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BLOOD FLOW DYNAMICS THROUGH A FRACTAL MODEL

BY

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Abstract. We propose an original model for blood flow dynamics using the Theory of Scale Relativity with an arbitrary and constant fractal dimension in the fractal hydrodynamic representation. We show that the flow is directed towards the walls. This type of flow could lead, in our opinion, to a thickening effect. This type of effect leads to cholesterol deposition on the blood vessels walls and is one of the main sources of atherosclerosis.

Keywords: blood; complex fluid; non-differentiability; scale relativity.

1. Introduction

Blood flow dynamics and also the physiological and pathological changes that blood undergoes along the lenght of the whole arterial trunk are still topics of interest for physicians worldwide.

We can assimilate blood to a complex non-Newtonian fluid, having the following structural and functional entities: plasma, red blood cells, platelets, white blood cells, cholesterol and also other suspended particles (Popescu, 1999). In such a conjecture, we can state that the laws of fractal physics can be applied to sanguine circulation. The structure of the arterial system, being a complex one, further justifies the use of fractality, due to its numerous and

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various ramifications, because they are causing areas of turbulence and also interruptions of the linear flowing, thus making the widely-used classical physics models not applicable in such a context. We can therefore consider "a morphological" multi-fractality induced by the complex structure of the whole-body blood vessels system, and also a "functional" multi-fractality induced by the blood flow "regimes" (Tesloianu, 2014). Let us note that a viscoelastic fluid's stress, as opposed to that of a Newtonian fluid, depends both on the actual manifested stress as well as on the stress applied during previous deformations of the fluid in question (Mitchel, 2009; Badii and Politi, 1997).

If we take a look at the specialized literature, we can see that standard theoretical models employed in complex fluid dynamics analysis, especially that of flow through blood vessels are ambiguous (Schwartz and Murry, 1998). The "classical" model in which blood entities move along continuous and differentiable trajectories is, in our opinion, false, because it cannot encompass all the types of dynamics induced by blood flow (the separation of blood components through turbulence regimes, blood-blood vessels interactions etc.).

Taking the above into consideration, we propose a new model in which blood structural units move on continuous but non-differentiable curves (fractal curves). Our aim is not to predict the entirety of interactions which take place in a blood-vessel system. Our model represents a simple and effective solution, because all these interactions can be substituted by employing fractality (Nottale, 1989).

One can thus be conducted to specific dynamics of a particular kind of fluid, interactions-free, for which the flow lines are continuous but non-differentiable curves.

Current theoretical models used to describe a complex fluids' dynamics are sophisticated (Thomas, 2009; Michel and Thomas, 2012). These models can be made as simple as possible if we take into consideration the fact that the interaction processes' complexity imposes various time-dependent resolution scales, and also the evolution of the patterns imposes different freedom degrees (Badii and Politi, 1997).

In this context, new and original theoretical models can be designed by supposing that the complex fluids showing chaotic behavior gain self-similarity, associated with high degrees of fluctuations at all the possible space-time scales (Mandelbrot, 1983; Michel and Thomas, 2012). In the case in which temporal scales are large when compared to the inverse of the highest Lyapunov exponent (Cristescu, 2008; Federer and Aharoner, 1990), we can replace both the deterministic trajectories with a set of potential trajectories and also the concept of definite positions with the concept of probability density.

Since complex fluids hold the universal property of fractality (non-differentiability), we find it necessary to construct a fractal physics. If we now consider the fact that non-differentiability replaces the complexity of interactions processes, we find that it is not necessary to employ the entirety of

classical quantities from standard physics. This subject was developed in the Theory of Scale Relativity (Nottale, 2011; Nottale, 1993) and in the non-standard Theory of Scale Relativity , *i.e*. the Theory of Scale Relativity with an arbitrary constant and fractal dimension (Mercheş and Agop, 2016). In this framework we can make the assumption that the movements of a complex system's structural components occur on continuous but non-differentiable curves, which leads us to affirm that all the physical phenomena which influence these dynamics are dependent not only on the space-time coordinates but also on the space-time scales resolution. As a result, the quantities used to describe the complex systems' dynamics might be considered fractal functions (Nottale, 2011; Mercheş and Agop, 2016).

In the present paper, various blood flow dynamics are analysed with the purpose of defining new mechanisms for the evolution of biophysical processes in the arterial tree, these having implications in different pathologies related to blood (occlusion of the arteries, cholesterol accumulation on blood vessels etc).

2. Materials and Methods

Let us now assume that the movements of blood's structural entities occur along continuous but non-differentiable curves. Then, the following consequences can be seen (Nottale, 2011; Mercheş and Agop, 2016):

i) Any continuous and non-differentiable curve of blood's structural entities (blood fractal curve) is explicitly scale resolution δt dependent, *i.e.*, its length will tend to infinity when δt will tend to zero.

Let us mention that a curve can be characterized as non-differentiable if it obeys the Lebesgue theorem (Mandelbrot, 1983), in other words its length will become infinite when the scale resolution will tend to zero. In this limit, a curve is as zig-zagged as can be imagined. Therefore, every point of this curve holds the property of self-similarity, *i.e*. it is holographic in nature (the whole will be reflected in every part) (Mandelbrot, 1983).

ii) The physics of blood phenomena are intrinsically related to the behaviour of a set of functions taking place during the magnifying operation of δt (the scale resolution). Then, by using the substitution principle, δt can be identified with dt, *i.e*., $\delta t \equiv dt$ and, thus, it will be considered an independent variable. The notation dt is reserved for the usual time, like in the Hamiltonian blood dynamics.

iii) The dynamics of blood's structural entities are described by using fractal variables (functions which are both space-time coordinates and scale resolution dependent). Therefore, in any point of the blood fractal curve, we can define two derivatives for the variable field $Q(t,dt)$:

$$\begin{aligned}\frac{d_{+}Q(t,dt)}{dt}&=\lim_{\Delta t\to 0_{+}}\frac{Q(t+\Delta t,\Delta t)-Q(t,\Delta t)}{\Delta t}\\ \frac{d_{-}Q(t,dt)}{dt}&=\lim_{\Delta t\to 0_{-}}\frac{Q(t,\Delta t)-Q(t-\Delta t,\Delta t)}{\Delta t}\end{aligned}\tag{1}$$

Here, the "+" sign has been attributed to the forward processes of blood's structural units, while the "–" sign has been attributed to the backward ones.

iv) The spatial coordinate field differential $dX^{i}(t,dt)$, employed in describing blood dynamics, can be written as the sum of these two differentials, one of which is scale resolution independent (differential part $d_{\pm}x^{i}(t)$), and the other one is scale resolution dependent (fractal part $d_{\pm}\xi^{i}(t)$), *i.e.*,

$$d_{\pm}X^{i}(t,dt)=d_{\pm}x^{i}(t)+d_{\pm}\xi^{i}(t,dt)\tag{2}$$

v) The spatial coordinate field non-differentiable part, employed in describing blood dynamics, satisfies the equation (Mandelbrot, 1983):

$$d_{\pm}\xi^{i}(t,dt)=\lambda_{\pm}^{i}(dt)^{1/D_F}\tag{3}$$

where $\lambda_{\pm}^{i}$ are constant coefficients through which the type of fractalization that describes the blood dynamics is specified and D_F gives us the fractal dimension of the blood fractal curve.

It is our opinion that blood processes imply dynamics which take place on geodesics having various fractal dimensions. These numerous fractal dimensions for blood geodesics comes as a result of the blood's structure. Specifically, for $D_F = 2$, processes of a quantum type are generated in blood dynamics. For $D_F < 2$, processes of a correlative type are induced, while for $D_F > 2$ processes of a non-correlative type can be determined (Nottale, 1989; El Naschie *et al*., 1995).

vi) The differential time reflection invariance of blood's dynamical variables is obtained by using and combining the derivatives d_{+}/dt and d_{-}/dt in the non-differentiable operator

$$\frac{\hat{d}}{dt}=\frac{1}{2}\left(\frac{d_{+}+d_{-}}{dt}\right)-\frac{i}{2}\left(\frac{d_{+}-d_{-}}{dt}\right)\tag{4}$$

Eq. (4) comes as a result of a specific mathematical procedure: the complex prolongation procedure applied to blood dynamics (Mercheș and Agop, 2016; Cresson, 2006). Applying the non-differentiable operator to the spatial coordinate field, employed in describing blood dynamics, blood's complex velocity field can be obtained:

$$\hat{V}^i = \frac{\hat{d}X^i}{dt} = V_D^i - V_F^i \tag{5}$$

with

$$V_D^i = \frac{1}{2}\frac{d_+X^i + d_-X^i}{dt}, \quad V_F^i = \frac{1}{2}\frac{d_+X^i - d_-X^i}{dt} \tag{6}$$

The real part V_D^i of the blood complex velocity field is differentiable and independent of scale resolution (differentiable velocity field). The imaginary part V_F^i is non-differentiable and depends on the scale resolution (fractal velocity field).

vii) Not taking into account any external constraint, an infinity of geodesics that can relate any pair of points can be found, it being true for all scales of blood dynamics. In blood's fractal space, the geodesics themselves substitute all of the structural units. In this way, any external constraint can be defined as a selection of blood geodesics. The infinite number of geodesics, their non-differentiability and the two values of the derivative imply a generalized statistical fluid-like description (blood). Therefore, the average values of the blood variables must be analyzed in the above-stated sense, so that the average of $d_\pm X^i$ is

$$\left\langle d_\pm X^i \right\rangle \equiv d_\pm x^i \tag{7}$$

with

$$\left\langle d_\pm \xi^i \right\rangle = 0 \tag{8}$$

Eq. (8) implies that the mean of the fractal fluctuations is null.

viii) By using a covariant derivative we can describe blood dynamics. The explicit form of this derivative can be thus obtained. We consider now that the blood fractal curves are immersed in a 3-dimensional space and that X^i is the spatial coordinate field of a point from this fractal curve. Let us also consider a variable field $Q\left(X^i, t\right)$ and the Taylor expansion up to the second order

$$d_\pm Q\left(X^i, t\right) = \partial_t Q dt + \partial_i Q d_\pm X^i + \frac{1}{2}\partial_l \partial_k Q d_\pm X^l d_\pm X^k \tag{9}$$

The above-written relations are valid in any point and more for the points X^i on the blood fractal curve that we have selected in Eq. (9). It results that the blood variables' forward and backward values from (9) can be written as

$$\left\langle d_\pm Q \right\rangle = \left\langle \partial_t Q dt \right\rangle + \left\langle \partial_i Q d_\pm X^i \right\rangle + \frac{1}{2}\left\langle \partial_l \partial_k Q d_\pm X^l d_\pm X^k \right\rangle \tag{10}$$

Let us now suppose that the mean values of the all variable field Q and its derivatives coincide with themselves and also that the differentials $d_\pm X^i$ and dt are independent. It results that the average of their products is the same as the product of averages. Hence (10) becomes

$$d_{\pm}Q = \partial_t Q dt + \partial_i Q\left\langle d_{\pm}X^i\right\rangle + \frac{1}{2}\partial_l\partial_k Q\left\langle d_{\pm}X^l d_{\pm}X^k\right\rangle \tag{11}$$

Even if the mean value of $d_{\pm}\xi^i$ is null, a different situation can be observed for the higher order of $d_{\pm}\xi^i$. We will now shift our focus on the averages $\left\langle d_{\pm}\xi^l d_{\pm}\xi^k\right\rangle$. Taking Eq. (3) into account it results that

$$\left\langle d_{\pm}\xi^l d_{\pm}\xi^k\right\rangle = \pm\lambda_{\pm}^l\lambda_{\pm}^k\left(dt\right)^{(2/D_F)-1}dt \tag{12}$$

where it has been accepted that the sign + corresponds to $dt>0$ and the sign – corresponds to $dt<0$

Then, Eq. (11) can be written as follows:

$$d_{\pm}Q = \partial_t Q dt + \partial_i Q\left\langle d_{\pm}X^i\right\rangle + \frac{1}{2}\partial_l\partial_k Q d_{\pm}x^l d_{\pm}x^k \pm \frac{1}{2}\partial_l\partial_k Q\left[\lambda_{\pm}^l\lambda_{\pm}^k\left(dt\right)^{(2/D_F)-1}dt\right] \tag{13}$$

Now, if we divide by *dt* and we do not take into consideration the terms that contain differential factors, we can write:

$$\frac{d_{\pm}Q}{dt} = \partial_t Q + v_{\pm}^i\partial_i Q \pm \frac{1}{2}\lambda_{\pm}^l\lambda_{\pm}^k\left(dt\right)^{(2/D_F)-1}\partial_l\partial_k Q \tag{14}$$

Then we can define the operators

$$\frac{d_{\pm}}{dt} = \partial_t + v_{\pm}^i\partial_i \pm \frac{1}{2}\lambda_{\pm}^l\lambda_{\pm}^k\left(dt\right)^{(2/D_F)-1}\partial_l\partial_k \tag{15}$$

In this context, if Eqs. (4), (5) and (15) are taken into account, we can then calculate $\hat{d}/dt$. It results:

$$\frac{\hat{d}Q}{dt} = \partial_t Q + \hat{V}^i\partial_i Q + \frac{1}{4}\left(dt\right)^{(2/D_F)-1}D^{lk}\partial_l\partial_k Q \tag{16}$$

where

$$\begin{aligned} &D^{lk} = d^{lk} - i\bar{d}^{lk} \\ &d^{lk} = \lambda_+^l\lambda_+^k - \lambda_-^l\lambda_-^k, \quad \bar{d}^{lk} = \lambda_+^l\lambda_+^k + \lambda_-^l\lambda_-^k \end{aligned} \tag{17}$$

Eq. (16) permits us to define the blood dynamics' covariant derivative in the form

$$\frac{\hat{d}}{dt} = \partial_t + \hat{V}^i\partial_i + \frac{1}{4}\left(dt\right)^{(2/D_F)-1}D^{lk}\partial_l\partial_k \tag{18}$$

3. Results and Discussions

If the principle of scale covariance is taken into consideration (the laws of physics - blood dynamics specific - are scale transformations invariant) we can state that the transition from differentiable physics to non-differentiable physics can be solved by substituting the standard time derivative d/dt with the non-differentiable operator $\hat{d}/dt$. This operator plays the role of a scale

covariant derivative, *i.e.* we can use it to write the blood dynamics' fundamental equations just like the ones in the differentiable case. Taking these into account, if we apply operator (18) to the complex velocity field (5), and without any external constraint, then the blood geodesics can be written as:

$$\frac{\hat{d}\hat{V}^i}{dt} = \partial_t \hat{V}^i + \hat{V}^l \partial_l \hat{V}^i + \frac{1}{4}(dt)^{(2/D_F)-1} D^{lk} \partial_l \partial_k \hat{V}^i = 0 \tag{19}$$

We can thus observe that the local acceleration $\partial_t \hat{V}^i$, the convection $\hat{V}^l \partial_l \hat{V}^i$ and the dissipation $D^{lk} \partial_l \partial_k \hat{V}^i$, are balanced in any point of the blood fractal curve. Furthermore, the complex coefficient of viscosity-type $4^{-1}(dt)^{(2/D_F)-1} D^{lk}$ present in the bloods dynamics specifies that this is a rheological medium. Therefore, we can state that it has memory by its own structure.

If fractalisation is achieved through Markov-type stochastic processes, involving Lévy type movements of blood's structural entities, then:

$$\lambda_+^i \lambda_+^l = \lambda_-^i \lambda_-^l = 2\lambda \delta^{il} \tag{20}$$

where δ^{il} is the Kronecker's pseudo-tensor.

In this context, the equation of blood geodesics takes the following form

$$\frac{\hat{d}\hat{V}^i}{dt} = \partial_t \hat{V}^i + \hat{V}^l \partial_l \hat{V}^i - i\lambda (dt)^{(2/D_F)-1} \partial^l \partial_l \hat{V}^i = 0 \tag{21}$$

or more, if we separate movements on differential and fractal scale resolutions,

$$\begin{aligned} \frac{\hat{d}V_D^i}{dt} &= \partial_t V_D^i + V_D^l \partial_l V_D^i - \left[V_F^l - \lambda (dt)^{(2/D_F)-1} \partial^l \right] \partial_l V_F^i = 0 \\ \frac{\hat{d}V_F^i}{dt} &= \partial_t V_F^i + V_D^l \partial_l V_F^i - \left[V_F^l - \lambda (dt)^{(2/D_F)-1} \partial^l \right] \partial_l V_D^i = 0 \end{aligned} \tag{22}$$

Employing the procedure from (Solovăstru *et al.*, 2016), we can model, at a fractal scale resolution, the blood dynamics by using the following equations:

$$\partial_t V_F{}^i + V_F{}^l \partial_l V_F{}^i = \lambda (dt)^{(2/D_F)-1} \partial^l \partial_l V_F{}^i \tag{23}$$

$$\partial_i V_F{}^i = 0 \tag{24}$$

Therefore, at a fractal scale resolution, the specific impulse conservation law is expressed through Eq. (23), while the states density conservation law is explicitated by Eq. (24).

Finding the solutions for Eqs. (23) and (24) can be quite difficult, because this equation system is a non-linear one (Batchelor, 2000; Landau and Lifshitz, 1987). However, one can find an analytical solution of this systems, in the case of a plane symmetry (*x*,*y*) stationary flow. In this context, Eqs. (23) and (24) for $\boldsymbol{V}_F = (V_x, V_y, 0)$ take the form:

$$V_x \frac{\partial V_x}{\partial x} + V_y \frac{\partial V_x}{\partial x} = \lambda (dt)^{(2/D_F)-1} \frac{\partial^2 V_x}{\partial y^2} \tag{25}$$

$$\frac{\partial V_x}{\partial x}+\frac{\partial V_y}{\partial y}=0 \tag{26}$$

We can also write the boundary conditions for the flow:

$$\lim_{y\to 0}V_y\left(x,y\right)=0,\ \lim_{y\to 0}\frac{\partial V_x}{\partial y}=0,\ \lim_{y\to\infty}V_x\left(x,y\right)=0 \tag{27}$$

and also state that the flux momentum per length unit is constant

$$\Theta=\rho\int_{-\infty}^{+\infty}V_x{}^2dy=\text{const.} \tag{28}$$

By using the method developed in (Solovăstru *et al.*, 2016; Batchelor, 2000; Landau and Lifshitz, 1987) for solving Eqs. (25) and (26), with the limit conditions (27) and (28), we find the following solutions:

$$V_x=\frac{\left[1.5\left(\frac{\Theta}{6\rho}\right)^{2/3}\right]}{\left[\lambda\left(dt\right)^{(2/D_F)-1}x\right]^{1/3}}\cdot\sec\mathrm{h}^2\frac{\left[\left(0.5y\right)\left(\frac{\Theta}{6\rho}\right)^{1/3}\right]}{\left[\lambda\left(dt\right)^{(2/D_F)-1}x\right]^{2/3}} \tag{29}$$

$$V_y=\frac{\left[4.5\left(\frac{\Theta}{6\rho}\right)^{2/3}\right]}{\left[3\lambda\left(dt\right)^{(2/D_F)-1}x\right]^{1/3}}\cdot \left[\frac{y\left(\frac{\Theta}{6\rho}\right)^{1/3}}{\left[\lambda\left(dt\right)^{(2/D_F)-1}x\right]^{2/3}}\cdot\sec\mathrm{h}^2\frac{\left[\left(0.5y\right)\left(\frac{\Theta}{6\rho}\right)^{1/3}\right]}{\left[\lambda\left(dt\right)^{(2/D_F)-1}x\right]^{2/3}}-\tanh\frac{\left[\left(0.5y\right)\left(\frac{\Theta}{6\rho}\right)^{1/3}\right]}{\left[\lambda\left(dt\right)^{(2/D_F)-1}x\right]^{2/3}}\right] \tag{30}$$

Eqs. (29) and (30) show that the blood velocity field holds a high degree of non-linearity through topological solitons of kink-type (tanh), non-topological solitons of breather-type (sech^2), and also through topological – non-topological soliton mixtures of kink-breather-type (sech^2-tanh). Due to the fact that blood has an increased structural complexity, (due to its various structural entities, that retain their own velocity field) we will write Eqs. (29) and (30) in a more accurate way, so that indexes will be assigned for each component.

For $y=0$, we will obtain in Eq. (29) the blood's flow critical velocity in the form

$$V_x\left(x,y=0\right)=V_c=\frac{\left[1.5\left(\frac{\Theta}{6\rho}\right)^{2/3}\right]}{\left[\lambda\left(dt\right)^{(2/D_F)-1}x\right]^{1/3}} \tag{31}$$

while relation (28), taking into account (31), becomes

$$\Theta = \rho \int_{-\infty}^{+\infty} V_x^{\,2}(x,y)dy = \int_{-d_c}^{+d_c} V_c^{\,2}(x,0)dy \tag{32}$$

so that the critical cross section of the strain lines of the blood is given by:

$$d_c(x, y=0) = \frac{\Theta}{2\rho V_c^{\,2}} = 2.42\left[\lambda(dt)^{(2/D_F)-1} x\right]^{2/3}\left(\frac{\rho}{\Theta}\right)^{1/3} \tag{33}$$

Eqs. (29) and (30) can be clearly simplified if the normalized quantities are used

$$\xi = \frac{x}{x_0}, \eta = \frac{y}{y_0}, u = \frac{V_x}{w_o}, v = \frac{V_y}{w_0},$$

$$\Omega = \frac{\left(\frac{\Theta}{6\rho}\right)^{2/3}}{w_0\left[\lambda(dt)^{(2/D_F)-1} x_o\right]^{1/3}}, \omega = \frac{\left(\frac{\Theta}{6\rho}\right)^{1/3} y_0}{\left[\lambda(dt)^{(2/D_F)-1} x_o\right]^{2/3}} \tag{34}$$

where x_0, y_0, w_0 are the specific lengths and the specific velocity, respectively, of blood's laminar flow. It results that

$$u(\xi,\eta) = \frac{1.5\Omega}{\xi^{1/3}}\operatorname{sech}^2\left(\frac{0.5\Omega\omega\eta}{\xi^{2/3}}\right) \tag{35}$$

$$v(\xi,\eta) = \frac{4.5^{2/3}}{3^{1/3}}\frac{\Omega}{\xi^{1/3}}\left[\frac{\omega\eta}{\xi^{2/3}}\operatorname{sech}^2\left(\frac{0.5\Omega\omega\eta}{\xi^{2/3}}\right) - \tanh\left(\frac{0.5\Omega\omega\eta}{\xi^{2/3}}\right)\right] \tag{36}$$

The dependence of the normalized velocity field u on the normalized spatial coordinates ξ, η for various nonlinearity degrees (ω = 0.3; 6) are shown in Figs. 1a, b and 2a, b.

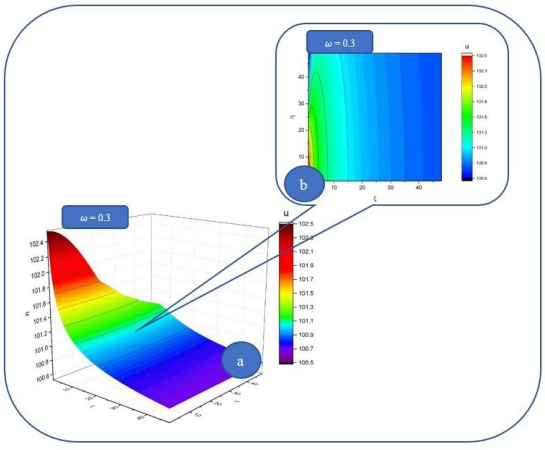

Fig. 1 – The normalized velocity field u dependence on the normalized spatial coordinates ξ, η for the nonlinearity degree ω = 0.3: (*a*) 3D representation and contour plot (*b*).

These results show that the velocity field along the blood flow direction (ξ) is lightly affected by the nonlinearity degree (there is always a decrease in velocity on the flow axes no matter the degree of nonlinearity). But we must also highlight that the blood flow direction (η) is heavily affected. Blood flow starts from constant values on the η axis, and we must highlight that, with the increase of ω, preferential blood flow directions can be identified.

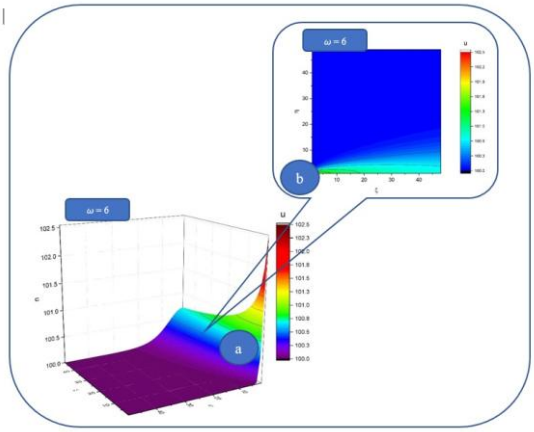

Fig. 2 – The normalized velocity field u dependence on the normalized spatial coordinates ξ, η for the nonlinearity degree ω = 6: (a) 3D representation and contour plot (b).

We represent in Figs. 3a, b and 4a, b the dependences of the normalized velocity field u on the normalized spatial coordinates ξ, η for various nonlinearity degrees (ω = 0.3; 6). For small degrees of nonlinearity, the variations of the velocity field behave in a similar matter on both directions (ξ, η). We can also see that for higher values of the nonlinearity degree these variations are only focused in a single direction (ξ).

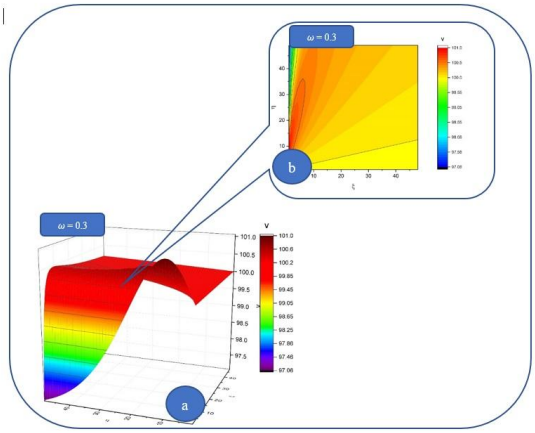

Fig. 3 – 3D representation (a) and the contour plot (b) of the normalized velocity field v on the normalized spatial coordinates ξ, η for the nonlinearity degree ω = 0.3.

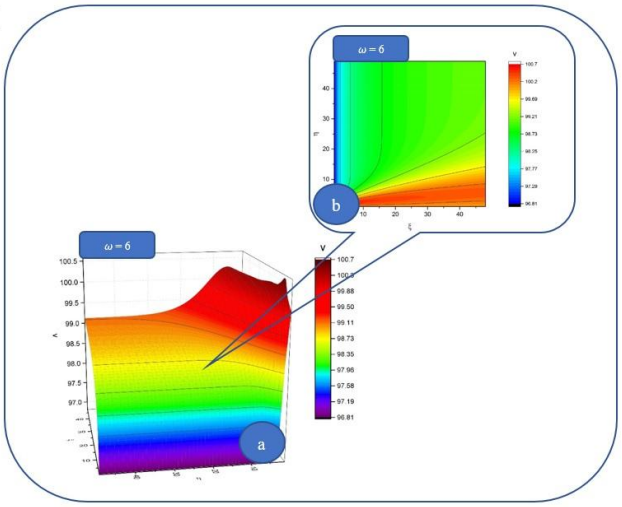

Fig. 4 – The 3D representation (*a*) and the contour plot (*b*) of the normalized velocity field v on the normalized spatial coordinates ξ, η for the nonlinearity degree $\omega = 6$.

Considering the above, we can see that the force with which blood will act upon the walls of the flow vessels can be crucial for understanding arterial occlusion and other circulatory system diseases.

In our model the normalized force is given by:

$$f(\xi,\eta)=\partial_\eta u-\partial_\xi v=-\frac{1.5\Omega\operatorname{sech}^2\left(\frac{0.5\omega\eta}{\xi^{2/3}}\right)\tanh\left(\frac{0.5\omega\eta}{\xi^{2/3}}\right)\omega}{\xi}-\frac{1}{\xi^{1/3}}\cdot$$
$$\cdot\left\{0.9\Omega\left(-\frac{2}{3}\frac{\omega\eta\operatorname{sech}^2\left(\frac{0.5\omega\eta}{\xi^{2/3}}\right)}{\xi^{5/3}}+\frac{0.66\omega^2\eta^2\operatorname{sech}^2\left(\frac{0.5\omega\eta}{\xi^{2/3}}\right)\tanh\left(\frac{0.5\omega\eta}{\xi^{2/3}}\right)}{\xi^{7/3}}+\right.\right.$$
$$\left.\left.+\frac{0.33\left[\left(1-\tanh^2\left(\frac{0.5\Omega\eta}{\xi^{2/3}}\right)\right)\omega\eta\right]}{\xi^{5/3}}\right)3^{\frac{3}{2}}+\right.$$
$$\left.0.3\Omega\left(\frac{\omega\eta\operatorname{sech}^2\left(\frac{0.5\omega\eta}{\xi^{2/3}}\right)}{\xi^{2/3}}-\tanh\left(\frac{0.5\omega\eta}{\xi^{2/3}}\right)\right)3^{\frac{3}{2}}\right\}$$
$$+\frac{}{\xi^{4/3}} \tag{37}$$

Figs. 5a, b and 6a, b show, for various degrees of nonlinearity, the evolution of the normalized force field on the two-flow direction (ξ, η). Thus, we can see that if blood's nonlinearity increases, the force aimed at the walls also increases. This can lead to developing a framework for understanding the complex mechanisms which appear in various occlusions of arteries (partial or total occlusions) – see Fig. 7.

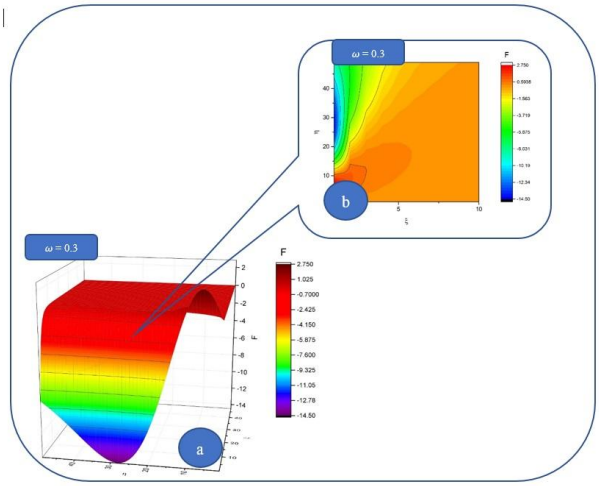

Fig. 5 – The dependence of the normalized force field F of a blood flow on the vessels, on the normalized spatial coordinates ξ, η for two resolution scales: 3D representation (a) and contour plot (b) for the nonlinearity degree ω = 0.3.

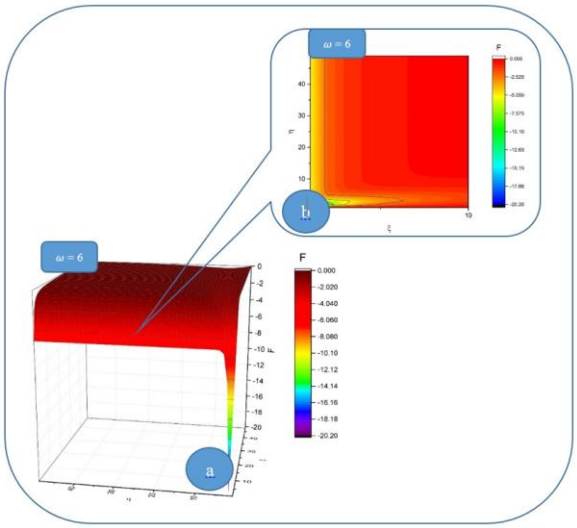

Fig. 6 – The dependence of the normalized force field F of a blood flow on the vessels, on the normalized spatial coordinates ξ, η for two resolution scales: 3D representation (a) and contour plot (b), for the nonlinearity degree ω = 6.

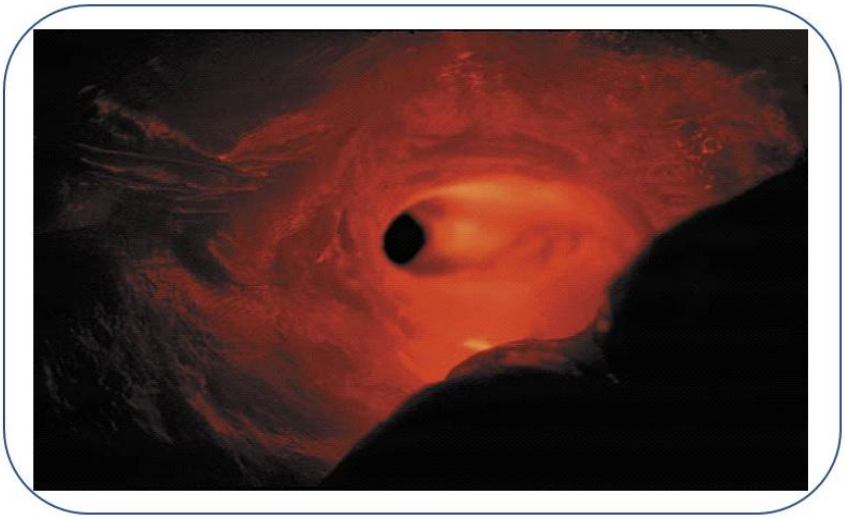

Fig. 7 – Endoluminal view of a major atherosclerotic plaque with thrombus (image obtained through optical microscopy).

Our new theoretical model explains, by employing fractality, the atherogenesis process (Tesloianu *et al*., 2015), basically "adapting" to the most commonly used (classical) anatomical and histopathological descriptions; in this context, this fractal physics model represents an original method for sustaining well documented research regarding the morpho-pathological aspects of blood flow. In specialized literature one can find a large number of microscopy images that describe the spatial-temporal hologram of the phenomenon; we can thus observe non-fractal - fractal and microscopic - macroscopic translations through holographically reproductible auto-similarity (Tesloianu *et al*., 2015). In our opinion, fractality introduces new mathematical and semantic notions for defining atherogenesis. Therefore, the process if atherogenesis can be accurately described by fractal physics methods.

4. Conclusions

The present paper develops a fractal model for analyzing blood flow dynamics. In the case of a laminar flow of the blood, we obtain and apply fractal hydrodynamic equations.

In this way a new model for blood flow and for cholesterol deposition on blood vessel walls can be designed. Our results showed that a directional flow aimed at the walls can be determined. In our opinion, this can be an explanation for why the thickening effect appears, it being one of the main causes of atherosclerosis.

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UN MODEL FRACTAL PENTRU DINAMICILE DE CURGERE ALE SÂNGELUI

(Rezumat)

În această lucrare propunem un model original pentru analiza dinamicilor curgerii sangvine folosind Teoria Relativității de Scară într-o dimensiune fractală constantă arbitrară în reprezentarea hidrodinamică fractală. Rezultatele arată o curgere direcționată către pereți. Acest lucru ar putea explica în opinia noastră atât efectul de îngroșare al vaselor, sursa principală a aterosclerozei, cât și depunderea de colesterol pe pereții vaselor de sânge.