

Modeling of damage evolution in a patient-specific stenosed artery upon stent deployment

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Abstract

Computational models provide a powerful tool for pre-clinical assessment of medical devices and early evaluation of potential risks to the patient in terms of plaque fragmentation and in-stent restenosis (ISR). Using a suitable constitutive model for arterial tissue is key for the development of a reliable computational model. Although some inelastic phenomena such as stress softening and permanent deformation likely occur due to the supra-physiological loading of arterial tissue during the stenting procedure, hyperelastic constitutive models have been employed in most of the previously developed computational models. This study presents a finite element model for stent deployment into a patient-specific stenosed artery while inelastic arterial behaviors due to supra-physiological loading of the tissue have been considered. Specifically, the maximum stress in the plaque and the arterial layers which is the main cause of plaque fracture during stent deployment and the surgically-induced injury (damage) in the arterial wall, as the main cause of ISR, are presented. The results are compared with the commonly-used hyperelastic behavior for arterial layers. Furthermore, the effects of arterial material parameter variation, analogues to different patients, are investigated. A higher amount of damage is predicted for the artery which shows a higher stress in a specific strain.

Keywords: Stent, artery, inelastic constitutive model, damage, patient-specific, finite element.

1. Introduction

Atherosclerosis is one of the most common types of cardiovascular disease in which fibrous and fatty materials, called plaque, build up inside the artery and cause a partial or total occlusion of the artery. One way to restore physiological blood flow to an occluded artery involves the deformation of plaque using intravascular balloon angioplasty with or without stenting. This clinical intervention is accompanied with supra-physiological loading of the arterial wall causing tissue damage (injury) (see (Holzapfel and Gasser, 2007) for more details), and that may lead to tissue in-growth and a re-blockage, termed in-stent restenosis (ISR) (Boyle and Prendergast, 2011; Fereidoonnezhad *et al.*, 2017).

Computational modeling of the stenting procedure provides a powerful tool for an early evaluation of the potential risk of a specific stent for the patient in terms of (i) vessel wall damage, as the main cause of ISR, and (ii) plaque fragmentation which can cause a downstream occlusion of tiny blood vessels.

Although the literature concerning the modeling of the stent deployment into a stenosed artery is rich in terms of stent design and deformation (Gu *et al.*, 2012; Wang *et al.*, 2012; Liu *et al.*, 2020), material of the stent and its constitutive model (Liu *et al.*, 2019), the majority of the studies employed a rather simple cylindrical shape for the artery (Rivlin and A, 2006; Cui *et al.*, 2010, Kai *et al.*, 2020). Some studies focused

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on more realistic geometries of the stenosed artery (Holzapfel, 2005; Voß *et al.*, 2019) and investigated the effects of stent insertion or blood flow on the artery. However, these studies employed hyperelastic constitutive models for the arterial tissues which are not enough for supra-physiological loading that occurs during stent deployment (Gu, Zhao and Froemming, 2012; Mozafari *et al.*, 2018; Ruan *et al.*, 2018). In supra-physiological loading inelastic phenomena such as stress softening and permanent deformation may occur in arterial tissues (Maher *et al.*, 2012; Fereidoon nezhad, Naghdabadi and Holzapfel, 2016). Although some approaches aim to model the inelastic phenomena able to investigate the injury in arterial tissues (Holzapfel and Fereidoon nezhad, 2017), several inelastic models are still not considered in the computational modeling of stent deployment in a patient-specific lesion morphology. It is then critical that the constitutive models for arterial tissue account for these inelastic phenomena so that computational models can properly predict the outcome of the stenting procedure.

Therefore, the goal of the current study is to incorporate the aforementioned inelastic phenomena into the computational modeling of the stenting procedure in a patient specific-artery, which have not been considered in previous research studies. To accomplish that, we develop a finite element (FE) model for the expansion of a stent into a patient-specific stenotic artery by accounting for the damage-induced inelasticity in the tissues based on the constitutive model proposed in *et al.*, 2016) and compared the results of the inelastic model to the commonly used hyperelastic model.

It is noted that the word 'damage' in this work points to the changes in the microstructure of the tissue such as breaking of the bonds between collagen fibers which consequently results in some inelastic phenomena such as stress softening and permanent deformation in the tissue scale.

The stress distribution in the plaque and the arterial tissue, which may be used for the assessment of plaque rupture risk in future studies, and the induced damage (injury) in the arterial wall, as the main cause of ISR, are presented. Moreover, to investigate the effect of the stent design on the induced injury in the arterial wall we perform the simulations for two different stents geometry. We also examine the effect of arterial properties variation on the final outcome of the procedure.

The paper is structured as follow: In Section 2 the geometry and mechanical behavior of the stent and the artery are introduced, and the simulation of the process of the analysis, i.e., boundary conditions, meshing and loading are described in detail. In Section 3 the related results are presented, and the effects of different parameters on the results are discussed. Finally, we draw some conclusions in Section 4.

2. Materials and methods

A finite element simulation of the stent expansion into a (patient-specific) stenotic artery requires the geometry and material properties of the stent and artery, the stent-artery interaction, the boundary and loading conditions that are described in the following.

2.1. Material models

Proper constitutive models for the plaque materials and the arterial tissues are required for a reliable computational model of the stenting procedure. In this section, the constitutive models, employed in computational modelling of the procedure, are discussed.

2.1.1. Artery and plaque

In this study we use two different constitutive models for the arterial layers and the results are compared. The first model is the hyperelastic model (Gasser, Ogden and Holzapfel, 2006), the so-called HGO model, which extends the constitutive framework for arterial walls (Holzapfel, Gasser and Ogden 2000), by considering distributed collagen fiber orientations. The second model is the inelastic model

(Fereidoonhezad, Naghdabadi and Holzapfel, 2016), which considers the stress softening and permanent deformation in arterial tissues. The latter is also able to evaluate the damage in arterial tissue after stent deployment. It is also noted that in all simulations the plaque are assumed to be a hyperelastic material. Both models are briefly reviewed in the following.

The HGO model

The anisotropic hyperelastic constitutive model (Gasser, Ogden and Holzapfel, 2006) was initially developed to describe the elastic properties of arterial tissue, but now is used extensively for modeling a variety of soft fibrous tissues, and it has been implemented in several commercially available FE programs to simulate soft tissue elasticity. In the following a brief review of this model is provided. The kinematics is described in terms of the deformation gradient $\mathbf{F}$. Hyperelastic constitutive models often split the local deformation into volumetric and isochoric parts. Accordingly a multiplicative decomposition of the deformation gradient $\mathbf{F} = J^{\frac{1}{3}}\bar{\mathbf{F}}$ is partly considered, where $J = \det \mathbf{F}$ is the volume ratio, and $\bar{\mathbf{F}}$ is the modified deformation gradient with $\det \bar{\mathbf{F}} = 1$. Consequently, $\mathbf{C} = \mathbf{F}^T \mathbf{F}$ denotes the right Cauchy-Green tensor, and $\bar{\mathbf{C}} = \bar{\mathbf{F}}^T \bar{\mathbf{F}}$ is the modified right Cauchy- Green tensor. Arterial tissues can be regarded as materials that consist of an isotropic matrix material within which (at least) two families of fibers are embedded (Schriefl *et al.*, 2012). Here, the arterial layers in the initial configuration are characterized by two collagen fiber families, symmetrically distributed by an angle θ with respect to the circumferential direction of the artery. Thus, the arterial layers respond orthotropically, and the families of collagen fibers are characterized by the two direction vectors $\mathbf{a}_{0i}, i = 4, 6$ with $|\mathbf{a}_{0i}| = 1$. The following invariants, required for the model, can then be defined.

$$I_1 = \text{tr} \mathbf{C}, I_4 = \mathbf{a}_{04} \cdot \mathbf{C} \mathbf{a}_{04}, I_6 = \mathbf{a}_{06} \cdot \mathbf{C} \mathbf{a}_{06} \quad (1)$$

$$\bar{I}_1 = J^{-2/3} I_1, \bar{I}_4 = J^{-2/3} I_4, \bar{I}_6 = J^{-2/3} I_6 \quad (2)$$

The strain-energy function Ψ is then given in this form

$$\Psi(\mathbf{C}, \mathbf{a}_{04}, \mathbf{a}_{06}) = \Psi_{\text{vol}}(J) + \bar{\Psi}_{\text{iso}}(\bar{\mathbf{C}}) + \Psi_f(\mathbf{C}, \mathbf{a}_{04}, \mathbf{a}_{06}) \quad (3)$$

Where Ψ_{vol} is the volumetric, $\bar{\Psi}_{\text{iso}}$ the isochoric, isotropic and Ψ_f the fiber contribution. Recent investigations demonstrate that using deviatoric invariants for fiber-reinforced materials may lead to some non-physical results when assuming some degree of compressibility for tissue (Nolan *et al.*, 2014; Gültekin, Dal and Holzapfel, 2018). To avoid these non-physical results, henceforth, we use the right Cauchy- Green tensor $\mathbf{C}$ (instead of $\bar{\mathbf{C}}$) in the last term of eq. (3), as suggested in (Nolan *et al.*, 2014). Thus, the HGO strain-energy function is reformulated as

$$\Psi(\mathbf{C}, \mathbf{a}_{04}, \mathbf{a}_{06}) = \Psi_{\text{vol}}(J) + \bar{\Psi}_{\text{iso}}(\bar{I}_1) + \sum_{i=4,6} \Psi_{f,i}(I_i, I_i) \quad (4)$$

where

$$\Psi_{\text{vol}}(J) = \frac{K}{2} \left(\frac{J^2 - 1}{2} - \ln J \right), \bar{\Psi}_{\text{iso}}(\bar{I}_1) = \mu(\bar{I}_1 - 3) \quad (5)$$

and

$$\bar{\Psi}_{f,i}^0(\bar{I}_1, \bar{I}_i) = \frac{k_1}{2k_2} \left[\exp(k_2 \bar{E}_i^2) - 1 \right], \bar{E}_i = \kappa \bar{I}_1 + (1 - 3\kappa) \bar{I}_i - 1 \quad (6)$$

in which K is the initial bulk modulus; μ , k_1 , and k_2 are material parameters, and $\kappa \in [0, 1/3]$ denotes the dispersion parameter. We enforced the material incompressibility by using a high value of K/μ . For further details on the material compressibility the reader is refer to the recent work by Moerman *et al.*, (2020).

The inelastic models

Fereidoon nezhad et al. (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016) proposed a constitutive model which accounts for the damage-induced inelastic phenomena, the stress softening and permanent deformation, in arterial tissue under supra-physiological loading conditions which occur during balloon angioplasty. The strain-energy function Ψ is presented as (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016)

$$\Psi(\mathbf{C}, \mathbf{a}_{04}, \mathbf{a}_{06}, \eta_{s,4}, \eta_{s,6}, \eta_{in}) = \Psi_{vol}(J) + \tilde{\Psi} \quad (7)$$

$$\tilde{\Psi} = \bar{\Psi}_{iso}(\bar{I}_1) + \sum_{i=4,6} \left[\eta_{s,i} \Psi_{f,i}(I_1, I_i) + \phi_i(\eta_{s,i}) \right] - \left[(1 - \eta_{in}) N(\mathbf{C}^*) + \phi_{in}(\eta_{in}) \right] \quad (8)$$

where $\eta_{s,i}$, $i = 4, 6$ and η_{in} are damage variables introduced to capture the stress softening and permanent deformation, respectively. The functions ϕ_i and ϕ_{in} are dissipation functions and describe the inelastic energy dissipation. Moreover, the inelastic energy N is presented as (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016)

$$N(\mathbf{C}^*) = \mu^* (\bar{I}_1^* - 3) + \frac{k_1^*}{2k_2^*} \sum_{i=4,6} \left[\exp(k_2^* \bar{E}_i^{*2}) - 1 \right] \quad (9)$$

$$\bar{E}_i^* = \kappa^* \bar{I}_1^* + (1 - 3\kappa^*) \bar{I}_i^* - 1 \quad (10)$$

Where μ^* , k_1^* , k_2^* , and κ^* are material parameters describing the anisotropic damage in collagen fibers. The damage variables are defined as (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016)

$$\eta_{s,i} = 1 - \frac{1}{r_1} \operatorname{erf} \left[\frac{1}{m_1} (\bar{\Psi}_{f,i}^{\max} - \bar{\Psi}_{f,i}^0) \right] \quad (11)$$

$$\eta_{in} = \tanh \left[\left(\frac{\bar{\Psi}^0}{\bar{\Psi}^{\max}} \right)^{m_2} \right] / \tanh(1) \quad (12)$$

in which r_1 , m_1 and m_2 are material parameters. $\bar{\Psi}_{f,i}^{\max}$ and $\bar{\Psi}^{\max}$ are maximum strain energy of the fiber family i and tissue, respectively, which obtained by the deformation history while $\bar{I}_1^* = \operatorname{tr} \bar{\mathbf{C}}^*$, $\bar{I}_4^* = \mathbf{a}_{04} \cdot \bar{\mathbf{C}}^* \mathbf{a}_{04}$, and $\bar{I}_6^* = \mathbf{a}_{06} \cdot \bar{\mathbf{C}}^* \mathbf{a}_{06}$. Here $\mathbf{C}^*$ is a function of the modified right Cauchy-Green tensor at the peak deformation of the loading history. The damage induced in each fiber family can be characterized by the minimum value $\eta_{s,i}^{\min}$ of the damage variable according to (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016)

$$\eta_{s,i}^{\min} = 1 - \frac{1}{r_1} \operatorname{erf} \left[\frac{1}{m_1} (\bar{\Psi}_{f,i}^{\max}) \right] \quad (13)$$

Stress-strain relationships are readily derived from (1) and (2) above, as described by Fereidoon nezhad et al., (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016).

$$\mathbf{S} = \frac{d\Psi_{vol}}{dJ} \mathbf{J}\mathbf{C}^{-1} + 2 \frac{\partial \bar{\Psi}_m^0}{\partial \mathbf{C}} + \sum_{i=4,6} \left[2\eta_{s,i} \frac{\partial \bar{\Psi}_{f,i}^0}{\partial \mathbf{C}} - 2(1-\eta_{in}) \frac{\partial N}{\partial \mathbf{C}} \right] \quad (14)$$

This constitutive model is implicitly discretized and implemented in a user material subroutine (UMAT) by Fereidoon nezhad et al. (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016) to be used in ABAQUS/Standard. This subroutine has been used for all simulations in this work.

Material parameters

Different human tissues have different material properties. In Table 1 elastic and inelastic parameters for the three layers (intima, media, and adventitia) of two arteries and plaques are reported. The material parameters of the arteries are taken from (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016). Material parameters of atherosclerotic plaque are obtained by calibrating the constitutive model (equation (14)) to the experimental data of sample I documented in (Holzapfel, Sommer and Regitnig, 2004), using a least-square optimization algorithm in MATLAB. It is noted that the plaque comprises of different components and an average mechanical response of all constituents are considered here, as shown in Figure 1.

Table 1: Elastic and inelastic properties of two human arterial layers (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016), and elastic properties of plaque according to the experimental data documented in (Holzapfel, Sommer and Regitnig, 2004).

	μ (MPa)	k_1 (MPa)	k_2	κ	θ	μ^* (MPa)	k_1^* (MPa)	k_2^*	κ^*	r_1	m_1 (MPa)	m_2
<i>Artery I</i>												
<i>Intima</i>	0.017	0.263	41.191	0.3	90	0.001292	0.014591	0	0	1.49	0.0192	3
<i>Media</i>	0.012	0.059	3.871	0.02	45.3	0.000098	0.000611	0.665	0.29	1.44	0.0077	3.32
<i>Adventitia</i>	0.008	0.222	9.948	0.23	53.1	0.001206	0.000001	0.011	0.33	4	0.001	4
<i>Artery II</i>												
<i>Intima</i>	0.003	1.073	41.7	0.33	69	1.18	0.0064	0.0003	0.27	1.43	0.0101	2.61
<i>Media</i>	0.02	0.078	19.4	0.32	0.9	1.26	0.00177	0.017	0	2.83	0.0084	1.87
<i>Adventitia</i>	0.008	0.212	4.63	0.28	34.4	1.45	0.001239	0.734	0.33	3.72	0.047	1.5
<i>Plaque</i>												
	0.0075	3.5806	12	0.225	58	-	-	-	-	-	-	-

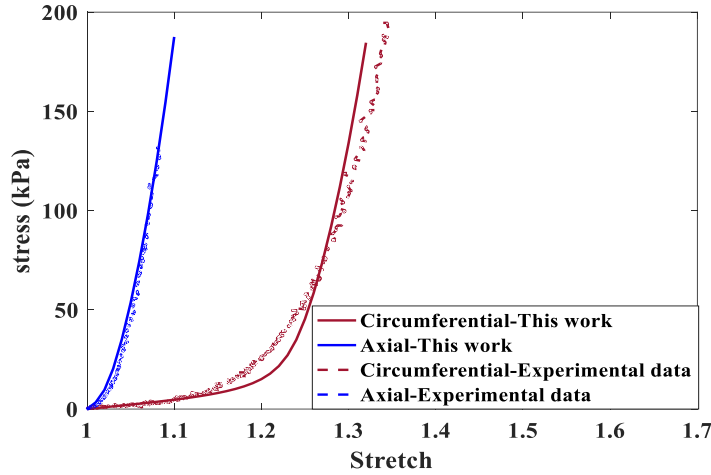


Figure 1: Conformability of the model to the uniaxial experimental data of atherosclerotic plaque (Holzapfel, Sommer and Regitnig, 2004).

2.1.2. Stent

Numerous balloon expandable stents are made from SS316LN stainless steel. Different values of material parameters are reported for SS316LN in the literature, which are in close agreement. Here, the material parameters are taken from (Auricchio *et al.*, 2001), where plastic deformation was considered (see Table 2).

Table 2: Material parameters for the stent

Young modulus (MPa)	196000
Poisson ratio	0.3
Yield stress (MPa)	205
Limit stress (MPa)	515
Limit nominal strain	60%

2.2. Model geometry

Clinical studies have identified factors such as the stent design and the deployment technique that are one cause for the success or failure of a stent treatment. In addition, the success rate may also depend on the stenosis type. Hence, for a particular stenotic artery, the optimal intervention can be identified by studying the influence of factors such as stent type, strut thickness and geometry of the stent cell (Holzapfel, 2005). In the present study we investigate the individual human stenosis, as presented in (Holzapfel, 2005). Therein, the geometrical model of the considered stenosis was composed of different tissue components, which were specified from high resolution magnetic resonance images and reconstructed by means of non-uniform rational B-splines. As illustrated in Figure 2, a high degree of non-symmetric stenosis is observed in the artery, in contrary to the symmetric assumption used in the majority of studies, also the dimensions of the stent, and the pattern which has been used for making the stent geometry in the CAD software Autodesk Inventor, are shown in Figure 3.

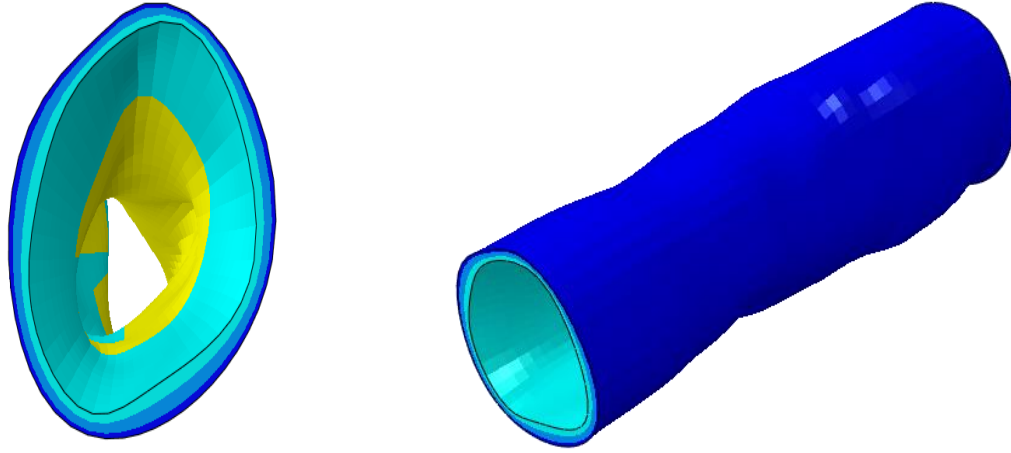


Figure 2: The geometry of the stenosis artery. The tissue components are adventitia ■, media ■, intima ■ and plaque ■.

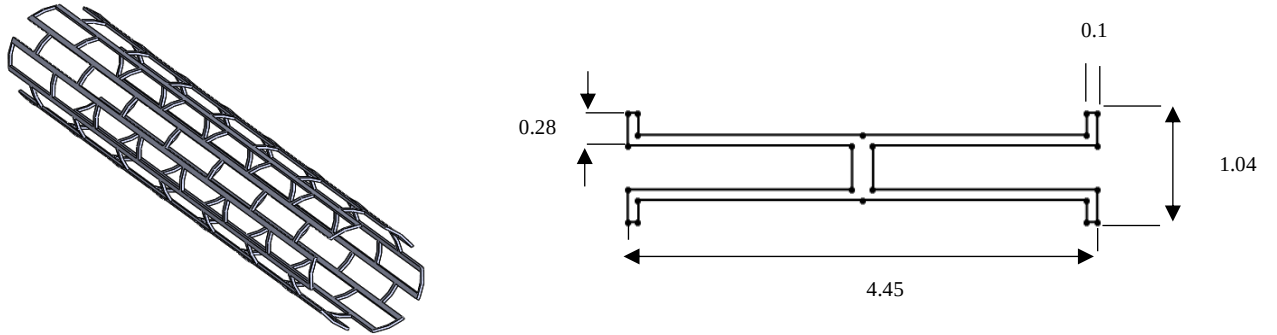


Figure 3: stent geometry. All dimensions are in mm.

The inner and outer diameter of the stent are 2mm and 2.1mm, respectively and the strut width is 0.1mm, and the initial lengths of the stent was 31mm.

2.3. Loading, boundary conditions, and meshing

The stent expansion is performed by imposition of a radial displacement to the stent. More sophisticated methods such as using an inflatable balloon has also been previously employed for this purpose (Cui *et al.*, 2010; De Beule *et al.* 2008). The radial displacement is applied on the inner surface of the stent struts to expand the stent from the crimped state, with the inner diameter of 2mm, to the fully-expanded state of 5.4mm diameter. Finally, the load is removed and the elastic recoil of stent is occurred.

The general contact in ABAQUS together with a frictionless tangential behavior are employed to simulate the contact between the outer surface of the stent and the inner surface of the plaque and the artery. Both ends of the artery are constrained in the longitudinal and circumferential directions, while a radial displacement is allowed. The stent is constrained in the longitudinal direction at one end; therefore, the stent length can change in the expansion process, and both ends of the stent are constrained in the circumferential direction.

Three-dimensional (3D) eight node linear, hybrid elements (C3D8H) are assigned to the mesh of the artery and the plaque and 3D eight node linear elements (C3D8) are employed for the stent. To check the mesh convergence, the analysis is performed with a refined mesh for the artery I. The results are shown in Table

3, which indicates the mesh independent result. After a mesh study, the total number of 4032 and 11825 elements have been used for the stent and the stenosed artery, respectively.

Table 3: The result of mesh analysis, which shows Max. principal stress and Max. principal strain distribution in simulations with different number of elements for the artery.

NO. of elements (artery+stent)	Max. principal stress (MPa)	Max. Principal strain	Max principal stress of the stent (MPa)
14487	0.33	0.41	363
15857	0.32	0.40	362.3
18602	0.32	0.39	362.1

3. Results and discussion

In this section, the results are presented and the implications of stenting on the arterial tissue is discussed. The cross sectional area of stenosed artery and the distribution of maximum principal stress after stent deployment is demonstrated in Figure 4. The inelastic constitutive model was used for arterial tissue. The results clearly show the lumen gain after stent deployment.

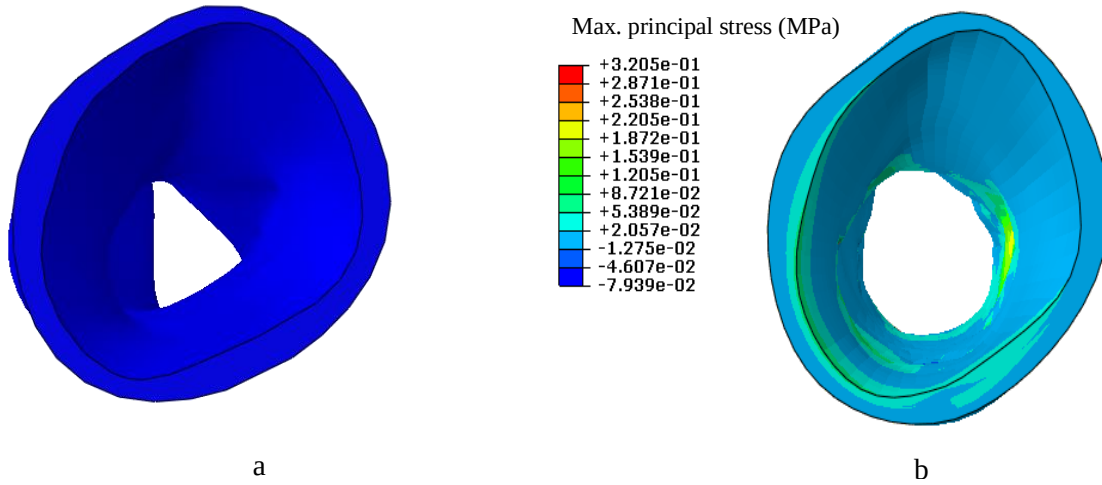


Figure 4: The distribution of the max. principal stress in arterial tissue a) before and b) after stent expansion, using the inelastic constitutive model.

As mentioned in the previous section, the diameter of the stainless steel stent decreases due to its recoils. In the Figure 5 the effect of this recoil is shown. After the stent recoil, the stress of the artery will decline, but the important point is that its stress-strain diagram, which is presented in Figure 6, is not compatible on the stress-strain diagram in the first step, which is the expansion step. This diagram is plotted for the point with the maximum principal stress in the intima layer.

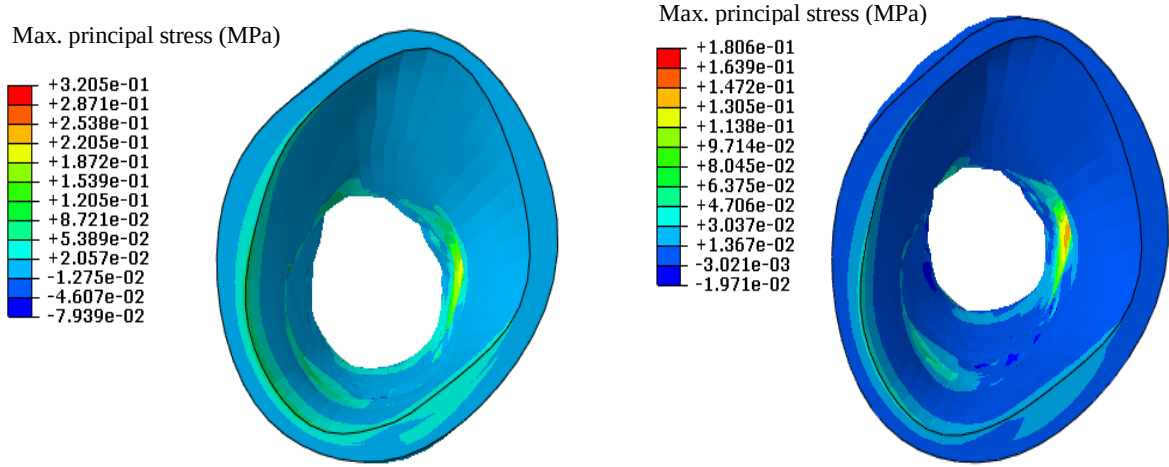


Figure 5: Max. principal stress of the artery and plaque after the first (in the left) and second step (in the right), which are the expansion and recoil of the stent, respectively.

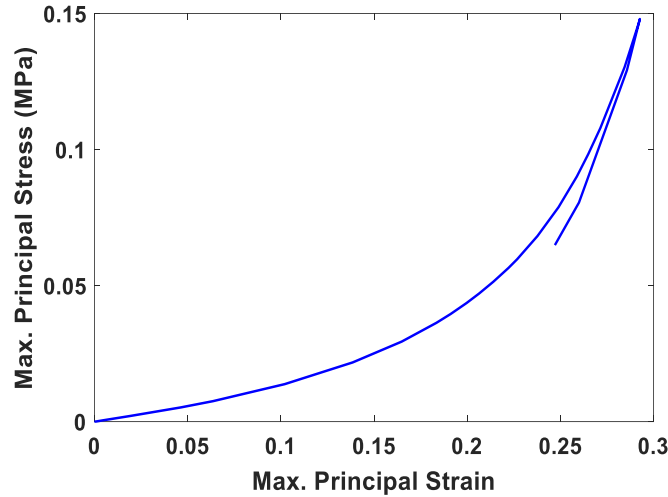


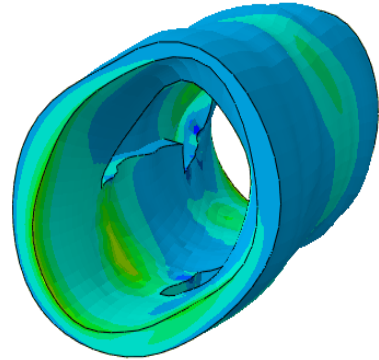
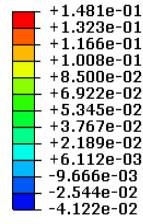
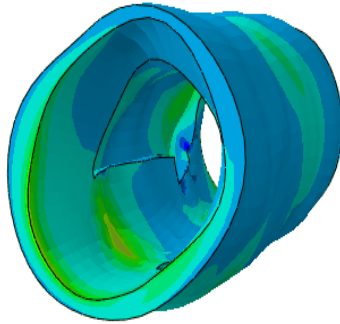
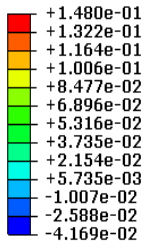
Figure 6: The variation of stress versus strain in the artery during expansion and recoil steps.

Given this result, in the next section results of the inelastic model and HGO model are compared. We examine how constitutive behavior of arterial tissue affect the stress distribution in arterial tissue and show the importance of considering damage on the correct prediction of the outcomes. In the next step, the effects of variation in arterial material parameters, analogous to different patients, on the final outcomes of the procedure are investigated.

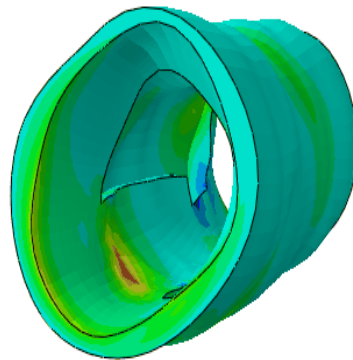
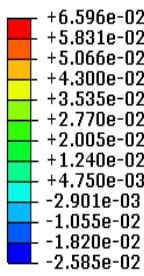
3.1 Effects of damage-induced arterial

Using the same stent geometry, two constitutive models i.e., the hyperelastic model and the inelastic model, as discussed in Section 2, are considered for the arterial wall. As depicted in Figure 7, the distributions of the maximum principal stress during expansion of the stent are generally similar for both the hyperelastic and inelastic models. However, as shown in Figure 7 the maximum principal stresses are different for hyperelastic and inelastic models after stent recoil. In the following, we do not show the plaque since we consider the inelastic model only for arterial layer, which are intima, media, and adventitia.

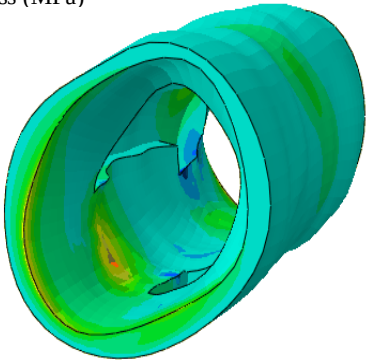
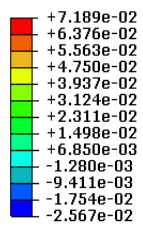
Max. principal stress (MPa)



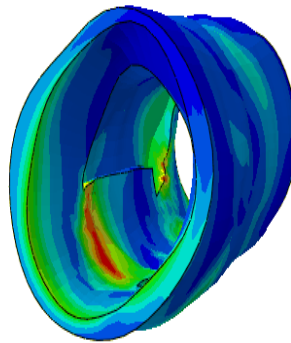
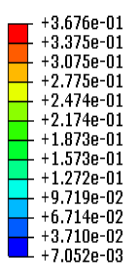
Max. principal stress (MPa)



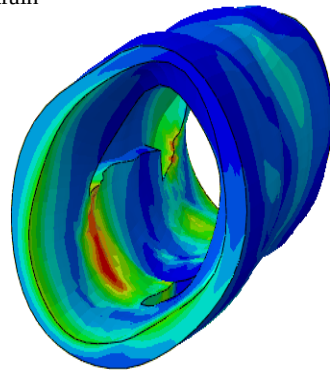
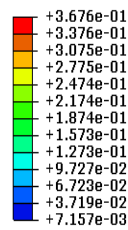
Max. principal stress (MPa)



Max. principal strain



Max. principal strain



Max. principal strain

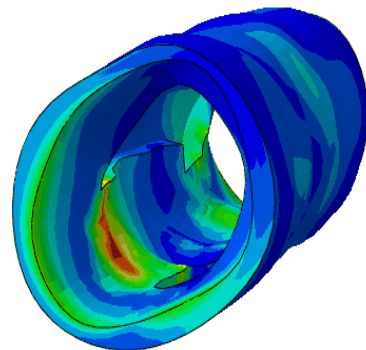
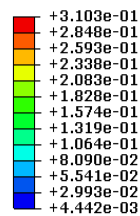
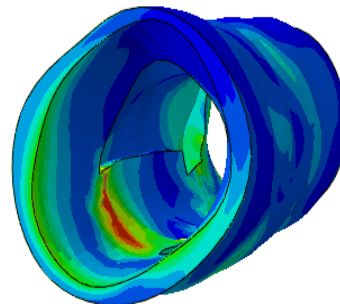
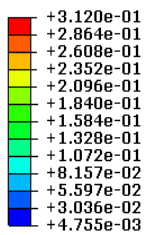


Figure 7: Distribution of maximum principal stress after (a) fully expansion of stent and (b) elastic recoil, and maximum principal strain after (c) fully expansion of stent, (d) elastic recoil of the stent. Left figures show the results for the inelastic model and the results of the hyperelastic model are shown in the right column.

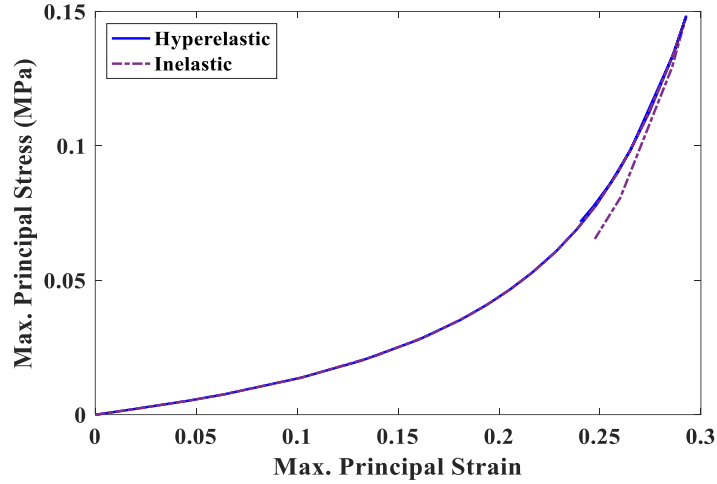
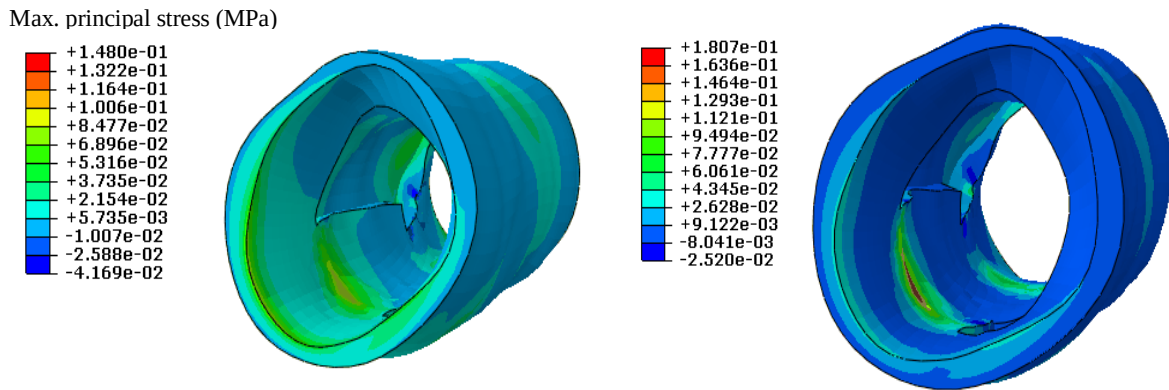


Figure 8: Maximum principal stress versus radial displacement for the hyperelastic and inelastic models during stent expansion and elastic recoil (This diagram is plotted for the point with the maximum principal stress in the intima layer).

It is noted that although the global stress distribution is not affected significantly by ignoring the inelastic behavior of arterial tissue, the hyperelastic models, as used in most of the previous computational models, are not able to predict the surgically-induced damage after procedure, contrary to the inelastic model considered in the present study. The importance of this behavior is clear when one experiences more than one stenting, as a result, using the inelastic model helps us to predict and analyze this procedure accurately.

3.2 Effects of arterial properties

To investigate the effect of variation in material properties of arterial tissue on the final outcomes of the procedure, the simulation is conducted for two sets of arterial parameters belonging to two different patients (see table 1). The distribution of the maximum principal stress, maximum principal strain and radial displacement of the artery are presented in Figure 9.



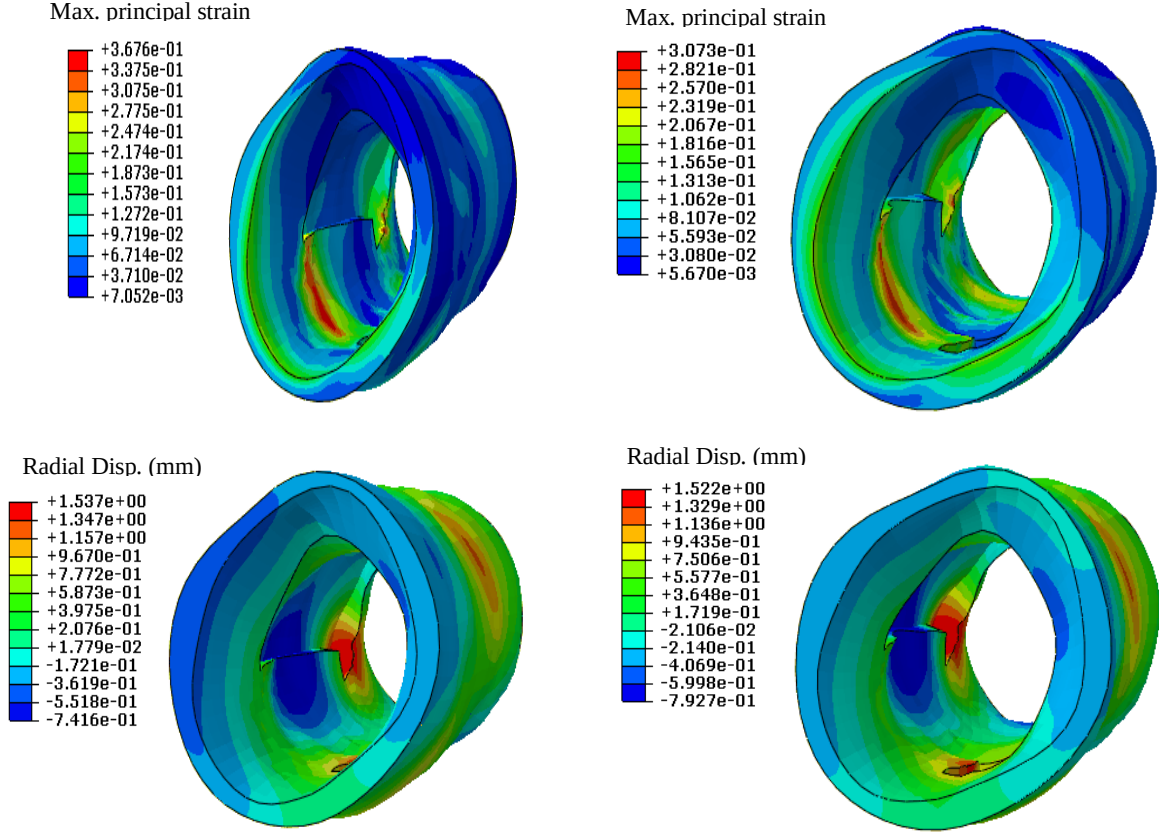


Figure 9: Distribution of (a) maximum principal stress, (b) maximum principal strain and (c) radial displacement of artery with parameters set I (left) and parameters set II (right).

In view of Figure 9, one may also observe that the location of the maximum stress are not coincident with the location of maximum strain and displacement. This is strongly dependent on the non-symmetric geometry of the stenosed artery and heterogeneous properties of plaque and artery. It is noteworthy to mention that this attribute could not be predicted where a symmetric geometry is assumed for the artery. In Figure 10, the maximum principal stress and damage evolution (equation (13)) versus maximum principal strain in the arterial tissue are demonstrated for two inelastic parameter sets, at the point with highest value of maximum principal stress. A higher amount of damage is predicted for artery I which also shows a higher stress in a specific strain compared to artery II.

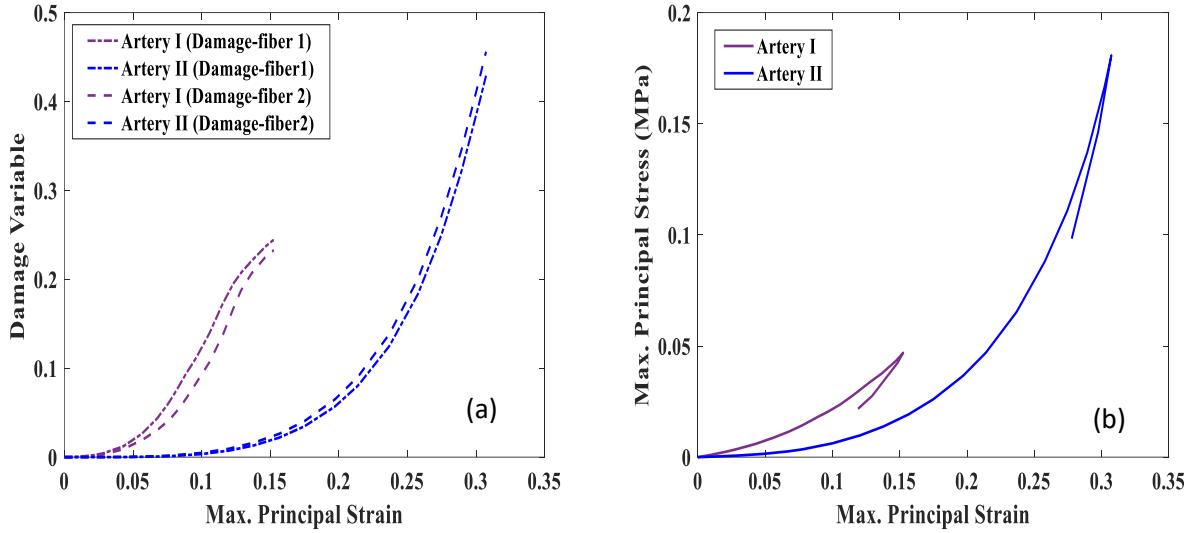


Figure 10: a) Damage evolution and b) stress-strain curve in arterial tissue for two different sets of inelastic properties.

4. Concluding Remarks

Balloon angioplasty with stent deployment is one of the most common treatments for atherosclerosis. Despite the significant benefits of this procedure, there are some short-term and long-term issues for the patients; such as plaque fragmentation and ISR. Computational models as a powerful tool for pre-clinical assessment of medical devices such as stent have gained a lot of momentum in the last years. However, most of those models are based on the simplified (idealized) geometry of stenosed artery. On the other hand, those models which consider the patient-specific geometry of stenosed artery have employed a hyperelastic constitutive model for arterial tissue, which is not enough for supra-physiological loading during clinical treatments, as discussed in (Fereidoon nezhad, Naghdabadi and Holzapfel, 2016).

Knowing that both patient-specific geometry of stenosed artery and suitable constitutive modeling of arterial tissue are critical for developing a precise computational model, a finite element model for stent deployment into a patient-specific stenosed artery was investigated in this paper, while inelastic arterial behavior due to the supra-physiological loading has been considered. Specially, the surgically induced injury (damage) in arterial wall, as the main cause of ISR, has been presented and the results are compared with the commonly-used hyperelastic behavior of arterial layers. Furthermore, the effects of arterial properties variation and analogues to different patients are investigated by doing the simulations for two different parameter sets.

In fact, the developed FE model provides a powerful tool and may serve as a ‘virtual workbench’ to investigate the stenting procedure computationally, allowing early evaluation of potential risks to the patient in terms of vessel wall damage, as the main cause of ISR (Fereidoon nezhad *et al.*, 2017), for a specific stent. The developed computational model is clinically relevant in two ways: (i) the proposed framework can be used in future studies to optimize stent design, e.g., to minimize tissue damage while lumen gain is maximum; (ii) the proposed framework could enable the prediction of surgically-induced damage evolution allowing a better prediction of the long-term adaptive behavior of an artery such as mechanically-induced growth which is the main cause of in-stent restenosis.

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References

- Auricchio, F. *et al.* (2001) "Finite-element analysis of a stenotic artery revascularization through a stent insertion", *Computer Methods in Biomechanics and Biomedical Engineering*, 4(3), 249–263.
- Boyle, C. J. and Prendergast, P. J. (2011) "InSilico Prediction of the Mechanobiological Response of Arterial Tissue : Application to Angioplasty and Stenting", *Journal of Biomechanical Engineering*, 133, 1–11.
- Cui, F. S. *et al.* (2010) "Effects of balloon length and compliance on vascular stent expansion", *International Journal of Applied Mechanics*, 2(3), 681–697.
- De Beule, M., Mortier, P., Carlier, S., Verheghe, B., Van Impe, R., and Verdonck, P., (2008) " Realistic finite element-based stent design: The impact of balloon folding", *Journal of Biomechanics*. 21(2) 383–389.
- Fereidoonzhad, B., Naghdabadi, R. and Holzapfel, G. A. (2016) "Stress softening and permanent deformation in human aortas: Continuum and computational modeling with application to arterial clamping", *Journal of the Mechanical Behavior of Biomedical Materials*. Elsevier, 61, 600–616.
- Fereidoonzhad, B., Naghdabadi, R., Sohrabpou, S., and Holzapfel, G. A. (2017) "A Mechanobiological model for damage-induced growth in arterial tissue with application to in-stent restenosis", *Journal of the Mechanics and Physics of Solids*. 101, 311–327.
- Holzapfel, G. A., Gasser, T. C., and Ogden, R. W. (2000) "A new constitutive framework for arterial wall mechanics and a comparative study of material models", *Journal of Elasticity*, 61, 1–48.
- Gasser, T. C., Ogden, R. W. and Holzapfel, G. A. (2006) "Hyperelastic modelling of arterial layers with distributed collagen fibre orientations", *Journal of the Royal Society Interface*, 3(6), 15–35.
- Gu, L., Zhao, S. and Fromming, S. R. (2012) "Arterial Wall Mechanics and Clinical Implications After Coronary Stenting: Comparisons of Three Stent Designs", *International Journal of Applied Mechanics*, 04(02), 1250013.
- Gültekin, O., Dal, H. and Holzapfel, G. A. (2018) "On the quasi-incompressible finite element analysis of anisotropic hyperelastic materials", *Computational Mechanics*.
- Holzapfel, G. A. (2005) "Changes in the Mechanical Environment of Stenotic Arteries During Interaction With Stents: Computational Assessment of Parametric Stent Designs", *Journal of Biomechanical Engineering*, 127(1), 166.
- Holzapfel, G. A. and Fereidoonzhad, B. (2017) "Modeling of Damage in Soft Biological Tissues", in *Biomechanics of Living Organs*. Elsevier Inc., 101–123.
- Holzapfel, G. A. and Gasser, T. C. (2007) "Computational stress-deformation analysis of arterial walls including high-pressure response", *International Journal of Cardiology*, 116(1), 78–85.
- Holzapfel, G. A., Sommer, G. and Regitnig, P. (2004) "Anisotropic Mechanical Properties of Tissue

Components in Human Atherosclerotic Plaques", *Journal of Biomechanical Engineering*, 126(5), 657.

Liu R.X., Xu S., Luo X.Y. and Liu Z.S., (2020) "Theoretical and numerical analysis of mechanical behaviors of a metamaterial-based shape memory polymer stent", *Polymers*, 12(8), 1784.

Liu R.X., McGinty S., Cui F.S., Luo X.Y. and Liu Z.S., (2019) "Modelling and Simulation of the Expansion of a Shape Memory Polymer Stent" *Engineering Computations*, 36(8).

Maher, E. *et al.* (2012) "An anisotropic inelastic constitutive model to describe stress softening and permanent deformation in arterial tissue", *Journal of the Mechanical Behavior of Biomedical Materials*. Elsevier Ltd, 12, 9–19.

Moerman, K.M., Fereidoon nezhad, B., and McGarry, J.P. (2017) "Novel hyperelastic models for large volumetric deformations", *International Journal of Solids and Structures*. 193-194, 474–491.

Mozafari, H. *et al.* (2018) "Migration resistance of esophageal stents : The role of stent design", *Computers in Biology and Medicine*, 100, 43–49.

Nolan, D. R. *et al.* (2014) "A robust anisotropic hyperelastic formulation for the modelling of soft tissue", *Journal of the Mechanical Behavior of Biomedical Materials*. Elsevier, 39, 48–60.

Rivlin, R. S. and A, P. R. S. L. (2006) "Large elastic deformations of isotropic materials. V. The problem of flexure", *Proceedings of the Royal Society of London. Series A. Mathematical and Physical Sciences*, 195(1043), 463–473.

Ruan, X. L. *et al.* (2018) "Mechanical Design of Antichiral-Reentrant Hybrid Intravascular Stent", *International Journal of Applied Mechanics*, 10(10), 1–20.

Schriefl, A. J. *et al.* (2012) "Determination of the layer-specific distributed collagen fibre orientations in human thoracic and abdominal aortas and common iliac arteries", *Journal of the Royal Society Interface*, (December 2011), 1275–1286.

Song, K., Bi, Y., Zhao, H., Wu, T., Xu, F., Zhao, G., (2020) " Structural optimization and finite element analysis of poly-L-lactide acid coronary stent with improved radial strength and acute recoil rate"

Voß, S. *et al.* (2019) "Stent-induced vessel deformation after intracranial aneurysm treatment – A hemodynamic pilot study", *Computers in Biology and Medicine*. Elsevier Ltd, 111(June), 103338.

Wang, M. *et al.* (2012) "Influence of work hardening on the performance of stents", *International Journal of Applied Electromagnetics and Mechanics*, 39(1–4), 495–500.