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J Tsafack Nzifack, J Mabekou Takam, Rostand Moutou Pitti, M Fogue, P Talla. Study of the Influence of Plaque Growth and Hydrostatic Properties in an Atherosclerotic Artery, for the Prevention of Arterial Wall Damage: Application to Vascular Diseases. *Advances in Materials Science and Engineering*, 2021, 2021, pp.10. 10.1155/2021/6664988 . hal-03468634

HAL Id: hal-03468634

<https://uca.hal.science/hal-03468634v1>

Submitted on 7 Dec 2021

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Research Article

Study of the Influence of Plaque Growth and Hydrostatic Properties in an Atherosclerotic Artery, for the Prevention of Arterial Wall Damage: Application to Vascular Diseases

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Received 27 October 2020; Accepted 24 April 2021; Published 10 May 2021

Academic Editor: Jean-Michel Bergheau

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The objective of this work is to prevent damage of the arterial wall, using a theoretical model of hyperelastic, anisotropic, and dynamic behavior of the human arterial. This work is mainly focused on the properties of the hydrostatic stress and the evolution of stenosis. This work is mainly focused on the properties of the hydrostatic stress and the evolution of stenosis in order to understand the effect of the size of the plaque deposit, the loss of elasticity of the wall, and the increase in the density of the blood on the mechanical behavior of the human arterial wall. The great contribution of this work shows us that increasing the size of the plaque also increases arterial stress, and the radial growth of the plaque is very dangerous compared to the longitudinal growth. Furthermore, atherosclerosis promotes the loss of elasticity of the arterial wall and increases the density of blood mass. Indeed, all these subsequent phenomena increase arterial stress. All the results are in good agreement with the expected result of the literature and could play an important role in the diagnosis of the patient with an arterial injury. It will also help the doctor and the surgeon to make a good clinical decision and good treatment planning.

1. Introduction

Health problems are a great concern for people today [1]. For centuries, cardiovascular disease has been identified as one of the major diseases affecting many people [2]. Stenosis or arteriosclerosis is the abnormal and unnatural growth of the thickness of the arterial wall, which develops at various locations in the cardiovascular system under conditions of pathology [3, 4]. This may be due to unhealthy living conditions such as exposure to tobacco smoke, lack of physical activity, and poor eating habits. Carotid stenosis artery is a narrowing of the large arteries that carry blood to the head, face, and brain [5]. The narrowing is usually the result of an accumulation of plaque in the arteries (atherosclerosis). The stenosis can worsen over time and

completely block the artery, which can lead to a stroke [5]. Thus, it leads to the modification of blood pressure, causing flow disorders and increased hemodynamic stress of the arterial wall, which will damage the endothelium [6]. It is therefore important to study the hemodynamic stress of the artery in relation to the modification (of the density of the blood, the lumen of the artery, and the elasticity of the wall) and to understand the evolution of the parameter that is associated with arterial pathology. For this, we carried out the modelling of the hemodynamic stress of the elastic artery, with variation of the degree of stenosis, of the density of the blood, and of the elasticity of the wall. Yet in the clinical field, doctors and surgeons determined the arterial narrowing by simply measuring the height of the stenosis [7]. This is insufficient for clinical decision-making and

treatment planning because it is not the only geometric parameter affecting the stress of the arterial wall. Thus, the anomalies of the deformation of the arteries due to narrowing, dilation, and properties of materials such as the elasticity of the wall and the mass density of the blood should be considered. Indeed, the latter allows us to assess the parameters of hemodynamic stress more realistically, and this in turn allows doctors to better understand arterial disease and the consequences of the progression of plaque growth, the modification of the mass density of blood, and elasticity of the wall. To achieve this, we will focus our view on certain works in the literature on the mechanical behaviour of the arterial wall. For example, reference [8] has shown the involvement of the disturbance of the metabolism of smooth muscle cells and the degradation of collagen in the medial layer with the appearance of aneurysm and arterial dissection. To understand the tear phase of an aortic dissection, reference [9] studied the response of the thoracic aorta due to radial and longitudinal forces. The acquisition of the modification of the mechanical properties of the different arterial layers in vivo is undertaken in reference [10] to demonstrate the recovery of the wall during a pathological manifestation. The evaluation of Von-Mises stress was investigated in reference [11] to show that the maximum stress is at the media level and to validate its numerical simulations with the results obtained by medical techniques. For the aneurysm rupture study, references [12, 13] used hemodynamic, morphological, and mechanical parameters. The variation of viscosity of the fluid was considered by reference [14] to study the shear stresses generated on the wall of the aneurysm. Reference [15] studied health problems that have apparently concerned people today [1]. Stenosis or arteriosclerosis is the abnormal and unnatural growth of the thickness of the arterial wall which develops at various places in the cardiovascular system under conditions of pathology [3, 4]. This may be due to unhealthy living conditions such as exposure to tobacco smoke, lack of physical activity, and poor eating habits. Carotid stenosis artery is a narrowing of the large arteries that carry blood to the head, face, and brain [5]. The narrowing is usually the result of an accumulation of plaque in the arteries: atherosclerosis. The stenosis can worsen over time and completely block the artery, which can lead to a stroke [5]. Thus, it leads to the modification of blood pressure, causing flow disorders and increased hemodynamic stress of the arterial wall, which will damage the endothelium [6]. It is therefore important to study the hemodynamic stress of the artery in relation to the modification (of the density of the blood, the lumen of the artery, and the elasticity of the wall) and to understand the evolution of the parameter that is associated with arterial pathology. For this, we carried out the modelling of the hemodynamic stress of the elastic artery, with variation of the degree of stenosis, of the density of the blood, and of the elasticity of the wall. Yet in the clinical field, doctors and surgeons determined the arterial narrowing by simply measuring the height of the stenosis [7]. This is insufficient for clinical decision-making and treatment planning because it is not the only geometric parameter affecting the stress of the arterial wall. Thus, the anomalies of the deformation of the arteries due to narrowing, dilation, and properties of materials

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2. Theoretical Modeling of the Mechanical Behaviour of the Arterial Wall

This article is concerned with the mathematical modelling of the effects of deposit plaque on the mechanical properties of the soft biological tissues that constitute the walls of arteries. Many important aspects of the mechanical behaviour of

arterial tissue can be treated on the basis of elasticity theory. The focus of the article is therefore on the constitutive modelling of the anisotropies and highly nonlinear elastic properties of the atherosclerosis artery wall. The fundamental features of the relevant theory are summarized briefly, and particular forms of the elastic strain-energy function are discussed and then applied to an artery considered as a thick wall, a circular cylindrical tube in order to illustrate its extension inflation behaviour. The wide range of applications of the constitutive modelling framework to artery walls in both health and disease and to the other fibrous soft tissues is discussed in detail.

2.1. Kinematics. The kinematics of the structure can be described by two successive transformations in a system of cylindrical coordinates relative to the base (e_r, e_θ, e_z) ; the deformation is measured by the gradient of the F transformation [16]:

$$F = \begin{bmatrix} \lambda_r & 0 & 0 \\ 0 & \lambda_\theta & 0 \\ 0 & 0 & \lambda_z \end{bmatrix}, \quad (1)$$

where $\lambda_r, \lambda_\theta$, and λ_z are the main elongations in the radial, circumferential, and axial directions, respectively. The right and left Cauchy-green expansion tensor is given by $C = F^t F$ and $B = FF^t$ and F^t represents the transpose tensor F .

2.2. Anisotropic Behavioural Relationship. The arterial wall is essentially composed of water, it is generally considered an incompressible medium [17], and the fluid movement inside the porous tissue induces stress gradients. This hypothesis of wall incompressibility has been demonstrated by experimental studies. Reference [18] has shown that artery samples deform in isochoric ways without the action of a large number of different loads.

In this work, we have exploited the deformation energy of Holzapfel, proposed in 2005. This energy takes into account the radial dispersion of fiber distribution in the arterial wall and has the following expressions:

$$W = (I_1, I_4) = \frac{\mu}{2} (I_1 - 3) + \frac{k_1}{k_2} \left[\exp k_2 \left((1 - \rho)(I_1 - 3)^2 + \rho(I_1 - 3)^2 \right) \right] - 1, \quad (2)$$

where $\rho \in [0, 1]$ is a dispersion factor that regulates the degree of anisotropy [19]. In an original way, if $\rho = 0$, we obtain the composition of a neo-Hookean model with the [20] model, and if $\rho = 1$, we obtain the [17] model. The [21] model has been used by several authors to study physiological stress by different arterial segments. Reference [22] uses this model to simulate coronary surgery and also by [23] to model the behaviour of healthy arteries. Finally, this model will be the most appropriate in our current study.

2.3. Stress. Cauchy's stress can be broken down into a passive contribution and an active contribution [16].

2.3.1. Passive Contribution. The Cauchy tensor is given by

$$\sigma = \frac{2}{J} F \frac{\partial W(I_1, I_4)}{\partial c} F^t, \quad (3)$$

with

$$\begin{aligned} I_1 &= \lambda_r^2 + \lambda_\theta^2 + \lambda_z^2, \\ I_4 &= \lambda_\theta^2 \cos(\varphi) + \lambda_z^2 \sin(\varphi). \end{aligned} \quad (4)$$

For an incompressible material $J = \det F$, the work of [18] has shown that the arterial wall is incompressible and the Cauchy stress becomes $\sigma = -\Delta P I + (2/J) F (\partial W(I_1, I_4)/\partial c) F^t$, where ΔP represents a hydrostatic stress contribution. Let us now write this expression in the form of [24]

$$\begin{aligned} \sigma &= -\Delta p I + 2W_1 B + 2W_2 (I_1 1 - B) B + 2W_4 a \otimes a \\ &\quad + 2W_5 (a \otimes Ba + Ba \otimes a), \end{aligned} \quad (5)$$

where \otimes represents the tensor product.

With $W_j = (\partial W / \partial I_j)$ ($j = 1, 2, 3, 4, 5$). It should also be noted that an energy function that characterizes a material must respect the principle of material indifference, also called the principle of objectivity. It must be independent of the reference system. Other mathematical criteria must normally be met, such as convexity and ellipticity.

(1) Modeling of Hydrostatic Stress. In continuum mechanics, a hydrostatic stress is anisotropy stress that is given by the movement of blood inside the artery. It is often used interchangeably with pressure and is also known as confining stress. Its magnitude can be given by

$$\Delta P = R_p Q, \quad (6)$$

where R_p is the resistance and Q the blood flow. This relationship is only valid for a perfectly developed and symmetric axial flow. The resistance explains the viscous loss along the pipe and is calculated as follows:

$$R_p = \frac{8\mu L}{\pi r^4}, \quad (7)$$

where μ is the dynamic viscosity of the blood and L and r are the length and radius of the vessel, respectively. Due to the fact that the resistance is proportional to the radius, a small change or a great change of the radius will modify the resistance. This can be caused by the presence of the stenosis in the lumen of the artery.

(2) Modeling of Stenosis. Fat accumulation, observed in the arterial stenosis, forms a rough and uneven surface, thus decreasing the effective diameter of the vessel. In a 0D representation of the vessel, a stenosis can be modeled with ad-hoc resistance. To calculate the resistance, the vessel is divided into two segments, one part stenosis and the other part steroid. For simplicity, the radius lumen in the stenosis

portion is assumed to be a constant value along the stenosis. Figure 1 shows the idealized geometry of a stenosis.

The effective resistance of the vessel stenosis can be calculated as follows:

$$R_p = \frac{8\mu}{\pi r_0^4} (l_0 - l_s) + \frac{8\mu}{\pi r_s^4} l_s, \quad (8)$$

where r_0, l_0 represent the resistance and length of the healthy part of the vessel. r_s, l_s denote the resistance and length of the stenosis part of the vessel.

Since our aim is to study the dynamic of blood flow of the cerebral circulation, it will be advantageous to fix our eyes on the Fahraors–Lindqvist effect, which will allow us to express the resistance above according to the discharge hematocrit H_D and the vessel diameter D , defining the μ viscosity in vivo as

$$\mu = \mu_p \left[1 + (\mu_1^{0.45} - 1) \frac{(1 - H_D^c) - 1}{(1 - 0.45)^c - 1} * \left(\frac{D}{D - 1.1} \right)^2 \right] * \left(\frac{D}{D - 1.1} \right)^2, \quad (9)$$

with μ_p the viscosity of the initial plasma $9 * 10^{-6}$ mmHgS and $\mu_1^{0.45}$ the viscosity of the blood for a hematocrit flow of 0.45, which is given by the expression

$$\mu_1^{0.45} = 6 \exp(-0.085 D) + 3.2 - 2.44 \exp(-0.006 D^{0.0645}),$$

$$C = (0.8 + \exp(-0.075 D)) * \left(-1 + \frac{1}{1 + 10^{-11} D^{12}} \right) + \frac{1}{1 + 10^{-11} D^{12}}. \quad (10)$$

(3) Artery Brain Posterior.

Model definition

The modified Lumped parameter model [26] is to study CBF regulation. A lumped parameter model was developed (Figure 2), describing the segment of the posterior cerebral artery (PCA) and its distal vessels, up to the large cerebral veins. This segment was chosen since it supplies the primary visual cortex with blood [27]. The model consists of three lumped parts: the PCA, the arteriolar circulation and microcirculation, and the venous circulation.

The vessel compliance (C_p)

The PCA is modeled by a line model. Full PCA compliance (C_p) is considered using a capacitor

$$C_p = \frac{3\pi r_p^2 \left(\left(\frac{r_p}{h_p} \right) + 1 \right)^2}{E_p \left(\left(\frac{r_p}{h_p} \right) + 1 \right)} l_p, \quad (11)$$

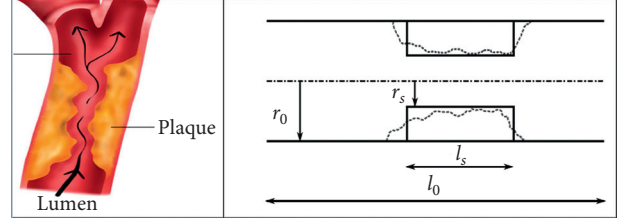


FIGURE 1: Sketch of arterial vessel segment with stenosis and 0D-model [25].

where $r_p, E_p, h_p,$ and l_p are in respect artery inner radius, Elasticity modulus, thickness of artery and PCA length.

For the reason of this simulation, it should be noted that, by passing blood thorough vessels, the vessels would be expanded or contracted. So, they can keep blood or release it and this is exactly like what a capacitor does.

The blood inertia (L_p)

The blood inertia (L_p) is simulated by inductors:

$$L_p = \frac{\rho_b l_p}{\pi r_p^2}, \quad (12)$$

where $\rho_b, l_p,$ and r_p are, respectively, blood density, PCA length, and artery inner radius.

The reason for this consideration is the variability of flow acceleration in pulsatile blood flow. So, an inductor can model inertia of blood flow very clearly.

(4) State Equations of Lumped Parameter Model. The states equations of lumped parameter and its parameters are given in this section

Artery brain posterior

The PCA model involves three equations of states:

$$\frac{dP_{ai}}{dt} = \frac{2(Q_{ai} - Q_{lp})}{C_p},$$

$$\frac{dQ_{lp}}{dt} = \frac{P_{ai} - P_p - Q_{lp} R_p}{L_p}, \quad (13)$$

$$\frac{dP_p}{dt} = \frac{2(Q_{lp} R_a - P_p + 2P_a)}{R_a C_p},$$

where $P_{ai}, Q_{ai}, Q_{lp}, C_p, P_p, R_p, R_a,$ and P_a are respect arterial input, PCA inflow, inertia flow, compliance, PCA output pressure, PCA resistance, arteriolar resistance, and central arteriolar.

The passive becomes when $\Delta p = P_{ai}$ before stenoses and et $\Delta p = P_p$ after stenoses

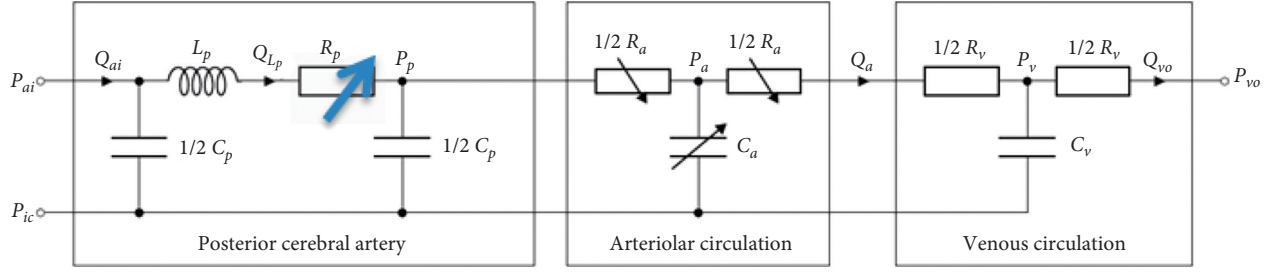


FIGURE 2: Lumped parameter model of the posterior cerebral artery (PCA) and its distal arteriolar and venous beds. P_{ai} , P_{ic} , and P_{v0} : arterial entry, intracranial, and venous exit blood pressure. P_p : output pressure of the PCA. P_a and P_v : central and venous blood pressure. Q_{ai} and Q_{Lp} : PCA input and inertia flow. Q_a and Q_{v0} : venous entry and exit streams. C_p , L_p , and R_p : PCA compliance, inertia, and resistance, respectively. c_a and R_a : arteriolar compliance and resistance. c_v and R_v : venous compliance and resistance. Posterior cerebral [26].

$$\begin{aligned}\sigma &= -P_{ai}I + 2W_1B + 2W_2(I_1 - B)B + 2W_4a \otimes a \\ &\quad + 2W_5(a \otimes Ba + Ba \otimes a), \\ \sigma &= -P_pI + 2W_1B + 2W_2(I_1 - B)B + 2W_4a \otimes a \\ &\quad + 2W_5(a \otimes Ba + Ba \otimes a).\end{aligned}\quad (14)$$

2.3.2. *Active Stress*. The first active stress models were developed in the context of small deformation [28] and then extended in the context of large deformation [29–31]. Thus, the mode of [32] allows to analyze the dynamic behavior of the arterial wall. This model has for expression:

$$\sigma_{act} = \lambda_i T_0 [C_a^{2+}] \left(1 - \left(\frac{\lambda_m - \lambda_i}{\lambda_m - \lambda_0} \right)^2 \right) (e_i \otimes e_i), \quad (15)$$

where \otimes is the tensor product, λ_i is the elongation in the direction of the smooth muscle cells, and λ_m is the elongation by which the stress is maximum and λ_0 the one for which it stops $T_0 [C_a^{2+}]$ and the activation function is function of calcium. his model was modified by reference [33] by taking the activation function in the form of:

$$T_0 [C_a^{2+}] = T_m [1 - \exp(-C^2)], \quad \text{with } C = C_B - C_S \left(\frac{\tau_w - \tau_w^h}{\tau_w^h} \right), \quad (16)$$

where T_m is the maximum tension generated by a contraction, C_B the basal value of the vasoconstrictor, and C_S allows the regulation of vasomotor activity by endothelial cells receiving shear stresses due to blood flow τ_w , compared to a homeostatic value τ_w^h .

The total stress will be the sum of passive, and active stress according to the expression:

$$\sigma = \sigma^p + \sigma_{act}, \quad (17)$$

σ^p is given by the relationship (5). σ_{act} is given by the relationship (15).

2.4. Parameters Determination

2.4.1. *Active Stress*. With regard to the active stress, we will use the parameters of [33] which are $T_m = 150$ kPa; $\lambda_m = 1.1$; $\lambda_0 = 0.4$; $C_b = 0.68$; $C_s = 20C_B$.

2.4.2. *Hyperelastic Model Parameters*. These parameters given by [21] are $\mu = 2.54$ kPa; $k_1 = 21.6$ kPa; $k_2 = 8.28$ [-]; and $\rho = 0.25$ [-]

2.4.3. *Lumped Parameters Model*. The constant lumped parameters model details is presented in Table 1.

3. Results and Discussion

3.1. *Study of the Influence of Atherosclerosis on the Arterial Wall Resistance*. Figures 3–5 show the influence of the growth of atherosclerosis on the resistance of the arterial wall. The results show that when the degree of stenosis increases, the stress on the wall also increases. These results are in agreement with literature [38]. This can be explained by the fact that the change in radius, leads to a significant change in wall resistance because the lumen become small and the platelet's activation are high [39]. In fact, when the lumen is gradually blocked, the lumen becomes small. This phenomenon has physiological consequences because it alters the transport of oxygen and other essential metabolites and may even trigger a pathological disorder. How is this possible?

The atheroma plaque, nourished by fats circulating in the blood, gradually develops on the wall of the artery while causing a narrowing (stenosis) hampering the passage of blood and the supply of oxygen to the organ that it irrigates [40]. It also constitutes a real clot trap [41]. When passing through the stenosis, the blood flow slows down, tending to deposit clotting elements (platelet and fibrin) there. This leads to the formation of stenosis, which decreases blood flow and compromises the oxygenation of the tissues supplied by the diseased artery. The installation of progressive stenosis can increase the wall stress [42] which will cause cerebral ischaemia or even haemorrhage.

This is why our result shows us an increase of stress as the stenosis increases. Why fixing the degree of longitudinal stenosis at 10% of the initial length, and we radially varied the height of the plaque (33.33%; 50%; and 83% reduction of the arterial lumen), this presents us the evolution of the stress.

3.2. *Comparative Study of the Deposition of Plaque*. The aim here was to assess the stress of the arterial wall as a function

TABLE 1: Constant lumped parameters model symbol, value, unit, description, and reference.

| Symbol | Value | Unit | Description | Reference |
|----------|--------|--------|---|--------------------|
| ρ_b | 1.05 | g/ml | Blood density | [34] |
| E_p | 1.6 | MPa | Elasticity modulus of PCA wall | [35] |
| P_{v0} | 14 | mmHg | Venous output pressure | [36] |
| P_{ic} | 10 | mmHg | Intracranial pressure | [35] |
| Q_{bl} | 53 | ml/min | Baseline flow | [37] |
| η_a | 47.8 | mmHg | Wall viscosity | [36] |
| R_p | 0.105 | cm | PCA inner radius | [34, 35] |
| L_p | 8.6 | cm | PCA length | Mulder et al. [35] |
| h_p | 0.026 | cm | PCA thickness | [35] |
| r_{a0} | 0.0075 | cm | Vessel inner radius in the condition of unstressed wall | [36] |
| h_{a0} | 0.0025 | cm | Wall thickness in the unstressed condition ra_0 | [36] |

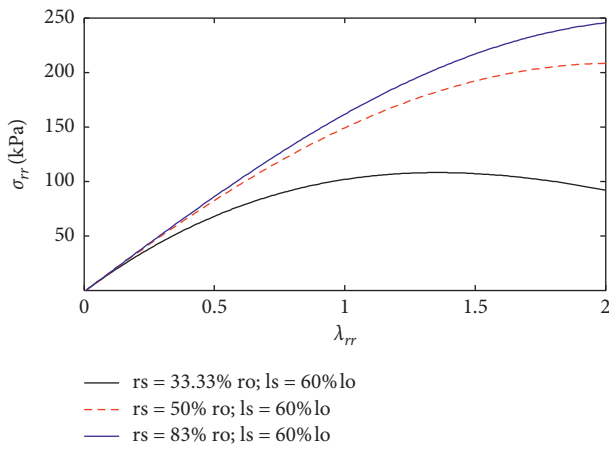


FIGURE 3: Study of the influence of stenosis on radial stress.

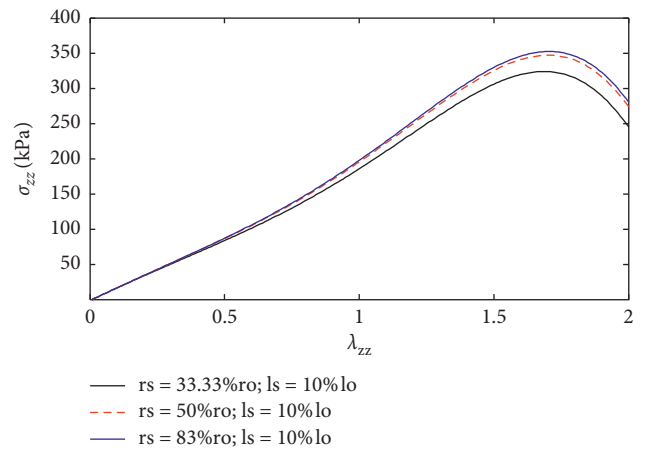


FIGURE 5: Study of the influence of stenosis on axial stress.

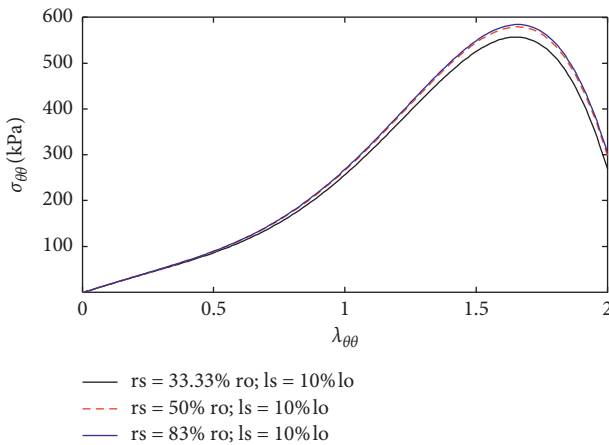


FIGURE 4: Study of the influence of the stenosis on the circumferential stress.

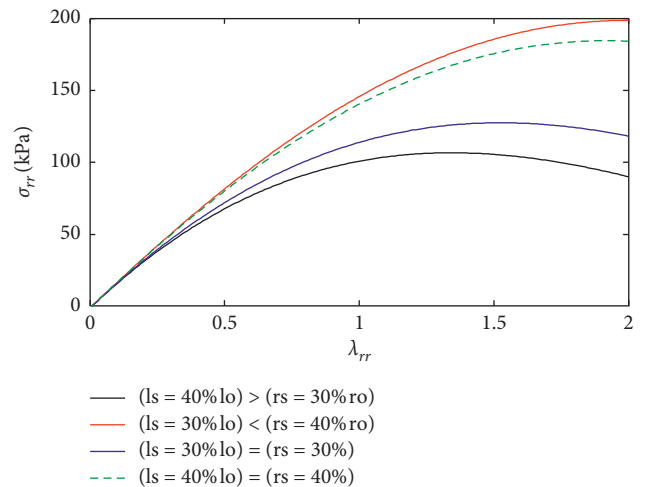


FIGURE 6: Comparative study of the deposition of plaque on radial stress.

of the percentage of longitudinal and radial reduction. Figures 6–8 present the situation. It is clear that when the percentage reduction in the longitudinal section of the stenosis is less than the percentage in the radial section, the stress is higher, and if not, the stress is less high. Then, when the latter is equal, the greater equality has the higher stress than the average equality. The detailed remarks of these

Figures 6–8 show us that the percentage of radial reduction is more dangerous than the percentage of longitudinal reduction, which is an agreement with the result of the literature [39].

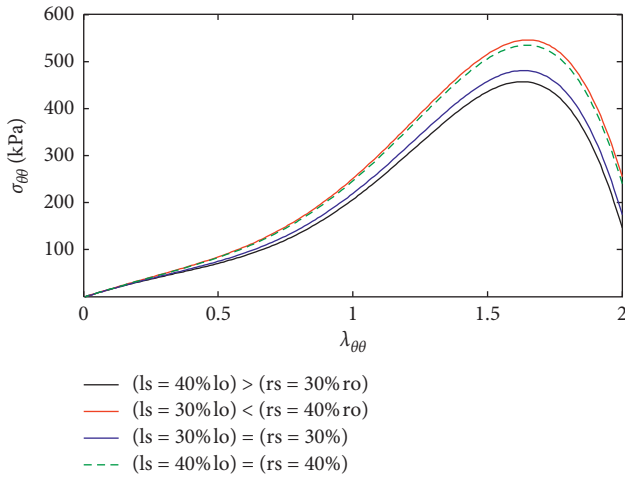


FIGURE 7: Comparative study of the deposition of plaque on the circumferential stress.

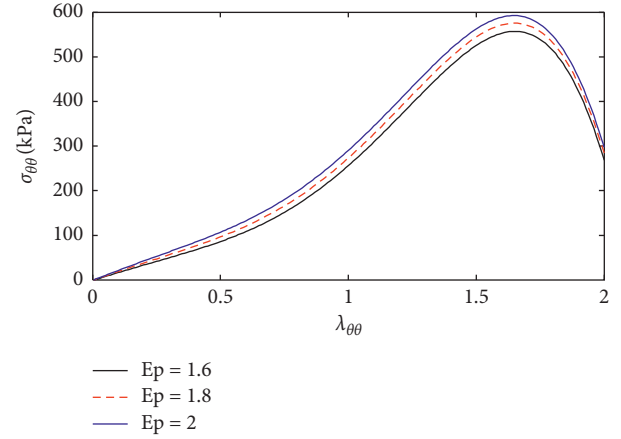


FIGURE 10: Study of the influence of the elasticity modulus on circumferential stress.

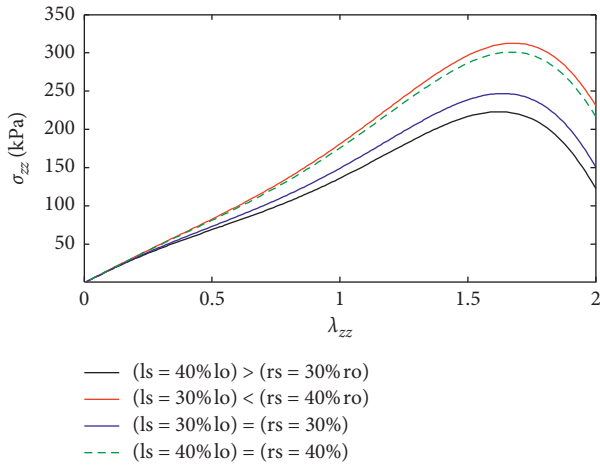


FIGURE 8: Comparative study of the deposition of plaque on the axial stress.

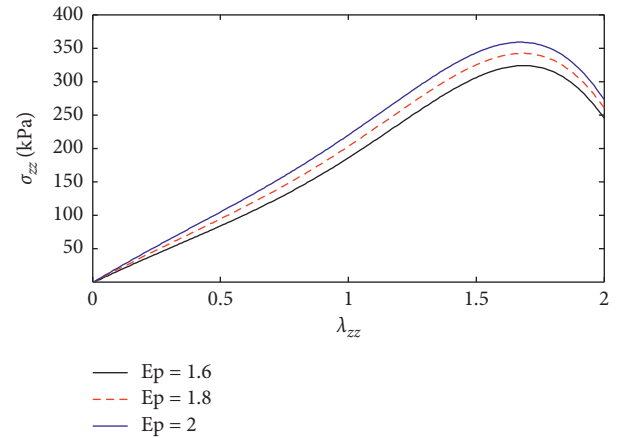


FIGURE 11: Study of the influence of the elasticity modulus on axial stress.

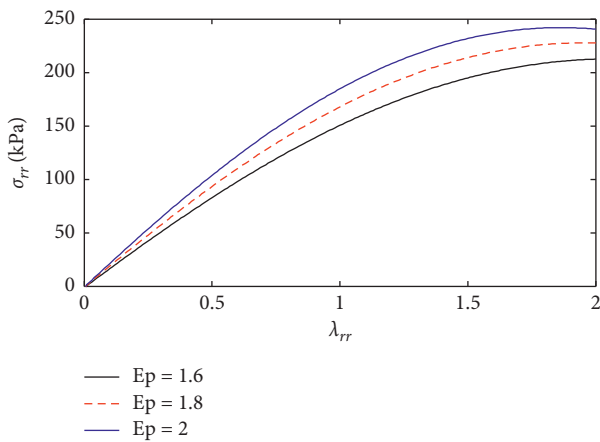


FIGURE 9: Study of the influence of the elasticity modulus on radial stress.

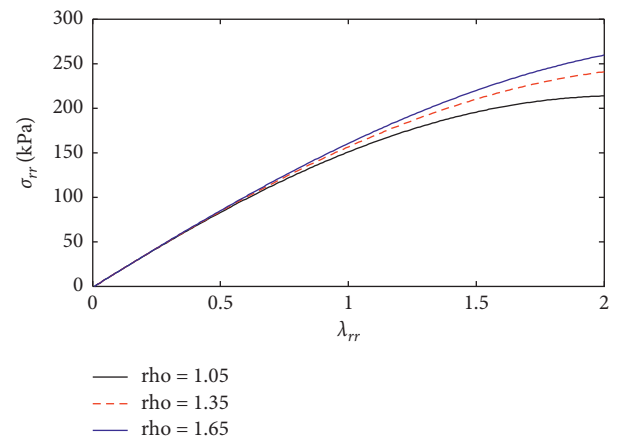


FIGURE 12: Influence of blood density on radial stress.

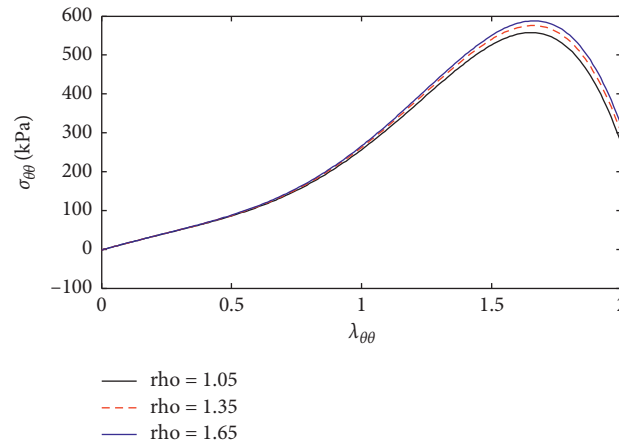


FIGURE 13: Influence of blood density on circumferential stress.

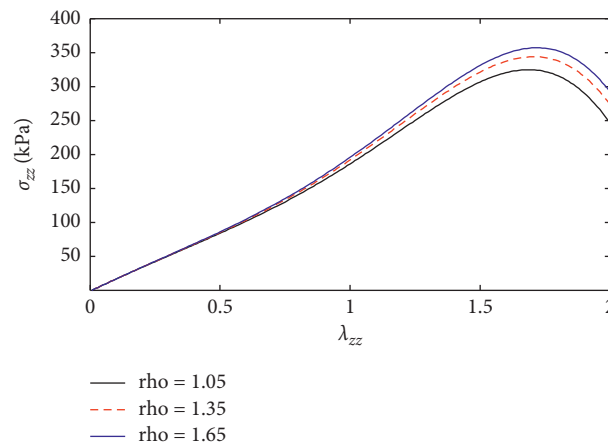


FIGURE 14: Influence of blood density on axial stress.

3.3. Study of the Influence of the Elasticity Modulus (EP).

Figures 9–11 show the influence of the elasticity modulus on the resistance of the arterial wall. It is obvious that when the elasticity modulus increases ($EP = 1.6; 1.8; 2$), the stress also increases. The rise of the latter (EP) leads to the loss of elasticity of the arterial wall [43]. This loss of elasticity is caused by arteriosclerosis. How is it possible?

Firstly, arteriosclerosis is characterized by a thickening, hardening, and loss of elasticity of the artery wall. It is a form of sclerosis that occurs in arteries. It is often defined as a natural, age-related phenomenon with normal thickening of the artery wall. However, many studies have also shown that this hardening of the wall can be accelerated by certain cardiovascular disorders. That is to say, the progressive deposition of lipids in the artery walls which causes the artery walls to thicken and harden [44]. In this case, one speaks more often of atherosclerosis in reference to atheroma, which refers to the plaque of fat formed. In addition, sclerosis of the arteries can be favoured by many factors such as genetic factors, metabolic disorders, poor eating habits, lack of physical activity, and certain stress factors. Finally, arteriosclerosis can remain asymptomatic for several years.

Nevertheless, in the most serious cases, it can block arteries that are essential to the proper functioning of the body, such as the coronary and carotid arteries. It leads to poor oxygenation and wall stiffness [45]. The prevention of arteriosclerosis consists in reducing the modulus of elasticity of the wall by limiting risk factors such as bad eating habits and sedentary lifestyle. To achieve, it is recommended to adopt a healthy and balanced diet by limiting the consumption of processed products and excess fat, sugar, and alcohol and by regular physical activity.

3.4. Study of the Influence of the Blood Density Mass.

Figures 12–14 present the influence of the density of the blood on the stress of the arterial wall. It is evident that an increase in the latter increases the stress on the arterial wall [46]. This is due to the fact that when the arterial lumen is reduced, the particle of blood (hematocrit...) is unable to move well, see Figure 15.

Thus, thrombosis joins the blood platelets and causes an increase in the density of the blood and the particle of blood is unable to move well [47]. For a longtime, this can cause blood clot and can lead to the destruction of the artery wall.

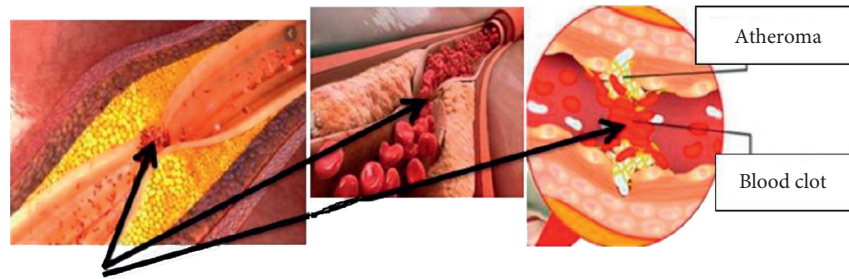


FIGURE 15: Blood density illustration (health on the net.com).

4. Conclusion

In this article, we studied the distribution of arterial stress, taking into account the properties of the hydrostatic and the evolution of the stenosis. We have coupled these two characteristics to the model of hyperelastic, anisotropic, and dynamic behavior of the human artery. This improvement of the model is useful for understanding the radial and longitudinal growth of plaque on arterial stress. The effects of the blood density mass, of the elasticity module, and of the atherosclerosis's growth have been deeply examined. The model allowed us to show that the radial growth of the plaque is very dangerous compared to the longitudinal growth. The plaque also promotes increased density and loss of elasticity. All this new knowledge will help us predict arterial damage and could also help the doctor and the surgeon to make a good clinical diagnosis, decisions, and good treatment planning.

Data Availability

The data used in this paper are from papers cited in the reference and in the text.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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