Journal of Materials Science and Engineering B 10 (3-4) (2020) 64-73
doi: 10.17265/2161-6221/2020.3-4.003

Study of the Osmosis Phenomenon to Predict the Stiffness of the Arterial Wall

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Abstract: In the present work we describe a new expression of total stress taking into account the passive and active contributions but especially the pore level stress. Special attention is paid to the effective stress and osmotic pressure gradient in numerical simulation to understand the mechanical behavior of the human arterial wall. The new model aims to predict the rigidity of the artery, by using the theoretical model of hyper-elastic, anisotropic and dynamical behavior of human common carotid artery. The principal obtained result showed that: the osmosis phenomenon is the best parameter to explain the loss water in arterial tissue. This loss of water causes the rigidity of the artery which thus can be controlled by the osmosis phenomenon. All the results are in good agreement with the expected results of the literature and could play the important role in the diagnosis of the patients with the CVD (Cardiovascular Disease).

Key words: Hyper-elastic, anisotropic, effective stress, osmosis phenomenon.

1. Introduction

CVD (Cardiovascular Disease) is the leading cause of death around the world [1] and is the leading cause of death in Europe and the most common cause of death in Portugal. It is estimated that by 2030, ischemic heart disease will be the third leading cause of death worldwide [2]. Despite recent decreases in mortality rates in many countries, CVD is still responsible for almost half of all deaths in Europe [3], constituting a major public health challenge in Western Europe [4]. However many of the CVD events could be prevented if we focused on the modification of main risk factors [5]. The observed reduction in CVD mortality in the last decades, albeit at a low rate, is the result of a set of changes, mainly by efforts made by most European countries in disease prevention, tacking main risk factors, as well as to the improvements made in disease management and treatment [6]. That is why many scientists were motivated to study the arterial behavior to prevent the later as the majority of these models rely on experimental *in vitro* data for obvious reasons of accessibility. The results obtained from these *in vitro* data do not always show their relevance, whether for understanding the normal *in vivo* physiology of the artery, its growth, its reorganization or for improving clinical diagnoses [7]. The confrontation of theoretical or numerical models with *in vivo* data therefore appears essential, if we want to help better understand the biological mechanisms linked to arterial pathologies and help with diagnosis or clinical therapy. One of the current problems is the identification of parameters of these theoretical models, in order to best reproduce the mechanical behavior of arteries *in vivo* and thus access an evaluation of satisfactory wall stress distributions [8]. Ref. [9] proposed a two-layer model to describe the nonlinear properties of carotid arteries. The materials are described by a polynomial form of the strain energy function, with one parameter

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for the isotropic inner layer and three parameters for the anisotropic outer layer. Ref. [10] also described the incompressible 2D mechanical behavior of the media and the adventitia of bovine carotids without taking into account the residual constraints. Ref. [11] has shown that the deformation energy functions of the exponential form better describe the incompressible passive behavior of carotid arteries than the polynomial forms, taking into account the residual stress and the incompressibility hypothesis. Ref. [12] proposed their model from 2D biaxial tests performed on thoracic aortas of dogs. They introduced an incompressible two-layer model. Ref. [13] proposed an incompressible and orthotropic strain energy function, which combines the polynomial and exponential forms to study abdominal aortas of rats. Ref. [14] introduced a 1D contribution to the activation of smooth muscle, which is added to the state of constraints describing the passive mechanical behavior of artery. They also used the strain energy function proposed by Ref. [11], in order to consider the nonlinear, incompressible and orthotropic behavior of the artery. Ref. [15] has shown the limited effect of dynamic wall movement and the significant effect of contractility on the increase in circumferential stresses, while suggesting the existence of multi-axial rather than uniaxial contractile forces. Ref. [16] has taken into account a certain dispersion radial distribution of fibers in the arterial wall, and proposed a new model. The new model was used by these same authors when studying the physiological stresses experienced by different arterial segments, including residual stresses [17]. This model was also used to simulate bypass surgery or by Ref. [18] to model the behavior of healthy arteries with the consideration of residual stresses, which they have extended to the behavior of carotid bifurcations. In general, most of these models of arterial mechanical behavior do not take into account the effective stress and the osmosis phenomenon. In this present study we use the theoretical model of hyper-elastic, anisotropic and dynamic mechanical behavior of human common carotid artery, taking into account the effective stress and the osmosis phenomenon to predict the rigidity of artery wall. Our results showed that the effective stress on the pore of arterial wall, due to the deposit of plaque that is close to the arterial pore, limited the flow passage of solute and nutriment. This leads to the increasing of shear modulus, consequently increasing the total stress.

The paper is organized as follows. In section 2, we give the formulations of the different contributions which constitute the theoretical model. Especially, we describe our pore level stress tensor defined by an effective stress and osmotic pressure gradient. In Section 3, we present numerical simulations and discussions. We draw the conclusion in Section 4.

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

The proper functioning of the arterial wall is vital to the health of the individual, as evidenced by the numerous debilitating medical problems which can arise when the wall falls prey to effects of disease. Inversely, the arterial wall's striking capabilities for growth; repair and continual remodeling under diverse physiological loads have yet to be reproduced in tissue engineered vessels, though rapid advances are being made [19].

The mechanical integrity of the arterial wall is dependent on its passive and active loads bearing, the osmotic pressure and an initial shear modulus. For the theoretical study of mechanical behavior, the arterial structure is represented by a cylindrical tube subjected to osmotic pressures, blood pressure and loading of certain plaque.

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

transformation **F** [20].

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

where $\lambda_r, \lambda_\theta, \lambda_z$ 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^tF and B = FF^t and F^t represents the tensor transposes F.

2.2 Anisotropic Behavioural Relationship

The arterial wall is mainly composed of water, it is generally considered by an incompressible medium [16], and the fluid movement inside the porous tissue induces stress gradients. This hypothesis of wall incompressibility has been demonstrated by experimental studies. Ref. [21] has shown that artery samples deform in isochoric ways without action of a large number of different loads.

In this work we have exploited the deformation energy of Ref. [22]. This energy takes into account the radial dispersion of fiber distribution in the arterial wall. The expression of this energy is given by:

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

In which ρ ∈ [0, 1] is a dispersion factor that regulates the degree of anisotropy. In an original way, if ρ = 0 we obtain the composition of a neo-hookeen model with Ref. [12] model and if ρ = 1 we obtain Ref. [22] model. Ref. [18] model has been used by several authors to study physiological stress by different arterial segments. 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 [23].

2.3.1 Passive Contribution

The Cauchy tensor is given by:

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

In which

$$I_1 = \lambda_r^2 + \lambda_\theta^2 + \lambda_z^2;\ I_4 = \lambda_\theta^2\cos(\varphi) + \lambda_z^2\sin(\varphi) \quad (4)$$

For an incompressible material J = detF the work of Ref. [21] has shown that the arterial wall is incompressible and the Cauchy stress becomes $\boldsymbol{\sigma}$ = -P I+ $\frac{2}{J}\boldsymbol{F}\frac{\partial \boldsymbol{W}(\boldsymbol{I_1}, \boldsymbol{I_4})}{\partial \boldsymbol{c}}\boldsymbol{F^t}$ where P is the Lagrange multiplier and represents a hydrostatic stress contribution. Let us now write this expression in the form of Ref. [24]:

$$\sigma = -pI + 2W_1B + 2W_2(I_1 1 - B)\,\mathrm{B} + 2W_4 \mathrm{a}\otimes\mathrm{a} + 2W_5(\mathrm{a}\otimes\mathrm{Ba} + \mathrm{Ba}\otimes\mathrm{a}) \quad (5)$$

where $\otimes$ represents the tensor product.

With $\boldsymbol{W_j} = \frac{\partial \boldsymbol{W}}{\partial \boldsymbol{I_j}}$ (j = 1, 2, 3, 4 and 5) and p is a Lagrange multiplier related to the condition of incompressibility. 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 [25]. Other mathematical criteria must normally be met, such as convexity and elliptic.

2.3.2 Active Stress

The first active stress models were developed in the context of small deformation [26] and then extended in the context of large deformation [27-29]. Thus the mode of Ref. [15] allows them to analyze the dynamic behavior of the arterial wall. This model has for expression:

$$\sigma_{act} = \lambda_i T_0[C_a^{2+}](1 - (\frac{\lambda_m - \lambda_i}{\lambda_m - \lambda_0})^2)(e_i \otimes e_i) \quad (6)$$

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, this model was modified [30] by taking the activation function in the form of :

$$T_0[C_a^{2+}] = T_m[1 - \exp(-C^2)] \text{ with } C = C_B - C_S(\frac{\tau_w - \tau_w^h}{\tau_w^h}) \quad (7)$$

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.

2.4 Pore Level Stress

To model osmotic behavior at the level of different collagen fibers, Ref. [31] proposed a tissue constitutive model describing the photomechanical interaction between a hyper-elastic porous matrix saturated with intra-fibril fluid, a swelling stress simulating the osmotic effects of Donnan and viscoelastic collagen fibers. The stress tensor defined at the pore level [32].

$$\sigma_1=\sigma_{eff} - PI \quad (8)$$

I is the identity tensor and *P* pore pressure, where *P* is the sum of the chemical potential of the water V_w and osmotic pressure *DP*.

$$P = V_w + DP \quad (9)$$

2.4.1 Effective Stress

The response of the macroscopic effective stress of the solid matrix was controlled by an initial shear modulus G_m and an initial solid fraction $n_{s,0}$ and by a continuous homo-prorogated deformation. A modified neo-hookian model was used to describe the behavior of material deformation [32]:

$$\sigma_{eff}= -\frac{1}{6}\frac{Ln(J)}{J}G_m I[-1+\frac{3(J+n_{s,0})}{-J+n_{s.0}}+\frac{3JLn(J)n_{s,0}}{-J+n_{s.0}}]+\frac{G_m}{J}(B-J^{\frac{2}{3}}I) \quad (10)$$

Or *J* is the determining factor of the deformation gradient; *I* is the invariant tensor and *B* is the green Cauchy tensor. For an incompressible material **J** = 1, the expression becomes:

$$\sigma_{eff}= G_m(\text{B-I}) \quad (11)$$

2.4.2 Osmotic Pressure Gradient (DP)

Applying Darcy's law as the osmotic pressure gradient is given by the relationship of Ref. [32]:

$$DP=Q_i RT\left(\sqrt{C_{fe}^2+4(\frac{\gamma_e}{\gamma_i})^2}C_e^2\right)-2Q_e RTC_e \quad (12)$$

where Q_i, Q_e are the internal and external osmotic coefficients respectively.

γ_i , γ_e are the internal and external activity coefficients respectively.

C_e: the concentration of salt in arterial tissue.

C_{fe}: the fixed charge concentration.

Proteoglycan which depends on extra-fibril water (n_e) and normal fixed charge density per milliliter of total fluid C_f can be expressed by:

$$C_{fe}=\frac{n_f}{n_e}C_f \text{ whit } n_e=n_f - \varphi_{ci}\rho_{c,tot} \quad (13)$$

n_f: the total fraction of water.

φ_{ci}: a parameter that defines the intra-fibril of water not collagen mass.

$\rho_{c,tot}$: the collagen tensor in relation to the weight of total moisture.

2.5 Total System Stress

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

$$\sigma=\sigma^p + \sigma_{act} + \sigma_{eff} - DP \quad (14)$$

In which:

σ^p is given by Eq. (5);

σ_{act} is given by Eq. (6);

σ_{eff} is given by Eq. (10);

DP is given by Eq. (12).

3. Parameter Determinations

3.1 Active Stress

With regard to the active stress, we will use the parameters of Ref. [30] which are: $T_m = 150$ kPa; $\lambda_m = 1.1$; $\lambda_0 = 0.4$; $C_b = 0.68$; $C_s = 20C_B$.

3.2 Effective Stress

The parameters of the tissue model given in Refs. [33, 34] were grouped and used in this study. They were obtained from experimental protocols that we briefly describe here for the information of the reader. To determine the water content, they first determined the weight (*ww*) of the tissue sample and then the

sample is lyophilized and Persian again to determine the dry weight (*Dw*) and the initial total water content (*FF*) from Eq. (13) are calculated and determined according to:

$$FF = \frac{ww-Dw}{ww} \quad (15)$$

$$n_f = \frac{FF-1+J}{j} \quad (16)$$

in our case J = 1, Eq. (16) becomes n_f = FF-1.

To calculate C_f they used the expression [35] which is given by: $C_{f,0}=\frac{\tau_{cs}C_{cs}}{Mw_{cs}}$ and $C_f = C_{f,0}\frac{n_{F,0}}{n_{F,0}-1+J}$ in the incompressible environment, this expression becomes $C_f = C_{f,0}\frac{n_{F,0}}{n_{F,0}}$ or J = 1 where $\tau_{cs}, Mw_{cs}\ and\ C_{cs}$ are valency (2 meq/mmol), molecular weight (51,000 µg/mmol) and $n_{F,0}$ the initial water content.

3.3 Hyperelastic Model Parameter

These parameters given by Ref. [17] are μ = 2.54 kPa; k_1 = 21.6 kPa; k_2 = 8.28 [-]; ρ = 0.25 [-] ([-] means no unity).

4. Results and Discussions

4.1 Validations of the Results

It was a question of using the phenomenon of osmosis to study the rigidity of the arterial wall. To arrive there, we will first make the comparison of the proposed model with that of the literature see Fig. 1. Indeed many authors [18, 36] have studied the behavior of the arterial wall taking into account only the passive and active stress, yet the wall has pores that can become clogged and result in the effective stress, thus influencing the osmotic pressure. So we took into account these two previous phenomena. The results show us that:

In a first step when we cancel our previous contribution we obtain the model use by Ref. [18] when it studied the constraint of the wall with an elongation inferior to 1.5, and the result of Refs. [17, 36, 37] for an elongation of between [1; 1.12], [0; 0.6] and [1; 2] respectively.

In the second stage, when the effective stress is equal to the osmotic pressure, we always find the previous results, and the equality of this phenomenon simply cancels our contribution.

Thirdly, when the effective stress is greater than the osmotic pressure, an increase in the total stress of the wall is observed to that of the literature. This can mean that when the pores are blocky it impedes an effective stress which increases the total stress of the wall.

Lastly, when the effective stress is lower than the osmotic pressure, the total stress of the arterial wall decreases compared to that of the literature.

4.2 The Effect of Shear Modulus (G_m)

The shear modulus G_m help us to control effective stress. As we see in Eq. (11), the effective stress σ_{eff} is strongly depend on G_m. Fig. 2 shows us that, when the G_m increases, the total stress increases. This makes us understand according to Fig. 1 that for the small value of G_m we have normal osmosis phenomenon. When the latter is important, we stated the abnormal osmosis phenomenon. That is why in Fig. 1, we see that when σ_{eff} > DP the total stress is higher than when σ_{eff} < DP.

4.3 Osmostic Pressure as a Function of Salt Concentration

Fig. 3 shows us that osmotic pressure decreases as salt concentration increases. This is due to the fact that cellular functions are disrupted. Cells during degeneration produce more agene of degradation of macromolecules than synthetic macromolecule as in Ref. [38]; their disappearance is accompanied by a change in arterial property. The number of arterial tissues decreases as in Ref. [39], so with the development of degeneration, the arterial wall loses its mechanical characteristics and the loss of proteoglycans induces a decrease in osmotic pressure and subsequently dehydration of the arterial wall [39].

The permeability of the tissues decreases with drying.

4.4 Relationship between Osmotic Pressure and Arterial Stress

Fig. 4 shows us that when osmotic pressure is low, the stress within the wall increases. This is due to the fact that the increase in salt reduces osmotic pressure. Since sodium is the main cause of osmotic pressure decrease [40], exposure to a high concentration of salt on the arterial wall accelerates arterial degeneration and also leads to stiffness of the arterial wall [41]. This also causes an increase in blood pressure. This is why clinical studies recommend consuming little salt [42-44].

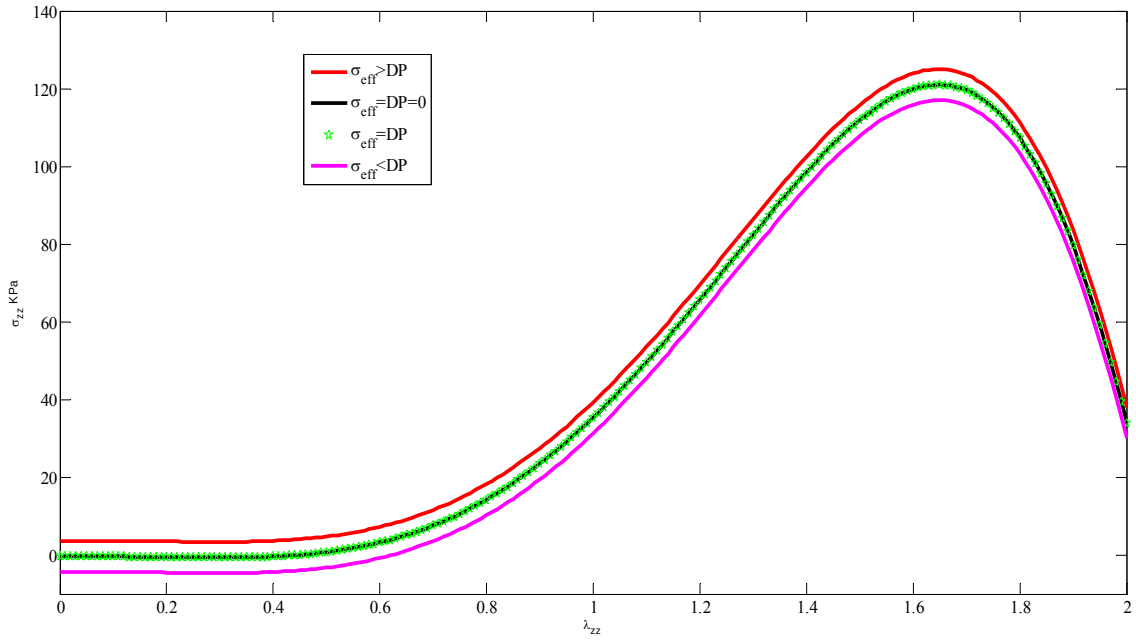

Fig. 1 Influence of simultaneous variation of effective stress and osmotic pressure on total stress.

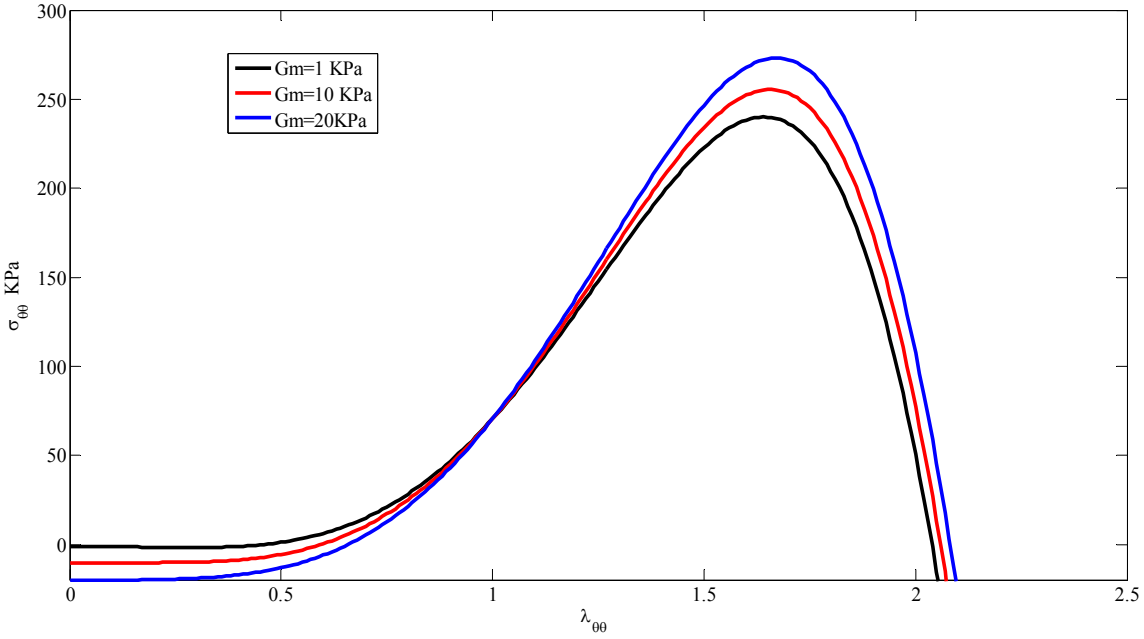

Fig. 2 Effect of shear modulus in circumferential stress.

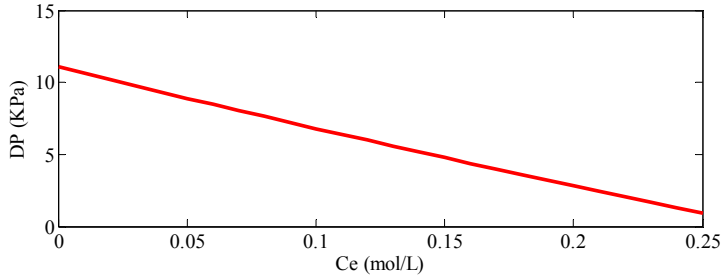

Fig. 3 Osmotic pressure as a function of salt concentration.

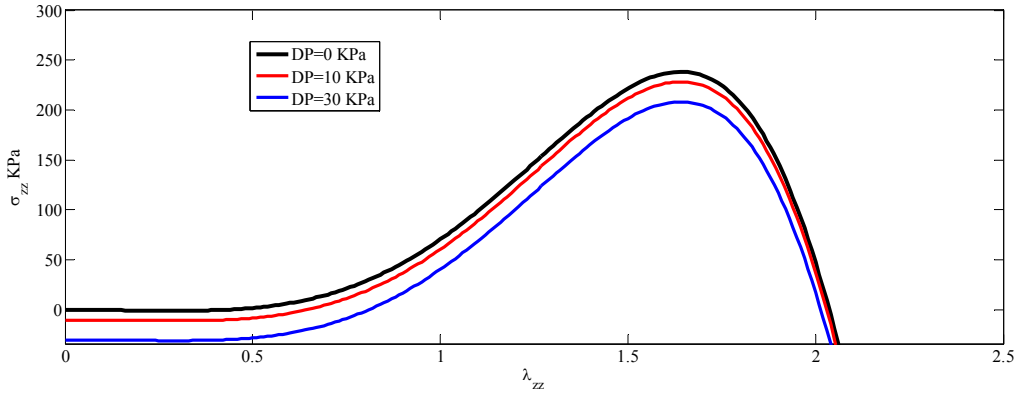

Fig. 4 Relationship between osmotic pressure and arterial stress.

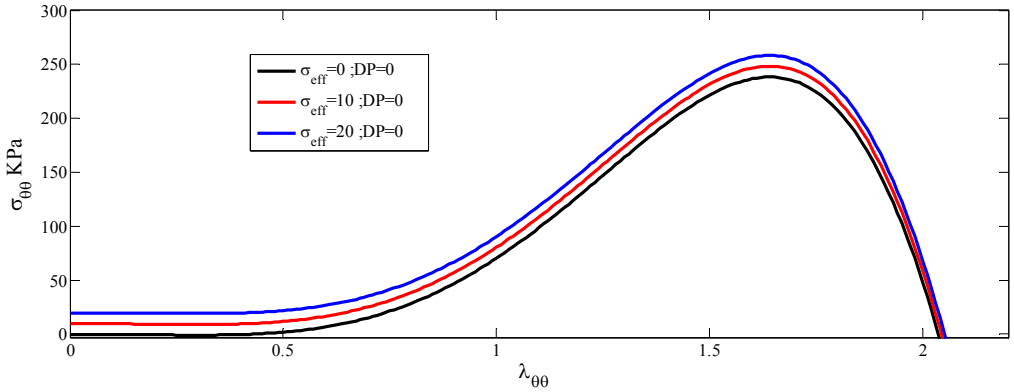

Fig. 5 Stress as function of elongation.

4.5 Effect of Effective Stress

This is the stress that occurs in the area surrounding the pores. Fig. 5 shows us that the increase in effective stress increases the stress on the arterial wall. This is due to the fact that the porosity is blocked by lipid deposits or by the large solute. It means that when the pores are blocked there is no more exchange between the external and internal environment, so it can lead to arterial insufficiency and increased risk of deformation as in Ref. [43]. This helps us to understand some pathology, since this increased stress promotes the degradation of collagen fiber properties and the latter starts to decrease [44]. Then when the porous are blocked this explains there is no longer a good functioning of water exchange and solute, because the arterial boundary is a separator between two media with different composition and electrochemical properties: the porous intracellular medium saturates by fluid phase and the extracellular medium containing the interstitial fluid which contains several

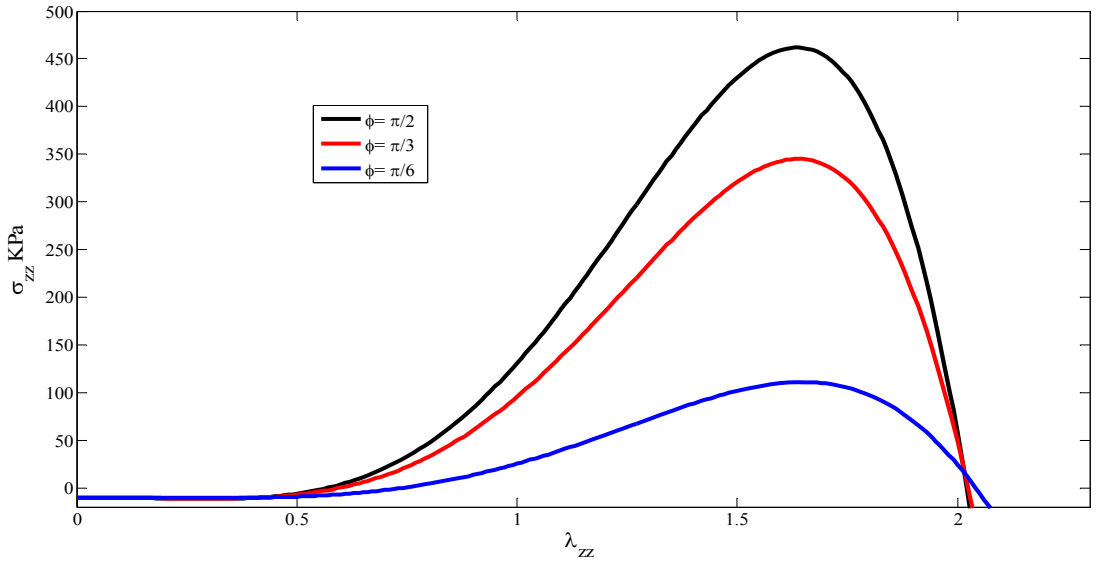

Fig. 6 Stress as function of angle of fibers.

ionic solute (N_a^+; Cl^-; C_a^{2+}; SO_4^{2-}) which can penetrate into the arterial wall so that the tendency of the intracellular medium is electrically neutral.

4.6 Influence of Fiber Dispersion

Fig. 6 shows that the increase in angles has no influence on the linear domain [0, 0.5]. In addition to the latter, which is the nonlinear domain, we observe a very great influence. So when the angle between the fiber increases and elongation also increases, the stress increases until it breaks. This result is in agreement with Ref. [17].

5. Conclusion

The pore level osmotic model associated to the hyperelastic model was presented to evaluate the rigidity of the arterial wall using osmotic pressure parameters and initial shear modulus. This study allowed us to understand that salt induces small degenerative changes in ECM (extracellular matrix) and can produce significant alterations in solutes. They also made it possible to identify the mechanism linked to know alterations in collagen fiber. Our results suggest in particular that the modifications of the tissue are capable of causing the dehydration of the collagen fiber.

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