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Constitutive framework for the modeling of damage in collagenous soft tissues with application to arterial walls

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ABSTRACT

In this paper a new material model is proposed for the description of stress-softening observed in cyclic tension tests of collagenous soft tissues such as arterial walls, for applied loads beyond the physiological level. The modeling framework makes use of terms known from continuum damage mechanics and the concept of internal variables introducing a scalar-valued variable for the representation of fiber damage. A principle is given for the construction of damage models able to reflect remanent strains as a result of microscopic damage in the reinforcing collagen fiber families. Particular internal variables are defined able to capture the nature of arterial tissues that no damage occurs in the physiological loading domain. By application of this principle, specific models are derived and fitted to experimental data. Finally, their applicability in numerical simulations is shown by some representative examples where the damage distribution in arterial cross-sections is analyzed.

1. Introduction

Hypertension, overweight, rich alimentation, smoking, diabetes and stress may lead to biochemical and mechanical degenerative processes in collagenous soft tissues such as arterial walls. One consequence is the formation of a narrowing of the lumen, i.e. the inside space of an artery. In severe cases, when untreated, this may lead to a heart attack, a smoker's leg or a stroke. To prevent such complications one frequently used treatment is balloon dilatation which is often accompanied by the implantation of a stent. Thereby, a balloon catheter is inserted into the affected artery and dilated with the goal to increase the lumen. During balloon inflation *microscopic damage* is induced in the arterial wall which also contributes to the treatment success since it results to increased strains under physiological blood pressure. In order to gain more insight into the complex biomechanical processes during therapeutical interventions such as angioplasty and for the optimization of treatment methods, the modeling of arterial tissues under supra-physiological (therapeutical) loading and related computer simulations are subject of current research.

In the last decades various experiments have been carried out to identify the material behavior of arterial walls within the physiological loading domain, see, for example, the reviews in Abé et al. [1], Fung [14], Holzapfel and Ogden [25] or Humphrey [29]. In the literature it is widely accepted that arterial walls respond quite distinctly to their mechanical environment (see, for example [31]). This poses a special challenge since healthy arteries consist of three layers with different mechanical properties: (i) the intima, the innermost layer, with a rather insignificant contribution to the solid mechanical properties for healthy young individuals. However, the intima thickens and stiffens with age (atherosclerosis) so that the mechanical contribution may become significant; (ii) the media, the middle layer, showing a complex 3D network of smooth muscle cells, elastin and collagen fibrils; (iii) the adventitia, the outermost layer, consisting of thick bundles of collagen fibrils forming a fibrous tissue. Knowledge about the mechanics of the individual layers is a prerequisite for an enhanced understanding of the complex interaction of all constituents within the arterial wall. In almost all arteries the media is stiffer than the adventitia within the low loading domain, see the experimental observations in, for example, Maltzahn et al. [32], Xie et al. [55], Yu et al. [56], or in the more recent studies for human coronary arteries [27] and human carotid arteries [48]. The above mentioned experimental approaches are related to the analysis of loading within the physiological domain. With respect to degenerative processes occurring during angioplasty especially supra-physiological loading is

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required (for an overview see, for example [16]). These load levels are characterized by loading conditions much higher than those occurring under normal (physiological) conditions. Experimental studies as, for example, the one by Castaneda-Zuniga et al. [8] show that remanent deformations are obtained due to supra-physiological loading if a certain load level is exceeded. These observations are also confirmed by Oktay et al. [40] for carotid arteries of dogs, and by Holzapfel et al. [26] for human iliac arteries. Within the clinical context these effects are referred to as controlled vessel injury, see, for example, Castaneda-Zuniga [7]. The consequences of such damage are investigated in Zollikofer et al. [58] for canine arteries using electron- and optical microscopes. Profound microscopic damage is observed in the media by Castaneda-Zuniga [7]. In Schulze-Bauer et al. [46] layer-specific experiments of human adventitias are performed under physiological as well as supra-physiological conditions.

For analyzing the physiological material behavior of collagenous soft tissues numerous models have been proposed in the last decades. For example, in Vaishnav et al. [51] a two-dimensional model for a rabbit aorta is documented, while Fung et al. [15] take the exponential character of the stress–strain response into account. The proposed two-dimensional description has been extended to the three-dimensional case in Chuong and Fung [9]. In most of the recently proposed models a structural tensor is introduced with the aim to reflect the structural properties of the material leading to the invariant basis by using representation theorems for anisotropic tensor functions. The fundamental developments in the field of structural tensors are given in, for example, Boehler [5] and Spencer [50]. One of the first models for collagenous soft tissues such as tendons based on this concept was proposed by Weiss et al. [54]. However, the above mentioned models do not satisfy the polyconvexity condition and thus, they do not *a priori* guarantee the existence of solutions of underlying boundary-value problems. In Holzapfel et al. [24] a first polyconvex model is proposed as an exponential function of the fourth mixed invariant. Further polyconvex models, which are based on the fundamental polyconvex functions for transverse isotropy and orthotropy introduced by Schröder and Neff [45], able to describe collagenous soft tissues, are proposed in, for example, Itskov and Aksel [30], Balzani [2] or Ehret and Itskov [12]. A general principle for the construction of anisotropic polyconvex functions that automatically fulfill the condition of a stress-free reference configuration and its application to arterial tissues is given in Balzani et al. [3].

Since we are mainly interested in supra-physiological loading conditions, the modeling of *microscopical damage* is essential. The related damage leads to a softening phenomenon of the stress–strain response and thus, increased deformations under physiological conditions. For the description of isotropic softening there exist various models. One of the first representations of damage at large strains was pursued in Simo [47], see also Govindjee and Simo [18]. Therein the authors proposed a model for polymers, which is able to describe isotropic damage in the sense of the Mullins effect. By introducing different definitions of internal variables this approach has been extended to continuous damage by Miehe [34]. Another constitutive model for the description of the Mullins effect is based on filler matrix interaction and polymer chain interaction, see Marckmann et al. [33]. In order to describe damage, showing a saturation behavior during repeated unloading and reloading at fixed maximum load levels, Miehe and Keck [35] introduced a suitable model.

An alternative phenomenological form of describing damage mechanisms is linked with the notion of pseudo-elasticity. Thereby, the main idea is that different loading branches are described by different strain-energy functions. As one of the first works in this context one should mention Ogden and Roxburgh [38]. Basically, therein the Mullins effect is modeled, i.e. no remanent strains or distinct hysteresis for repeated unloading and reloading at fixed maximum load levels are obtained. An extension to the description of remanent strains is found in Ogden and Roxburgh [39], and to isotropic hysteresis in Dorfmann and Ogden [11]; specific hystereses in partial unloading and reloading cycles are treated in Dorfmann and Ogden [10]. For the incorporation of anisotropic damage a practical approach avoiding the usage of damage tensors is given in Balzani [2], see also Schröder et al. [44], where the anisotropic damage can be described by scalar-valued variables. The recently published model by Rodríguez et al. [43] uses scalar-valued variables as well and considers a stochastic framework on the basis of the wavy structure of the collagen fibers. A model for the preconditioning of soft biological tissues and the anisotropic Mullins effect is proposed in Ehret and Itskov [13]. Another recent approach provides the description of remanent strains after overstretch in the framework of finite plasticity based on the assumption of remaining deformations at the micro-scale of the fibers, see Gasser and Holzapfel [16]. A particular damage behavior for the matrix material is taken into account in, for example, Natali et al. [37] or Calvo et al. [6].

In the present study, the main goal is to define a construction principle for damage models that take into account remanent strains after unloading and to apply this principle to collagenous soft tissues such as arterial walls. Essentially, the paper is organized as follows: Section 2 explains the mathematical framework and introduces the construction principle. Furthermore, a model is given which is able to describe the complex softening hysteresis observed in cyclic uniaxial tension tests of collagenous soft tissues, where also a saturation behavior at fixed maximum load levels is shown. In Section 3 the model is specified for arterial wall tissues, and details regarding the algorithmic implementation are provided. In addition to that, it is shown that the model is able to capture the mechanical behavior of overstretched tissues also quantitatively, by fitting the model to experimental data. Section 4 provides a numerical example where the proposed model is implemented in a finite element environment, and the circumferential overstretch of a simplified atherosclerotic artery is analyzed with respect to the internal distribution of damage. Section 5 concludes the paper.

2. Mathematical modeling

2.1. Continuum mechanical framework

In the (undeformed) reference configuration $\mathcal{B}$ the body of interest is denoted by $\mathcal{B} \subset \mathbb{R}^3$ and parameterized in $\boldsymbol{X}$; in the (deformed) current configuration it is denoted by $\mathcal{S} \subset \mathbb{R}^3$ and parameterized in $\boldsymbol{x}$. The nonlinear deformation map $\boldsymbol{\varphi}_t : \mathcal{B} \to \mathcal{S}$ at time $t \in \mathbb{R}_+$ maps points $\boldsymbol{X} \in \mathcal{B}$ onto points $\boldsymbol{x} \in \mathcal{S}$. The deformation gradient $\boldsymbol{F}$ and the right Cauchy–Green tensor $\boldsymbol{C}$ are defined as

$$\boldsymbol{F}(\boldsymbol{X}) := \nabla \boldsymbol{\varphi}_t(\boldsymbol{X}) \quad \text{and} \quad \boldsymbol{C} := \boldsymbol{F}^T \boldsymbol{F} \tag{1}$$

with the Jacobian $J := \det \boldsymbol{F} > 0$. The mappings of the infinitesimal line $\mathrm{d}\boldsymbol{X}$, area $\mathrm{d}\boldsymbol{A} = \boldsymbol{N}\,\mathrm{d}A$ and volume elements $\mathrm{d}V$ to their spatial counterparts $\mathrm{d}\boldsymbol{x}$, $\mathrm{d}\boldsymbol{a} = \boldsymbol{n}\,\mathrm{d}a$ and $\mathrm{d}v$ are given by

$$\mathrm{d}\boldsymbol{x} = \boldsymbol{F}\,\mathrm{d}\boldsymbol{X}, \quad \boldsymbol{n}\,\mathrm{d}a = \mathrm{Cof}\,[\boldsymbol{F}]\boldsymbol{N}\,\mathrm{d}A \quad \text{and} \quad \mathrm{d}v = J\,\mathrm{d}V. \tag{2}$$

The cofactor is defined as $\mathrm{Cof}\,[\boldsymbol{F}] := J\boldsymbol{F}^{-T}$. It should be mentioned that the argument $X = (\boldsymbol{F}, \mathrm{Cof}\,[\boldsymbol{F}], \det \boldsymbol{F})$ plays an important role in the definition of polyconvexity. In the case of hyperelastic materials we postulate the existence of a strain-energy function $\Psi := \Psi(\boldsymbol{C})$, defined per unit reference volume. Then we compute the second Piola–Kirchhoff stress tensor and the Cauchy stress tensor as

$$\boldsymbol{S} = 2\partial_C \Psi \quad \text{and} \quad \boldsymbol{\sigma} = J^{-1}\boldsymbol{F}\boldsymbol{S}\boldsymbol{F}^T, \tag{3}$$