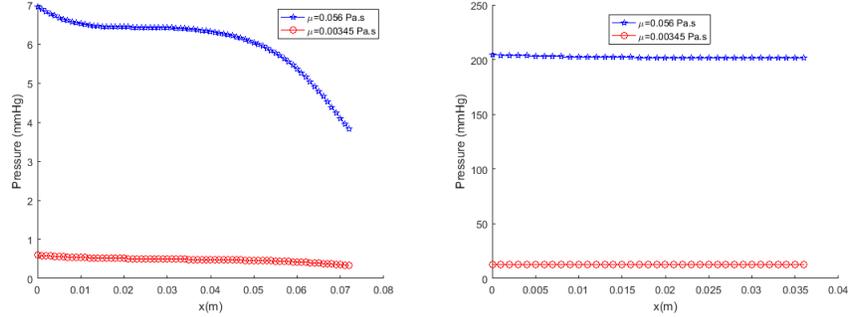


Figure 8: The pressure that corresponds to the velocity profiles  $u_x$  on the regime  $x \in [0, L]$  for a low and high rate, the left figure for the fusiform AAA model without bifurcation and the right figure for the fusiform AAA model with bifurcation.

Fig. (6) shows the velocity profiles of an AAA with and without bifurcation for a high and low shear rate. Comparing the results, we find that both models of the AAA have the same velocity profiles for a low viscosity  $\mu = 0.00345 Pa \cdot s$ . That was not the case for a low shear rate.

### 6.2.3. Discussions

When we treat the flow of blood and its viscosity, we talk about the concentration of red blood cells. It varies between  $130k/m^3$  and  $170kg/m^3$  in a healthy man and between  $110k/m^3$  and  $150kg/m^3$  in a woman. From the figure (7), we notice that the concentrations corresponding to the previous velocities for a high shear rate of two models decrease. On the other hand, for a very high viscosity, the concentration of hemoglobin throughout the hump remains stable at very important values for the two models of AAA with or without a bifurcation. This increase can produce a blockage of blood flow at this hump. On the one hand, for blood pressure Fig. (8), it's almost the same situation for both models of AAA at low viscosity. On the other hand, it is obviously important in the presence of the bifurcated geometry of the aorta. We can conclude that when the velocity is in the stage of acceleration Fig. (6), the pressure will be strongly increasing (8). This is a phenomenon that characterizes blood modeled as Bingham fluid. This is shown in Fig. (8) for pressure in case of a low shear

rate of the AAA model with bifurcation. And this shows the great influence of decay of the viscosity and thus the growth of concentration on pressure and velocity.

- [Anand et al. 2003] M. Anand, K. Rajagopal and K. R. Rajagopal; *Model Incorporating Some of the Mechanical and Biochemical Factors Underlying Clot Formation and Dissolution in Flowing Blood*, J. Theo. Medi. (5), 183–218.
- [Ataullakhanov et al. 1998] F. I. Ataullakhanov, G.T. Guria, V.I. Sarbash, R.I. Volkova; *Spatiotemporal dynamics of clotting and pattern formation in human blood*, Biochim. Biophys. Acta Gen. Subj, 1425 (3), 453–468 .
- [Brezis 1968] H. Brezis; *Equations et inéquations non linéaires dans les espaces en dualité*, Ann. Inst. Fourier, Tome 18(1), .
- [Brujan 2010] E. A. Brujan; *Cavitation in Non-Newtonian Fluids: With Biomedical and Bioengineering Applications*, Springer Science & Business Media, p. 269.
- [Chamekh and Elzaki 2018] Chamekh M, Elzaki TM. Explicit solution for some generalized fluids in laminar flow with slip boundary conditions. J Math Comput Sci ;(18)72–281.E. A. Brujan;
- [DeRubertis 2007] B. G. DeRubertis, S. M. Trocciola, E. J. Ryer, F. M. Pieracci, J. F. McKinsey, P. L. Faries, K. C. Kent; *Abdominal aortic aneurysm in women: Prevalence, risk factors, and implications for screening* , J. Vasc. Surg.46(4), 630–635
- [Finol and Amon 2003] E. A. Finol, C. H. Amon; *Flow Dynamics in Anatomical Models of Abdominal Aortic Aneurysms: Computational Analysis of Pulsatile Flow*, Acta Científica Venezolana, 54, 43–49.
- [Marrero et al. 2014] F.J.H. Gijssen, E. Allanic, F.N. van de Vosse, J.D. Janssen; *The influence of the non-Newtonian properties of blood on the flow in large arteries: Unsteady flow in a 90° curved tube*, J. Biomech. **32**, 705–713.

- [Glowinski and Ciarlet 2010] R. Glowinski, J. Xu, P. G. Ciarlet ; *Numerical Methods for Non-Newtonian Fluids*, 16: Special Volume (Handbook of Numerical Analysis).
- [Hallin et al. 2001] A. Hallin, D. Bergqvist, L. Holmberg; *Literature Review of Surgical Management of Abdominal Aortic Aneurysm*, Eur. J. Vasc. Endovasc. Surg. 22(3), 197–204 .
- [Hansen et al. 2015] K. B. Hansen, A. Arzani, S. C. Shadden; *Mechanical Platelet Activation Potential in Abdominal Aortic Aneurysms*. J. Biomech. Eng. 137.
- [Janela et al. 2010] J. Janela, A. Mouraa, A. Sequeira; *A 3D non-Newtonian fluid structure interaction model for blood flow in arteries*, J. Comp. and Appl. Math. 234 , 2783–2791.
- [Johnstona et al. 2004] B. M. Johnstona, P. R. Johnstona, S. Corneyb, D. Kilpatrickb; *Non-Newtonian blood flow in human right coronary arteries: steady state simulations*, J. Biomech. **37**, 709–720.
- [Khanafar et al. 2006 ] K. M. Khanafar, P. Gadhoke, R. Berguer, J. L. Bull; *Modeling pulsatile flow in aortic aneurysms: Effect of non-Newtonian properties of blood*, Biorheology. 43, 661–679.
- [Kato 1993] Y. Kato; *Variational inequalities of Bingham type in three dimensions*, Nagoya Mathematical Journal, Cambridge University Press . **(129)** , 53-95.
- [Kim 1987] J. U. Kim; *On the initial-boundary value problem for a Bingham fluid in a three-dimensional domain*. Transactions of the American Mathematical Society. 304(2),751–770.
- [Kim 2002] S. Kim; *A Study of Non-Newtonian Viscosity and Yield Stress of Blood in a Scanning Capillary-Tube Rheometer*. A Thesis. Drexel University .

- [Lions 1969] J. L. Lions ; *Quelques mthodes de rsolution des problmes Aux limites non linaires*. Dunod, Paris.
- [Marieb 1993] E. N. Marieb ; *Anatomie et physiologie humaines* . De Boeck Universit.
- [Marrero et al. 2014] V. L. Marrero, J. A. Tichy, O. Sahni, K. E. Jansen; *Numerical study of purely viscous non-Newtonian flow in an abdominal aortic aneurysm*, J. Biomech. Eng. 36(10).
- [Merouani and Messelmi 2015] A. Merouani, F. Messelmi; *Existence of weak solutions for the quasi-state flow of blood and and mathematical coagulation modeling*, Nonlinear Functional Analysis and Applications. 20(3)393–418.
- [Messelmi 2014] F . Messelmi ; *Diffusion-reaction problem for the Bingham fluid with Lipschitz Source*, Int. J. Adv. Appl. Math. and Mech. 1(3), 37 – 55 .
- [ Messelmi 2011] F. Messelmi; *The existence of weak solutions for the 3D-steady-state flow of blood in arteries*, Fasc. Matematica. Tom XVIII, 221-240.
- [Mofidi 2006] R. Mofidi, V. J. Goldie, J. Kelman, A. R. W. Dawson, J. A. Murie, R. T. A. Chalmers; *Influence of sex on expansion rate of abdominal aortic aneurysms*. Br. J. Surg. 94 , 310–314.
- [Papanastasiou 1987] T.C. Papanastasiou; *Flows of materials with yield*, J. Rheol. (31), 385–404 .
- [Paramasivam et al. 2010] V. Paramasivam, N. Filipovic, K. Muthusamy, M. R. Abdul Kadir; *Finite Element Modeling for Solving The Pulsatile Flow in a Fusiform Abdominal Aortic Aneurysm*, Biomed. Int. 1(2), 50–61.
- [Picart et al. 1998] C. Picart, J. M. Piau, H. Galliard, P. Carpentier; *Blood low shear rate rheometry: influence of fibrinogen level and hematocrit on slip and migrational effects*, Biorheology. (35)335–353.

- [Robertson et al. 2008] A. M. Robertson, R. Rannacher, G. P. Galdi, S. Turek; *Hemodynamical Flows. Modeling, Analysis and Simulation*, in: Oberwolfach Seminars. 37, Birkhauser , p. 501.
- [Robertson et al. 2009] A.M. Robertson, A. Sequeira, R.G. Owens; *Rheological models for blood*, in: A. Quarteroni, L. Formaggia, A. Veneziani (Eds.), Cardiovascular Mathematics. Modeling and Simulation of the Cardiovascular System, in: MS & A. Springer-Verlag. 1.
- [Simon 1987] J. Simon; *Compact sets in the space  $L^p(0, T, B)$* , Ann. Mt. Pura Appl. (146) 64–96.
- [Soto et al. 2010] H. P. Soto, M. L. Martins-Costa, C. Fonseca, S. Frey; *A numerical investiion of inertia flows of Bingham-Papanastasiou fluids by an extra stress-pressure-velocity galerkin least-squares method*, J. Braz. Sgatoc. Mech. Sci. & Eng. **32** no.spe Rio de Janeiro.
- [Toungara 2011] M. Toungara; *Contribution la prdiction de la rupture des Anvrismes de l'Aorte Abdominale (AAA)*. Life Sciences. Thse de doctorat, Universi de Grenoble.