# 1D generalized time dependent non Newtonian blood flow model

Arthur R. Ghigo, Pierre-Yves Lagrée, Jose-Maria Fullana

# 1D generalized time dependent non Newtonian blood flow model

**Arthur R. Ghigo**
Sorbonne Universités
UPMC Univ Paris 06
CNRS UMR 7190
Institut Jean le Rond ∂'Alembert
F-75005 Paris, France

**Pierre-Yves Lagrée**
Sorbonne Universités
UPMC Univ Paris 06
CNRS UMR 7190
Institut Jean le Rond ∂'Alembert
F-75005 Paris, France

**Jose-Maria Fullana**
Sorbonne Universités
UPMC Univ Paris 06
CNRS UMR 7190
Institut Jean le Rond ∂'Alembert
F-75005 Paris, France

**ABSTRACT**

Alterations in whole blood viscosity due to hemodialysis, pathologies or a low stress hemodynamic blood flow impact the viscosity generating local changes in blood pressure, blood flow rate, vessel cross sectional area, and giving unexpected global variations over the circulatory network. Despite this fact all 1D models use Newtonian viscosity rheological laws inside.

We present a 1D generalized time dependent non Newtonian model of viscosity and we compare the numerical predictions to experimental data of blood rheology available into the literature. We explore two well documented shear stress protocols and we show that the proposed 1D generalized non Newtonian model computed over an arterial segment compares accurately, qualitative as well as quantitatively, the time dependence of the shear stress over the rheological blood data.

We hope that 1D generalized blood non Newtonian flow model inside of large numerical 1D models will be useful to help even more the understanding of the global blood dynamics in micro and macro-circulatory networks.

## 1 Introduction

One dimensional flow models is an useful option for computing blood circulation in complex networks either for micro or macrocirculation. One of the main reasons is their capability to furnish good predictions in terms of blood pressure and blood flow rate being costless in terms of time computing compared to the complexity and time consuming of 3D approaches. We note that both the 1D and the 3D approaches are both derived from the same mass and momentum conservation principles.

Although we know that alterations in blood viscosity impact the whole blood circulation, in the literature until now all proposed 1D hemodynamic models for studying blood flow circulation are Newtonian: the viscosity is then considered constant. The Newtonian hypothesis is very often well supported

since in either largest arteries or small capillaries shear stresses are high in most of clinical situations. High shear stress implies a constant viscosity.

Nevertheless in a lot of current hemodynamic scenarii as stopping-starting blood flows, presence of geometrical artifacts or singularities like valves in veins, blood pathologies, aneurysms, it is admitted that the blood flow reaches a low shear stress behavior zone. In a low stress configuration, the viscosity will increase considerably generating consequently localized changes in blood pressure, blood flow rate, vessel cross sectional area, and we know that local changes could cause unexpected global variations.

Blood is a non Newtonian fluid exhibiting a pseudo-plastic behavior (a decrease in viscosity with an increase on the rate of shear) with a memory effect (called thyxotropy). Blood viscosity changes the with the hydrodynamical state and with the history of the blood flow. In a circulatory network as a consequence of the pulsed nature of blood flow and the geometrical topology of the circulatory network, the system is in a permanent non-equilibrium state. The passage from a state of high viscosity to another of low viscosity is done therefore continuously, and when the blood is on a lower shear situation a aggregation process develops naturally in red cells. The Red Blood Cell (RBC) aggregation is the capability of red cells to build stick arrangements called "rouleux" which affects both, the pseudo-plastic and the thixotropic characteristic of blood. The RBC aggregation is a natural phenomenon caused by biological factors and influenced by blood hydrodynamical interplay. Some pathologies change the RBC aggregation in different clinical circumstances having as a straightforward consequence diseases as hematological disorders, cardiovascular illness or being the edge of atherosclerosis risk factors. It is of clinical importance to determine in real time the RBC aggregation and to understand its global effect over a circulatory network.

We present an implementation in a 1D flow model of a generalized time dependent non Newtonian blood rheology from Rosemblatt [1]. The rheological model is based on a statistical mechanics theory relating macroscopic stresses with the micro-mechanics of the blood. The kinetic equation for the aggregation state is a particular case of the structural model of more general non Newtonian fluids. (See for instance [2,3]). This kinetic equation is moreover closely related to the rheological model proposed by Cross [2] in 1965 for pseudo-plastic material.

In Section 2 we present the 1D blood flow model, the non Newtonian approach of the stress tensors as well as their integration into the 1D blood flow model. Section 3 shows several numerical comparisons against published experimental data of blood rheology.

## 2 Flow model

The 1D blood model derives from the integration over a cross-sectional area $A$ of the Navier-Stokes equations (mass and momentum conservation) in cylindrical coordinates. Doing this integration or averaging process we lost indeed information about velocities profiles but the system of equations becomes numerically accessible. For the averaged variables of the problem (the cross-sectional area $A$ and the flow rate $Q$, and pressure $P$) we have the following 1D space-time evolution equations

$$\frac{\partial A}{\partial t} + \frac{\partial Q}{\partial x} = 0, \tag{1a}$$

$$\frac{\partial Q}{\partial t} + \frac{\partial}{\partial x}\left(\alpha \frac{Q^2}{A}\right) + \frac{A}{\rho}\frac{\partial P}{\partial x} = f_\mu, \tag{1b}$$

where $\rho$ is the fluid density and $\alpha$ the momentum shape coefficient depending of the blood velocity profile (sets to one from here). The term $f_\mu$ correspond to a model of viscous losses discuss later. All variables depend therefore on time and on the spatial coordinate $x$. The closure relation connecting the

cross-sectional area $A$ and the averaged pressure $P$ is:

$$P = P_{ext} + \beta(\sqrt{A} - \sqrt{A_0}) + \nu_s \frac{\partial A}{\partial t}, \tag{2}$$

where $P_{ext}$ is the external pressure, and the coefficients stiffness $\beta$ and viscosity $\nu_s$ define the arterial wall model. This is a Kelvin viscoelastic wall model and more details of the complete 1D blood model and the numerical integration are in reference [Wang 2011]. In terms of modelling is a crucial point since it links the wall mechanics to the blood flow.

### 2.1 Stress model: newtonian

When we derive the 1D model from the Navier-Stokes equation, the viscosity coefficient $\mu$ appears into the friction term $f_\mu$ in the r.h.s. of equation (1b) as a consequence of the integration of the surface forces over a control volume $V$, mathematically speaking $\int_V \nabla\sigma dS$ where $\sigma$ is the total stress tensor. Separating the isotropic part (the pressure) from the total stress tensor we have to evaluate the viscous tensor $\tau$ over the control surface $S$ bordering $V$, which results in the surface integral $\int_S \tau \mathbf{n} dS$ by application of the divergence theorem. We note that when the viscosity $\mu$ is constant we can write the viscous tensor using the shear rate $\dot{\gamma}$ to get directly $\tau = \mu\dot{\gamma}$.

We discuss briefly the setup of the friction term $f_\mu = \int_S \tau \mathbf{n} dS$ in our model. Starting from the cylindrical coordinates under the 1D hypothesis the only non null coefficient of the viscous tensor is this concerning the $x$ force into the $r$ direction, $\tau_{rx}$ then the above integral gives

$$f_\mu = 2\pi R \tau_{rx}. \tag{3}$$

The term $\tau_{rx} = 2\mu\frac{1}{2}\left(\frac{\partial u_x}{\partial r} + \frac{\partial u_r}{\partial z}\right)$ becomes explicitly $\sim 2\mu\frac{1}{2}\frac{\partial u_x}{\partial r}$ in the framework of long wavelength approach since the radial component of the velocity $u_r$ scales as $u_x\varepsilon$. The small parameter $\varepsilon$ used into the asymptotic study is the ratio between the radial and the longitudinal lengths of the problem and comes directly form the mass conservation equation. Using the averaged quantities of the problem we define the averaged shear rate as follow: $\frac{\partial u_x}{\partial r} \sim \frac{U}{R} \sim \frac{Q}{A^{3/2}} = \left.\frac{\mathrm{d}\phi}{\mathrm{d}r}\right|_{r=R} \dot{\gamma}_A$ where $\left.\frac{\mathrm{d}\phi}{\mathrm{d}r}\right|_{r=R}$ is the shape factor that takes into account the velocity profile. As an example, for a Couette flow is $-1$ and for a Poiseuille flow equal to $-4$. In large arteries, it is common to use $-11$. We have now the viscous friction term

$$f_\mu = 2\pi A^{1/2} \left.\frac{\mathrm{d}\phi}{\mathrm{d}r}\right|_{r=R} \tau_A, \tag{4}$$

as function of $\tau_A = \mu(\dot{\gamma}_A)\dot{\gamma}_A$, the averaged viscous stress. We write explicitly that the viscosity can depend on the averaged shear rate, $\dot{\gamma}_A$. This shape factor doesn't matter in the rest of our work and it will be set to -1 in numerical computations. However it should be modified for specific configurations. Note that if the viscosity is constant we retrieve the expected result $f_\mu = -2\pi\mu\frac{Q}{A}$.

### 2.2 Stress model: Non newtonian

The generalized time dependent non-Newtonian model proposed by Rosemblatt [1] begins by an equation for the structure dependent stress $\tau_{st}$

$$\lambda_{st} \frac{D\overset{\nabla}{\tau}_{st}}{Dt} + \tau_{st} = 2\mu_{st} D \tag{5}$$

where $\frac{\overset{\nabla}{D\tau_{st}}}{Dt} = \frac{D\tau_{st}}{Dt} - (\nabla \mathbf{u})^T \tau_{st} - \tau_{st}(\nabla \mathbf{u})$ stands by the upper convective derivative, $\frac{Df}{Dt}$ by the convective derivative, $D$ is the strain rate tensor and $\lambda_{st}$ a time constant for the structure dependent state (see [4] for details). We note that the shear $\tau_{st}$ depends on the structural state of the viscosity $\mu_{st}$ therefore the blood hydrodynamics and it's time history. It is important to remark that equation (5) spans in different known models : if $\lambda_{st}$ and $\mu_{st}$ are constants $(\lambda_0, \mu_0)$ we retrieve the upper convected Maxwell model, moreover if the Wiessenberg number (dimensionless number comparing the viscous forces to the elastic forces) is small we have a linear Maxwell model

$$\lambda_0 \frac{\partial \tau}{\partial t} + \tau = \mu_0 \dot{\gamma}.$$

If now the characteristic time $\lambda_0$ is small and $\mu = \mu(\dot{\gamma})$ depends on the shear rate we have a generalized non Newtonian model (e.g. Power flow, Casson, Cross [5]), and finally with $\mu$ constant we retrieve a Newtonian model ($\tau = \mu \dot{\gamma}$ ).

In a time depend approach the characteristic time $\lambda_{st}$ is a function of a structure function $f$. In the framework of blood hemodynamic the structure function $f$ is a continous parameter giving the measure of the degree of aggregation of the RBC. For instance $f = 0$ stands for all RBC disaggregated and to the contrary $f = 1$ defines a fully aggregation state. The kinetic equation for $f$ is

$$\frac{Df}{Dt} = k(1-f) - \alpha |\dot{\gamma}| f \tag{6}$$

where the l.h.s. is the convective transport, and $k$, $\alpha$ are the model parameter we will discuss later. The characteristic time of the structure equation relates to $f$ as follow, $\lambda_{st} = \frac{f}{k}$. The shear rate is $\dot{\gamma} = (2D : D)^{1/2}$ and the total viscous stress depending of the structure $\tau_{st}$ is

$$\tau = \tau_{st} + 2\mu_\infty D$$

where $\mu_\infty$ is the viscosity at the upper shear rate limit. The structural viscosity $\mu_{st}$ for equation (5) is then $(\mu_0 - \mu_\infty) f$.

### 2.3 Estimating the Stress model parameters

The model parameters $\alpha$, $k$, $\mu_0$ and $\mu_\infty$ should be evaluated using experimental data from classical viscosimeter. Using a simple plan shear viscometer with two plates separated apart of a distance $h$ in the plane $x, y$ we impose to the blood flow a constant velocity $u$ in the $x$ direction. The constant shear rate is then $\dot{\gamma} = \frac{u}{h}$ and the only non zero component of $\tau$ is $\tau_{xy}$. In a stationary configuration the equation (5) reduces to:

$$\tau_{xy_{st}} = \mu_{st} \dot{\gamma}$$

and the total stress tensor

$$\tau = \tau_{xy_{st}} + \mu_\infty \dot{\gamma}.$$

We are looking for a time depending viscosity $\mu$ then:

$$\mu = (\mu_0 - \mu_\infty) f + \mu_\infty.$$

The equilibrium value of $f$ comes from equation (6), $\frac{1}{f} = 1 + \frac{k}{\alpha}|\dot{\gamma}|$ therefore we find an expression for the viscosity

$$\mu = \frac{(\mu_0 - \mu_\infty)}{1 + \frac{k}{\alpha}|\dot{\gamma}|} + \mu_\infty \tag{7}$$

the values of $\mu_0$, $\mu_\infty$ and $\frac{k}{\alpha}$ are evaluated from an experimental blood rheological curve. We note that result was already found by Cross [2] for pseudo-plastic flows

$$\mu = \frac{(\mu_0 - \mu_\infty)}{1 + a\dot{\gamma}^{2/3}} + \mu_\infty \tag{8}$$

One of Cross's arguments about the power coefficient for $\dot{\gamma}$ was that the tensor $D$ should be an even function and that $2/3$ is the simplest admissible fraction fulfilling this condition, in our formulation $|\dot{\gamma}|$ does it.

In practice to discriminate $k$ from $\alpha$ we have to study the aggregation process without blood flow, thus the parameter $k$ is the solution of $\frac{\partial f}{\partial t} = k(1-f)$. Using blood experimental data from McMillan [6], references [1] and [7] found $\mu_0 = 0.12\ Pa\ s$, $\mu_\infty = 0.004\ Pa\ s$, $\alpha = 1.2$ and $k = 0.25\ s^{-1}$. Viscosity values ($\mu_0$ and $\mu_\infty$) are consistent with healthy normal values of blood viscosity. We use these values as known parameters in the numerical model.

### 2.4 Complete 1D model solved

For completeness we write the complete system of the 1D model equations, the governing fluid equations for mass and momentum conservation

$$\frac{\partial A}{\partial t} + \frac{\partial Q}{\partial x} = 0 \tag{9}$$

$$\frac{\partial Q}{\partial t} + \frac{\partial}{\partial x}\left(\frac{Q^2}{A}\right) + \frac{A}{\rho}\frac{\partial P}{\partial x} = -2\pi A^{1/2}\tau \tag{10}$$

together with the pressure-cross sectional area relation $P = P_{ext} + \beta(\sqrt{A} - \sqrt{A_0}) + \nu_s \frac{\partial A}{\partial t}$.

Using the averaged shear rate $\dot{\gamma}_A$ the time depending structural equation is

$$\lambda_{st}\frac{\partial \tau_{st}}{\partial t} + \tau_{st} = \mu_{st}\dot{\gamma}_A \tag{11}$$

where the convective part is now into the following kinetic equation

$$\frac{\partial f}{\partial t} + \frac{Q}{A}\frac{\partial f}{\partial x} = k(1-f) - \alpha|\dot{\gamma}_A|f. \tag{12}$$

The parameter $f$ is convected with averaged velocity $\frac{Q}{A}$ so does the structural parameter $\tau_{st}$. The last relations to close the system are

$$\mu_{st} = (\mu_0 - \mu_\infty)f$$
$$\lambda_{st} = \frac{f}{k}$$
$$\tau = \tau_{st} + \mu_\infty\dot{\gamma}_A$$

where the total stress $\tau$ is the source term of equation (10).

Equations (9) and (10) are of hyperbolic nature and we use a finite volume approach to solve them [Wang 2011, Wang 2013]. For the rest of the numerical problem we apply a semi implicit method to update $\lambda_{st}$ in Equation (11) and a 2nd order upright scheme for the convective term of equation (12).

## 3 Results

We propose to compare numerical results of the non Newtonian 1D model to blood experimental data available in the literature.

We note here that in a classical viscometer experimental device the shear rate is controlled because the device's walls are rigid, but in the flexible tube configuration we are presenting we can only to control the flow rate input, and therefore the shear rate inside of the tube will be never exactly the wished. Over these simulations we set the neutral cross sectional area $A$ to 1 $cm^2$, and a constant flow rate $Q$ at the input side of the elastic model in order to impose as close as possible the experimental shear rates. The length $L$ of the elastic segment in all numerical simulations is fixed and equal to $20cm$. To focus on the non Newtonian rheology in numerical simulations we set the viscoelastic coefficient $\nu_s$ to 0 in order to eliminate the viscoelastic effects coming from the wall dynamics. We have already shown that is quite complicated to separate both viscoelastic contributions from fluid and wall [8, 9].

### 3.1 One shear step

Over a series of experiences Bureau and co-workers [10, 11] and later Quemada et al. [3] have performed rheological experimental studies on shearing blood. In the light of published results two different and characteristic behaviors appear at low and high shear rates. For a experimental protocol at low constant shear rate (step of 0.05 $s^{-1}$ for 20 sec. before stopping) the experimental data have shown that the blood behaves as a viscoelastic material : for each experience the measured shear stress has been continuously rising and that is a signature of an absence of thixotropy. The main conclusion was that the shear stress imposed was too low to dissociate the blood structure. The stress-shear diagramme shows a Maxwell like behavior with a growing in $(1-e^{(t/\lambda_0)})$ and a relaxation dynamics in $e^{(-t/\lambda_0)}$ ($\lambda_0$ is a characteristic time). The same experimental configuration is simulated by the non Newtonian 1D blood model where we impose a flow rate of $Q = 0.05$ giving a average shear rate of 0.05. The corresponding time depend numerical solution of the shear stress is presented in Figure 1.

Experimental results for the stationary shear stres are 0.0578 (without bar error) for an hematocrit of 0.45 in [3] and $0.043 \pm 0.015$ for an hematocrit of 0.435 in [10]. Numerical predictions compare very well, quantitative and qualitatively, the experimental results considering that in our model the parameters were fitted using different rheological blood data ($\mu_0, \mu_\infty, \alpha, k$ from reference [1]). [to reviewer : Annexe A presents the figures of all experimental results, we're waiting for the copyrights]

For a high shear rate of 1 $s^{-1}$ experimental data shows a clear effect of the thixotropy : the maximum shear stress $\tau_{max}$ is reached very rapidly at $t_{max}$ and its value is greater that its stationary value $\tau_{stat}$. The Figure 2 shows the corresponding numerical results captured for the non Newtonian 1D model, the numerical findings for the maximum and stationary shear stresses as well as the time delay to reach the maximum shear stresses are in total accord with the experimental published data (ref. [12] $\tau_{max} = 0.326 \pm 0.015$, $\tau_{stat} = 0.215 \pm 0.011$ and $t_{max} = 1.2s$. and ref. [3] $\tau_{stat} = 0.257$).

We recover also the experimental observation that the relaxation process is faster in the high shear configuration. Besides the numerical predictions show the relaxation time for the case of shear rate of 0.05 $1/s$ is twice than for a shear rate of 1 $s^{-1}$ as expected from experimental published data [3, 12].

The state of structuration or aggregation of the blood is difficult to obtain in experimental setups, conversely it appears naturally into the non Newtonian 1D model. Figure 3 presents the structure function $f$ as function of time for the two simulated experiences (Figures 1 and 2). For low shear rate

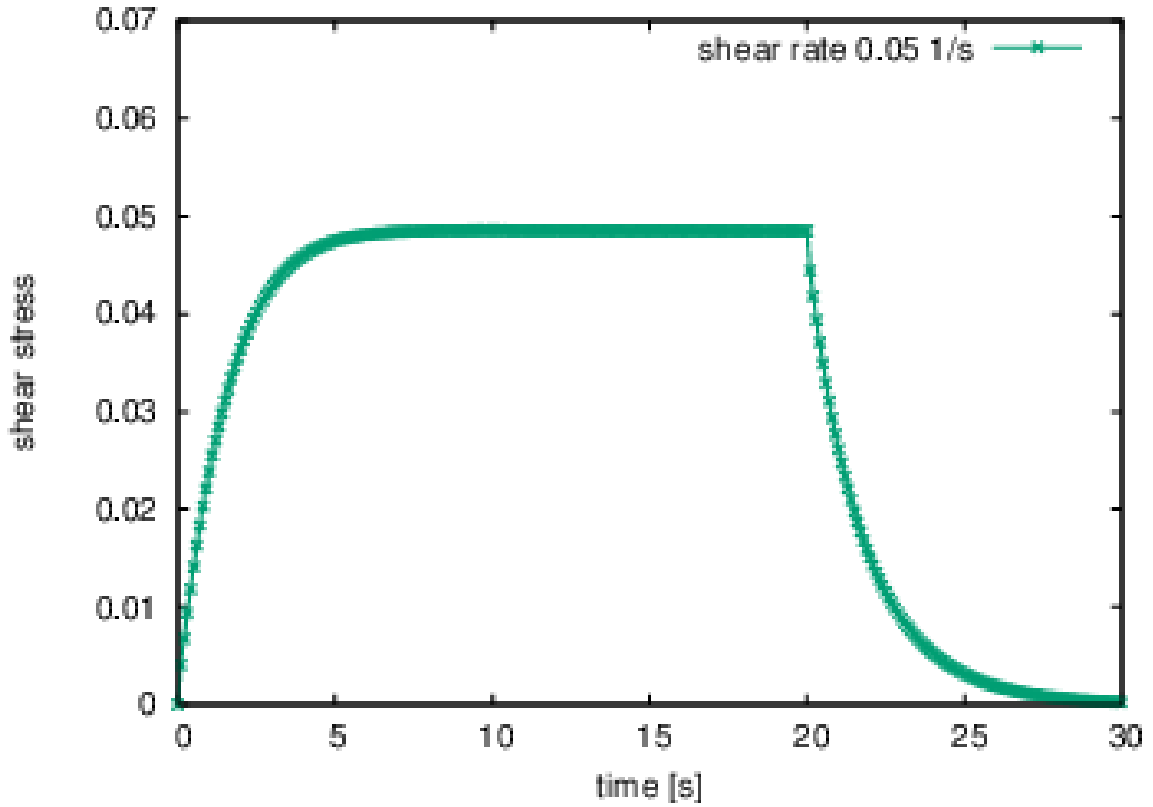


Fig. 1. Low Shear simulation : one step shear-rate of $0.05\ s^{-1}$ is imposed and then stopped at time 20 s.

(Fig 3 upper curve) the function $f$ is everywhere above 0.8 allowing to solve the equation (11) with a quasi constant value of the characteristic time. Conversely the fast decreasing of $f$ for high shear stress illustrates the thyxotropic behavior observed in the early times in Figure 2.

The relaxation dynamic of the shear stress after stopping the input flow at time 20 sec. (equivalent to the stopping shear rate process in the experimental setup) is better understood when we analyze the behavior of the function $f$ in Figure 3. It is clear that the shear stress dynamic and consequently the instantaneous blood viscosity depends on the state of aggregation of the fluid.

We can solve analytically the equations for a constant shear rate starting from a fluid at rest for which the structure function $f = 1$. To simplify the analysis, we assume that the advection term $\frac{Q}{A}\frac{\partial f}{\partial x}$ can be neglected.

The kinematic equation for the structure function writes:

$$\frac{\partial f}{\partial t} = -\left(k + \alpha|\dot{\gamma}|\right) f + k. \tag{13}$$

We introduce the notation $\tau_f = \frac{1}{k+\alpha|\dot{\gamma}|}$, and the solution of the previous equation is

$$f = k\tau_f + \left(1 - k\tau_f\right) \exp\left[-\frac{t}{\tau_f}\right] \tag{14}$$

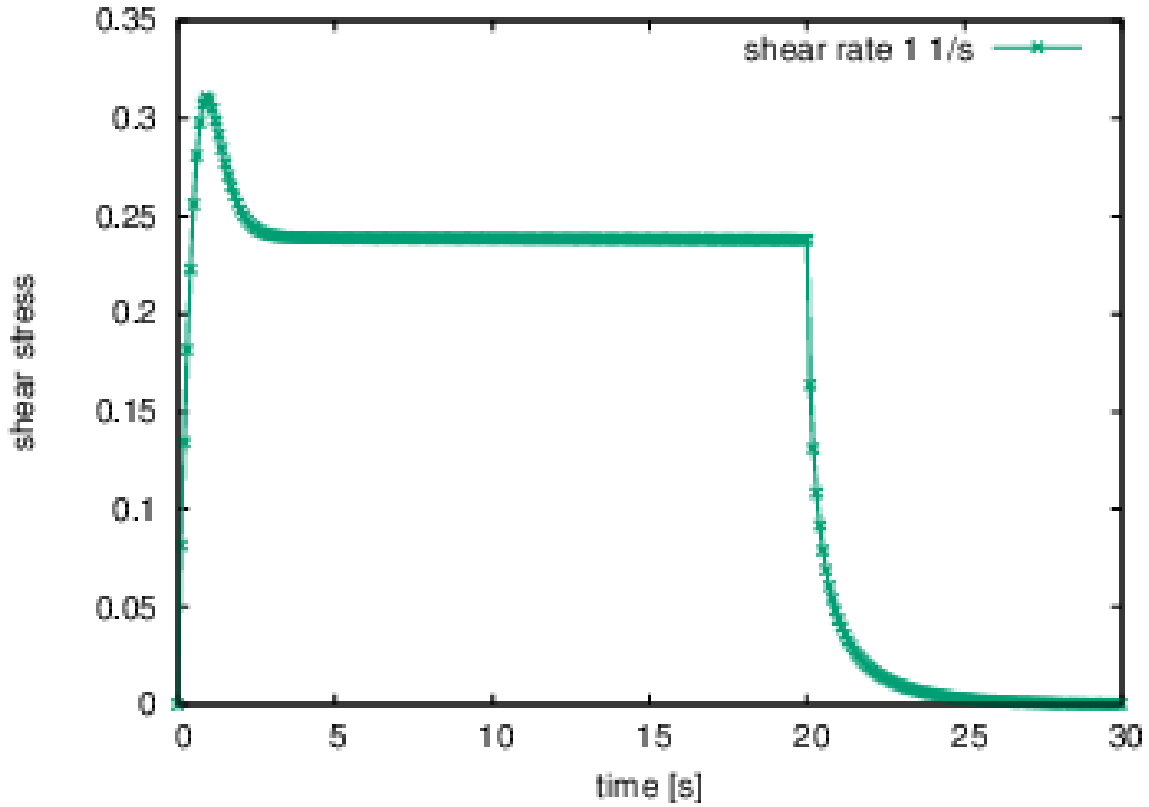


Fig. 2. High Shear simulation : one step shear-rate of $1\ s^{-1}$ is imposed and stopped at time 20 s.

Injecting this expression for $f$ in the equation (11) we have:

$$\tau_{st}(t) = \frac{(\mu_0 - \mu_\infty)\dot{\gamma}\left[k\left(1-k\tau_f\right)t + \left(k\tau_f\right)^2\left(\exp\left[\frac{t}{\tau_f}\right]-1\right)\right]}{1+k\tau_f\left(\exp\left[\frac{t}{\tau_f}\right]-1\right)} \tag{15}$$

When $t \to 0$, we find the following asymptotic values for $f$ and $\tau_{st}$ :

$$\begin{cases} f = 1 - \dfrac{1-k\tau_f}{\tau_f}t = 1 - \alpha|\dot{\gamma}|t \\ \tau_{st} = k(\mu_0 - \mu_\infty)\dot{\gamma}t. \end{cases} \tag{16}$$

When $t \to \infty$, we find the following asymptotic values for $f$ and $\tau_{st}$ :

$$\begin{cases} f_\infty = k\tau_f = \dfrac{1}{1+\frac{\alpha|\dot{\gamma}|}{k}} \\ \tau_{st,\infty} = k\tau_f(\mu_0 - \mu_\infty)\dot{\gamma} = (\mu_0 - \mu_\infty)\dfrac{\dot{\gamma}}{1+\frac{\alpha|\dot{\gamma}|}{k}}. \end{cases} \tag{17}$$

At small times thee are a competition between the destruction of $f$ governed by $\alpha$ and the production of $\tau_{st}$ governed by $k(\mu_0 - \mu_\infty)$. The overshoot in $\tau_{st}$ seems to be governed by the parameters $\alpha$ and $\dot{\gamma}$ and it seems to be independent of $k$. Furthermore, the overshoot happens when $f$ reaches relatively its constant value $f_\infty$.

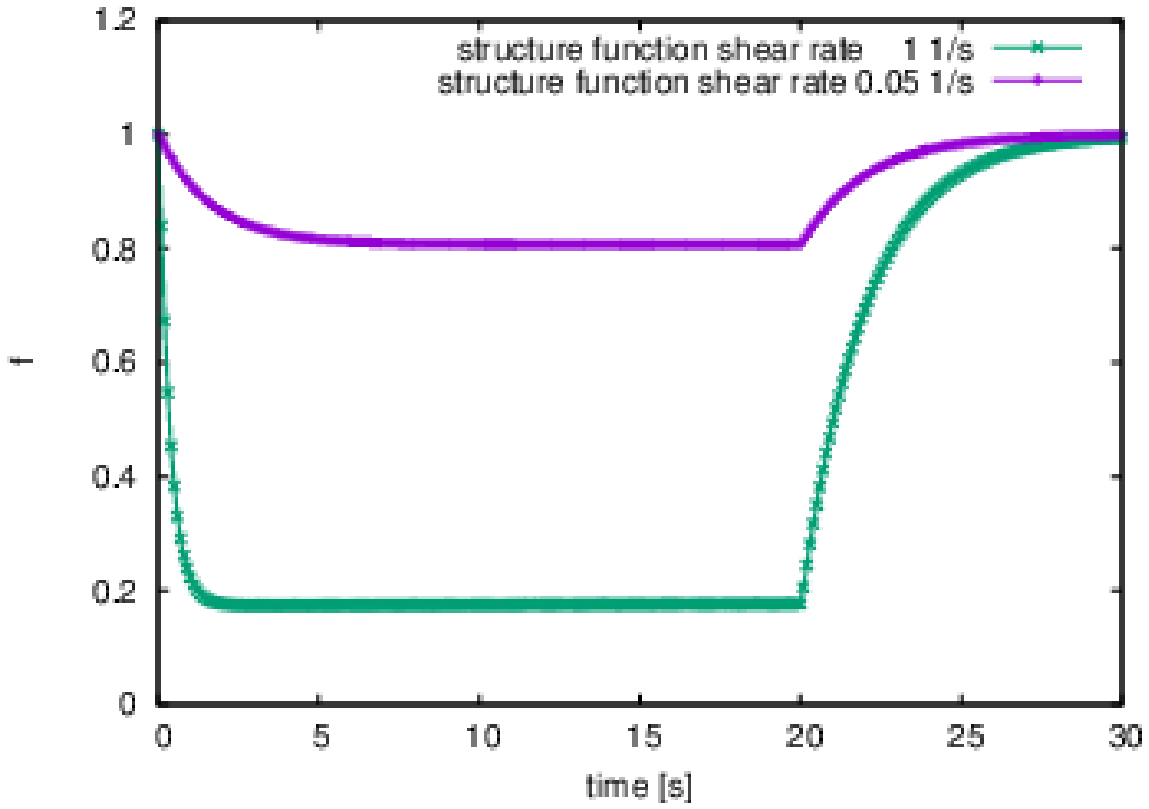


Fig. 3. Structure function $f$ for two shear rates (upper curve) $1\ 1/s$ (bottom curve) $0.05\ 1/s$

### 3.2 Multiple shear steps

Experimental data from McMillan [6] correspond to a study of the shear stress response to different start up shear flows setups. The shear stress experiences were done by considering two periods of constant shear stress separate appart by a controlled time without shearing. Experimental stress measurements covered the time stress evolution, the stress relaxation and the influence of the shear history.

For an experimental setup the protocol was done by imposing two constant shear stress of $\dot{\gamma} = 8\ 1/s$ for a fixed time of 2.5 *sec* separated by a time delay of 1.5 *sec* without shear stress. Experimental data from [6] have shown a thixotropic transient overshot in the measured shear stress together with a second peak always smaller than the first one. Figures 4 show the numerical results of the non Newtonian 1D model with the same experimental parameters (linear-linear and linear-log scales). We observe exactly the same behavior as experimental data. We note integrating directly model equations (5) for imposed shear stress but without blood flow dynamics, close numerical results were found [3, 7].

In ref. [6] the authors decreased the non shearing time delay from 1.5 to 0.5 *sec*. They observed that the second shear stress overshoot peak decreases in a positive correlation with the time delay. The Figure 5 presents the numerical results which are in total agreement with experimental data.

## 4 Conclusion

We have presented 1D generalized blood non Newtonian flow model. The blood model is based on a classical 1D approach for the conservation movement equations for the blood flow rate $Q$ and the cross sectional area $A$ but including now a time dependent blood viscosity. For that we have proposed a 1D version of a generalized time dependent non Newtonian blood rheology from Rosemblatt [1]. To take into account the time dependence in the blood dynamics we used a kinetic equation is a particular case of the structural model for pseudo-plastic fluids [2] and blood [3]. We note that a large game of

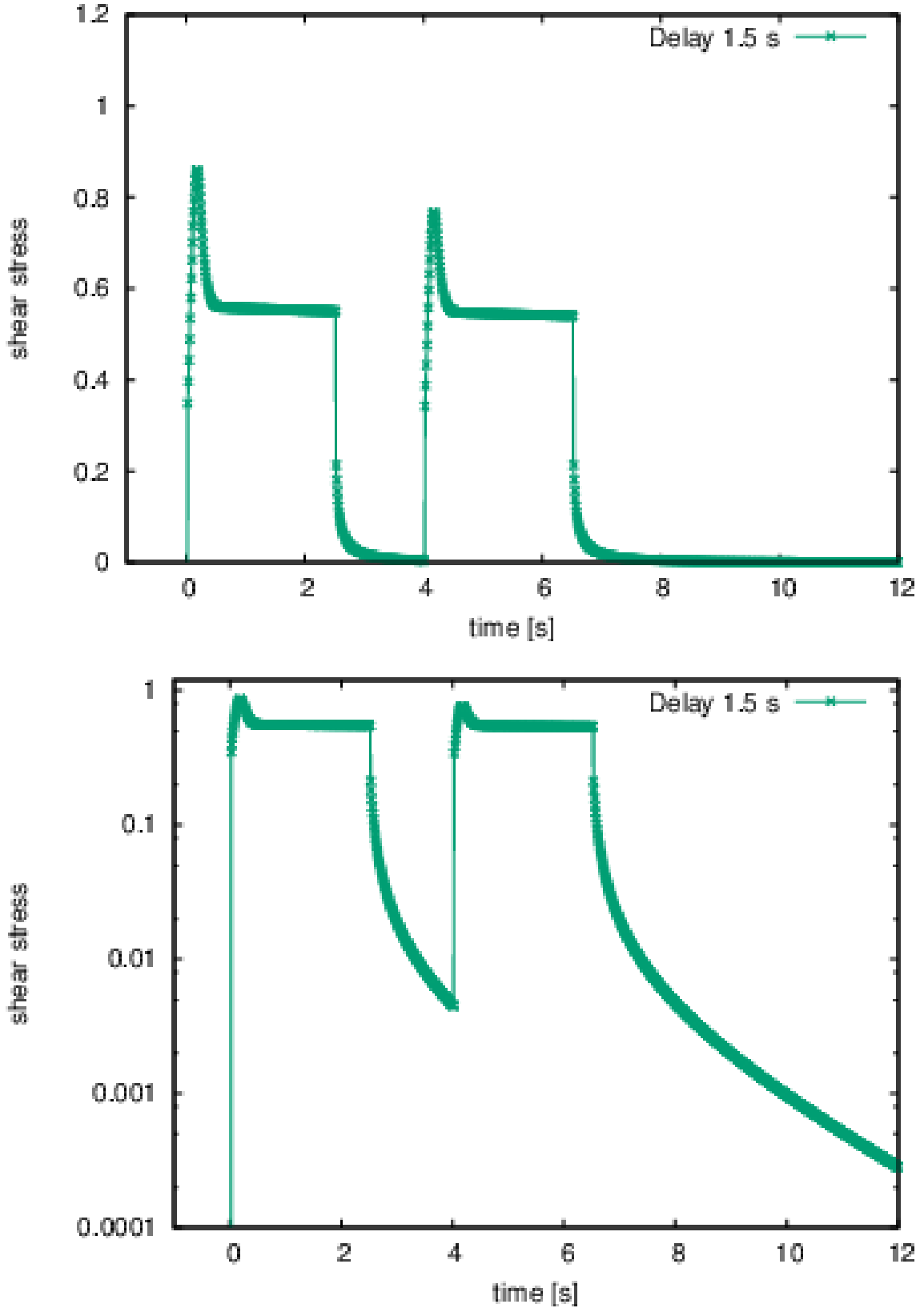


Fig. 4. Shear stress response to two shear rate steps separated of $2.5\ sec$ by a non shearing period of $1.5\ sec$. Numerical predictions in linear-linear (top) and linear-loo (bottom) sacles of the experimental data of McMillan [6].

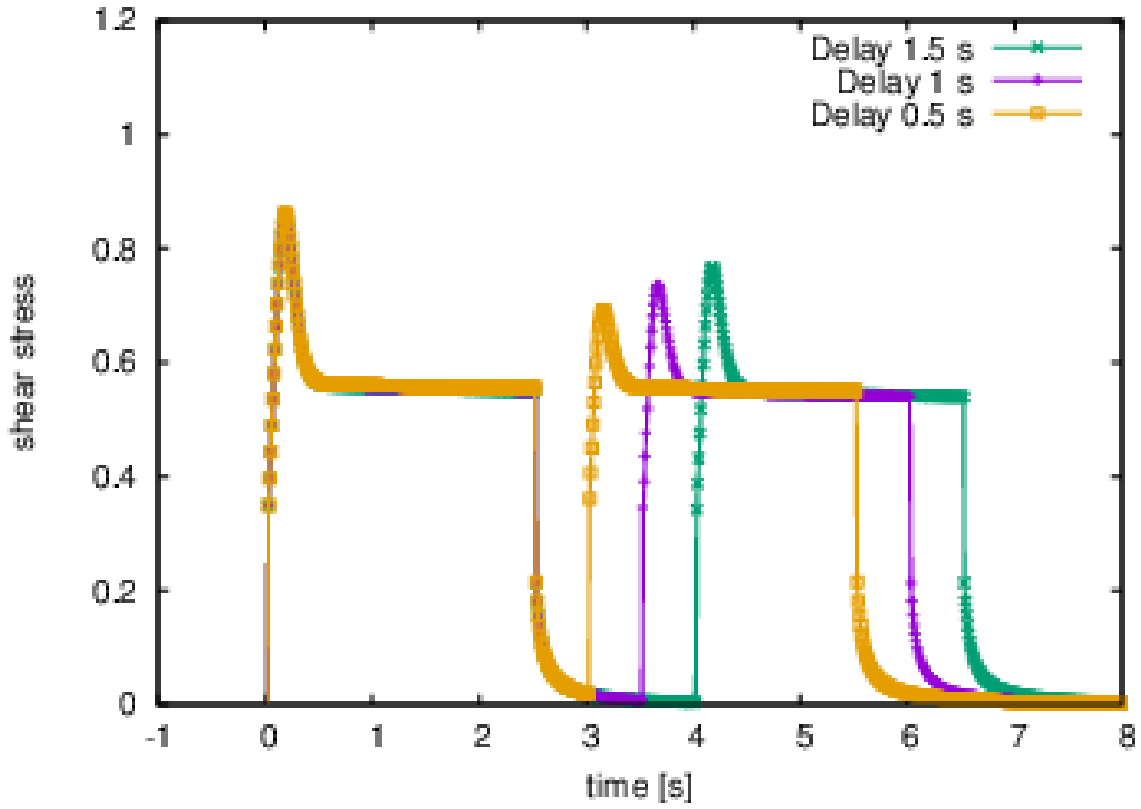


Fig. 5. Shear stress : two shearing shear rate steps separated of $2.5\ sec$ with time delay in a non shearing period from $1.5s$ to $0.5$.

rheological models are included in this approach.

We have confronted the numerical predictions of our blood model to experimental data available in the literature, and we have found that our 1D blood model computed over a arterial segment compares accurately the rheological blood data.

We hope that 1D generalized blood non Newtonian flow model will be useful to help the understanding of the global blood dynamics in micro and macro-circulatory networks.

## References


[1] Rosenblatt, J. S. *The rheology of human blood: a structured fluid approach based on rouleau behavior*. University of California, Berkeley.

[2] Cross, M. M. "Rheology of non-newtonian fluids: a new flow equation for pseudoplastic systems". pp. 417–437.

[3] Quemada, D., and Droz, R. "Blood viscoelasticity and thixotropy from stress formation and relaxation measurements: a unified model.". pp. 635–651.

[4] Bird, R. B. "Useful non-newtonian models". pp. 13–34.

[5] Fung, Y.-C. *Biomechanics: mechanical properties of living tissues*. Springer Science & Business Media.

[6] McMillan, D. E., Strigberger, J., and Utterback, N. G. "Rapidly recovered transient flow resistance: a newly discovered property of blood". pp. H919–H926.

[7] Baaijens, J. P. W. "Analysis of transient non-newtonian flow of blood".

[8] Wanga, X.-F., Fullana, J.-M., Lagrée, P.-Y., and Armentano, R., 2013. "Effect of viscoelasticity of arterial wall on pulse wave: a comparative study on ovine". *Computer methods in biomechanics and biomedical engineering,* **16**(sup1), pp. 25–26.

[9] Ghigo, AR, W. X.-F. A. R. F. J.-M. L. P.-Y., 2017. "Linear and nonlinear viscoelastic arterial wall models: application on animals". *Journal of Biomechanical Engineering,* **139**(1).

[10] Bureau, M., Healy, J. C., Bourgoin, D., and Joly, M. "Rheological hysteresis of blood at low shear rate.". pp. 191–203.

[11] Bureau, M., Healy, J. C., Bourgoin, D., and Joly, M. "Etude rhologique en rgime transitoire de quelques chantillons de sangs humains artificiellement modifis". pp. 756–768.

[12] Bureau, M., Healy, J. C., Bourgoin, D., and Joly, M. "Etude exprimentale IN VITRO du comportement rhologique du sang en rgime transitoire faible vitesse de cisaillement". pp. 612–625.

## A Experimental data

For Editor and reviewers : these figures are for comparaison, we have not still answer for copyrights.

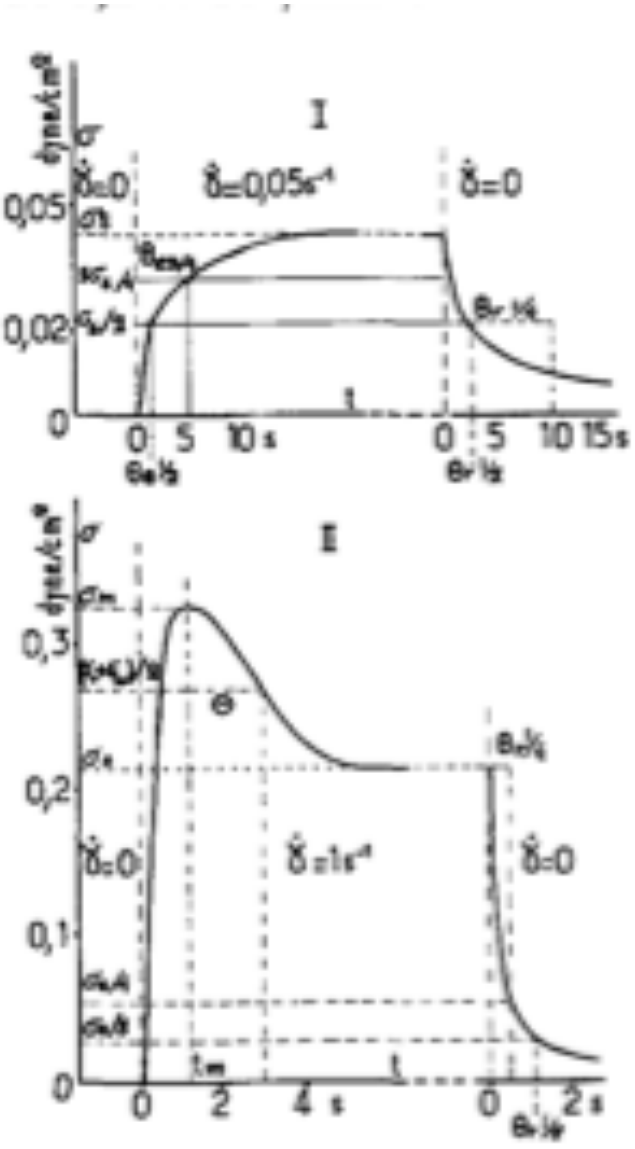


Fig. 6. Experimental data from Bureau's paper. (See Figures 1 and 2)

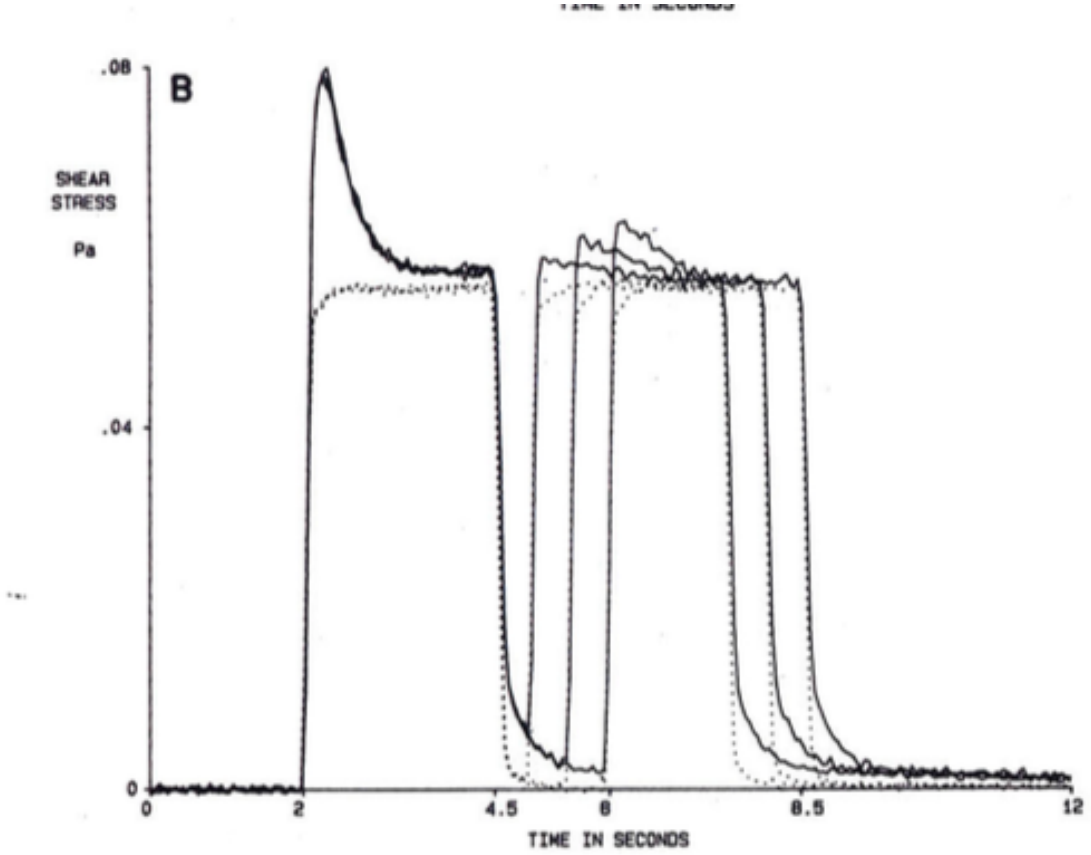


Fig. 7. Experimental data from McMillan's paper. (see Figure 5)