

Simulation of discontinuous damage incorporating residual stresses in circumferentially overstretched atherosclerotic arteries

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Abstract

When a balloon-angioplasty is performed, the arterial wall is overstretched and thereby damaged, which leads to a stiffness reduction in the arterial layers. An anisotropic damage model able to reflect the main damage mechanisms in overstretched arterial walls is used in combination with a polyconvex hyperelastic stored energy function. Furthermore, a method for the incorporation of residual stresses present in the wall of unloaded configurations is applied. The energy describes the anisotropic hyperelastic behavior of arteries under physiological conditions. Due to the assumption that the rupture of cross-bridges between collagenous micro-fibrils is responsible for the damage inside arterial walls, the damage function is applied to that part of the energy only which is associated to the fiber elasticity. For the incorporation of the residual stresses into the simulation, we apply a method which consists of two simulation steps. Finally, a numerical simulation of the overstretching of a simplified atherosclerotic artery is performed taking into account residual stresses.

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1. Introduction

The high number of deaths caused by cardiovascular diseases makes the biomechanical description and simulation of blood vessels more and more important. Numerous material models have been proposed to describe the hyperelastic behavior of arterial walls in the physiological range of deformations. As one of the first models taking some kind of anisotropy into account we should mention the model of Fung et al. [8]. Holzapfel et al. [10] proposed a model which is formulated in the framework of the invariant theory (see e.g., papers by Boehler [6] and Betten [5]). In their model, the orthotropy of arteries is represented by superposing two transversely isotropic layers having the same material parameters for each fiber direction.

Thereby, a weak interaction between these two fiber families is assumed. Due to the fact that this model involves a polyconvex stored energy, the existence of minimizers is ensured as well as material stability. For the definition of polyconvexity and its relationship to other generalized convexity conditions we refer to the paper by Ball [1].

Balzani et al. [3] introduced another polyconvex model, which satisfies a priori the condition of a stress-free reference configuration, also in the framework of the invariant theory. Furthermore, the model is adjustable to real biological soft tissues via simple “hand-fitting”, which means that no computational optimization scheme is required. This model is used in this contribution and adjusted to the stress–strain response in uniaxial tension tests of a human abdominal aorta.

In papers by Holzapfel et al. [10] and Gasser and Holzapfel [9] it is pointed out that damage is observed in experiments when arteries are overstretched. Generally, in anisotropic damage mechanics, damage tensors of second

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or fourth order are used, which leads to complicated functions containing numerous material parameters. For an overview of damage mechanics with respect to engineering applications see e.g., the work by Lemaitre and Desmorat [14]. Based on the introduction of a scalar-valued damage variable by Schröder et al. [17] a one-dimensional damage approach is extended to finite strains and embedded into the concept of internal variables, cf. the paper by Simo [18]. In order to obtain a thermodynamically consistent model we utilize the formalism described by Lemaitre and Chaboche [13]. The basic underlying assumption in this model is that damage occurs only in the fiber direction, which means that the damage function is applied only to the anisotropic part of the stored energy.

It is reported e.g., by Vaishnav and Vossoughi [19] that axial segments of arterial walls spring open when they are sliced in a radial direction. Therefore, the unloaded configuration cannot be stress-free, which means that residual stresses must exist in this state. As often stated in the literature, an artery opened by a radial cut can be assumed to be stress-free (see e.g., papers by Chuong and Fung [7] or Humphrey and Delange [12]). The straightforward approach to determining residual stresses is to consider the open artery and to perform a simulation in which the artery is closed. A method for the incorporation of residual stresses based on this approach is proposed by Balzani et al. [4]. This is the method applied in this contribution.

The degradation of the cardiovascular system results mostly from atherosclerotic degenerations of the blood vessels, which lead to the development of atherosclerotic plaques reducing the arterial lumen. A frequently applied treatment is to dilate the vessel lumen by balloon-angioplasty: a balloon-catheter is placed in the stenotic artery and inflated. This overstretches the artery circumferentially. In the present contribution, we simulate numerically this overstretching by means of the finite-element method.

This paper is organized as follows: in Section 2 we briefly recall the basic terminology of continuum mechanics needed for the following sections. Section 3 explains the utilized model consisting of a damage function which is used in combination with a polyconvex hyperelastic stored energy. Section 4 describes the method for including residual stresses and explains details of the procedure. In Section 5, the hyperelastic stored energy is adjusted to the physiological stress-strain response of the media and adventitia of a human abdominal aorta and an atherosclerotic artery is discretized for a finite-element calculation of its circumferential overstretching state. Section 6 summarizes the contribution.

2. Continuum mechanics and coordinate-invariant representation

Let $\mathcal{B} \subset \mathbb{R}^3$ be the body of interest in the reference configuration parametrized in \mathbf{X} , and let $\mathcal{S} \subset \mathbb{R}^3$ be the considered body in the current configuration parametrized in \mathbf{x} . The nonlinear deformation map $\varphi_t: \mathcal{B} \rightarrow \mathcal{S}$ at time $t \in \mathbb{R}_+$ maps points $\mathbf{X} \in \mathcal{B}$ onto points $\mathbf{x} \in \mathcal{S}$. The defor-

mation gradient \mathbf{F} and the strain measure, the right Cauchy–Green tensor \mathbf{C} , are defined by

$$\hat{\mathbf{F}}(\mathbf{X}) := \text{Grad}[\hat{\varphi}_t(\mathbf{X})] \quad \text{and} \quad \mathbf{C} := \mathbf{F}^T \mathbf{F}, \quad (1)$$

where the hat-symbol denotes functional dependencies. Due to the fact that the determinant of \mathbf{F} represents the change of infinitesimal volume elements, we require $\hat{J}(\mathbf{X}) := \det[\hat{\mathbf{F}}(\mathbf{X})] > 0$ in order to prevent the material from interpenetrating itself.

Focusing on hyperelasticity we consider the stored energy function ψ , which is defined per unit reference volume. In order to satisfy the principle of material frame-indifference we concentrate on reduced constitutive equations formulated in the right Cauchy–Green tensor. To account for the anisotropic material behavior of biological soft tissues, the material symmetries are represented by an additional argument tensor, the so-called structural tensor. Then the second Piola–Kirchhoff stress tensor \mathbf{S} , the Kirchhoff stress tensor $\boldsymbol{\tau}$, and the Cauchy stresses $\boldsymbol{\sigma}$ are given by

$$\mathbf{S} = 2\partial_{\mathbf{C}}\hat{\psi}(\mathbf{C}, \mathbf{M}), \quad \boldsymbol{\tau} = \mathbf{F}\mathbf{S}\mathbf{F}^T \quad \text{and} \quad \boldsymbol{\sigma} = \mathbf{J}^{-1}\boldsymbol{\tau}. \quad (2)$$

With the transformation \mathbf{Q} the structural tensor is given by

$$\mathbf{M} = \mathbf{Q}^T \mathbf{M} \mathbf{Q} \quad \forall \mathbf{Q} \in \mathcal{G}_i \subset \text{SO}(3) \quad (3)$$

and \mathcal{G}_i characterizes the transverse-isotropy group with respect to a local reference configuration. $\text{SO}(3)$ denotes the special orthogonal group with $\det \mathbf{Q} = 1$. It should be noted that we assume a weak interaction between the two fiber families in arterial walls and therefore the superposition of two transversely isotropic models reflects the orthotropic response. The principle of material symmetry requires the invariance of ψ under transformations with elements of the symmetry group, i.e.

$$\hat{\psi}(\mathbf{Q}^T \mathbf{C} \mathbf{Q}, \mathbf{M}) = \hat{\psi}(\mathbf{C}, \mathbf{M}) \quad \forall \mathbf{Q} \in \mathcal{G}_i, \mathbf{C}. \quad (4)$$

Let \mathbf{a} with $\|\mathbf{a}\| = 1$ be the preferred direction of the material; then, the suitable structural tensor, whose symmetry group has to be identical to the material symmetry group, is defined as $\mathbf{M} := \mathbf{a} \otimes \mathbf{a}$. This leads to an isotropic tensor function, and

$$\psi = \hat{\psi}(\mathbf{C}, \mathbf{M}) = \hat{\psi}(\mathbf{Q}^T \mathbf{C} \mathbf{Q}, \mathbf{Q}^T \mathbf{M} \mathbf{Q}) \quad \forall \mathbf{Q} \in \text{SO}(3), \mathbf{C}. \quad (5)$$

This representation reflects the transverse-isotropy group and we are able to express the functional dependence of ψ in terms of the invariants of the argument tensors (\mathbf{C}, \mathbf{M}) . The principle invariants of \mathbf{C} are given by

$$I_1 := \text{tr}[\mathbf{C}], \quad I_2 := \text{tr}[\text{cof} \mathbf{C}] \quad \text{and} \quad I_3 := \det[\mathbf{C}] \quad (6)$$

with the cofactor defined by $\text{cof}[\mathbf{C}] = \det[\mathbf{C}]\mathbf{C}^{-1}$. Due to $\text{tr}[\mathbf{M}] = 1$ we obtain the mixed invariants of \mathbf{C} and \mathbf{M} from the definitions

$$J_4 := \text{tr}[\mathbf{C}\mathbf{M}] \quad \text{and} \quad J_5 := \text{tr}[\mathbf{C}^2\mathbf{M}]. \quad (7)$$

These invariants form a possible polynomial basis for the stored energy, i.e.

$$\psi := \hat{\psi}(I_1, I_2, I_3, J_4, J_5). \quad (8)$$

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