VIII International Conference on Computational Plasticity
COMPLAS VIII
E. Oñate and D.R.J. Owen (Eds)

MODELING OF ANISOTROPIC DAMAGE IN ARTERIAL WALLS BASED ON POLYCONVEX STORED ENERGY FUNCTIONS

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Key words: Hyperelasticity, Polyconvexity, Anisotropic, Damage, Soft Tissues.

Summary. *In this contribution an anisotropic damage model for arterial walls is proposed assuming damage only in fiber direction. This approach is applied to a polyconvex stored energy function in order to obtain a Legendre-Hadamard stable material model for fixed damage states, which also guarantees the existence of minimizers of underlying boundary value problems. In the present paper the proposed model is adjusted for the Media and Adventitia of a human abdominal aorta and a numerical example is presented where the damage distribution in an overexpanded atherosclerotic artery is investigated.*

1 INTRODUCTION

Arterial walls are characterized by an anisotropic and incompressible material behavior. Due to the special composition, orientation and weak interaction of particular fibers within arterial walls the material can be approximated by the addition of an isotropic part representing the ground substance and the superposition of two transversely isotropic models for two embedded fiber families, cf. Holzapfel, Gasser and Ogden [1]. A suitable polyconvex model for the description of the hyperelastic basic behavior of soft biological tissues found in arterial walls is introduced in Balzani, Neff, Schröder and Holzapfel [2]. This model is founded on the concept of structural tensors and representation theorems of isotropic tensor functions are applied; for an introduction to this subject see Boehler [3]. Discontinuous damage effects are observed in experiments when arteries are overexpanded, cf. [1]. This effect can be described by the model proposed in Balzani, Schröder and Gross [4], see also Schröder, Balzani and Gross [5]. The damage model therein is formulated in the concept of internal variables, see Lemaitre and Chaboche [6]. Additionally, a referential damage state is introduced in order to satisfy the assumption that no damage occurs in the physiological range of deformations.

2 Constitutive Model

In the context of hyperelasticity the material behavior can be described by a stored energy function $\psi := \psi(\mathbf{C}, \mathbf{M})$, here defined per unit reference volume. In order to fulfill the principle of material frame indifference a priori, the energy depends on the right Cauchy Green deformation tensor $\mathbf{C}$. For the representation of the transverse isotropy, important for the description of the stress-strain response of the fibers inside arterial walls, a so-called structural tensor $\mathbf{M}_{(a)} := \mathbf{a}_{(a)} \otimes \mathbf{a}_{(a)}$ with the fiber direction $\mathbf{a}$ is introduced. This leads to the fact that the constitutive equation fits into the definition of an isotropic tensor function and therefore automatically fulfills the principle of material symmetry. We consider a stored energy of the form given by

$$\psi = \psi^{iso} + \sum_{a=1}^{2} \left[(1 - D_{(a)}) \hat{\psi}^0_{(a)} \right] , \quad \text{with} \quad \hat{\psi}^0_{(a)} = \psi^{aniso1}_{(a)} + \psi^{aniso2}_{(a)} , \tag{1}$$

with the isotropic part for the embedding ground substance

$$\psi^{iso} = C_1 \left(\frac{I_1}{I_3^{1/3}} - 3 \right) + \varepsilon \left(I_3^\gamma + \frac{1}{I_3^\gamma} - 2 \right) \tag{2}$$

and the two transversely isotropic parts for the fibers

$$\psi^{aniso1}_{(a)} = \begin{cases} \alpha_1 (J_4^{(a)} - 1)^{\alpha_2} & \text{for} \quad J_4^{(a)} \geq 1 \\ 0 & \text{for} \quad J_4^{(a)} < 1 \end{cases} \tag{3}$$

$$\psi^{aniso2}_{(a)} = \begin{cases} \alpha_3 (K_3^{(a)} - 2)^{\alpha_4} & \text{for} \quad K_3^{(a)} \geq 2 \\ 0 & \text{for} \quad K_3^{(a)} < 2 \, , \end{cases} \tag{4}$$

proposed in [2]. It is to be remarked that the hyperelastic isotropic and transversely isotropic stored energy functions are polyconvex. Hereby, the existence of solutions of boundary value problems is guaranteed if coercivity of the isotropic part is satisfied and as a byproduct the Legendre-Hadamard-condition is fulfilled a priori. The damage is characterized by the scalar damage variable D given by

$$\hat{D}_{(a)}(\hat{\psi}^0_{(a)}) = \gamma_1 \left(1 - e^{(-\beta_{(a)}/\gamma_2)} \right) , \tag{5}$$

which is a function of the internal variable $\beta_{(a)}$

$$\beta_{(a)} = \sup_{0 \leq s \leq t} \left[\frac{\langle \hat{\psi}^0_{(a),s} - \hat{\psi}^0_{(a),ini} \rangle}{|\hat{\psi}^0_{(a),ini}|} \right] . \tag{6}$$

This variable is defined in such a way, that the discontinuous character of the damage in arterial walls can be described. Additionally, $\hat{\psi}^0_{(a),ini}$ represents the transversely isotropic effective energy at a referential damage state, where the damage evolution starts. For details of the damage model, especially the function for the stresses, thermodynamical consistence and derivation of tangent moduli we refer to [5].

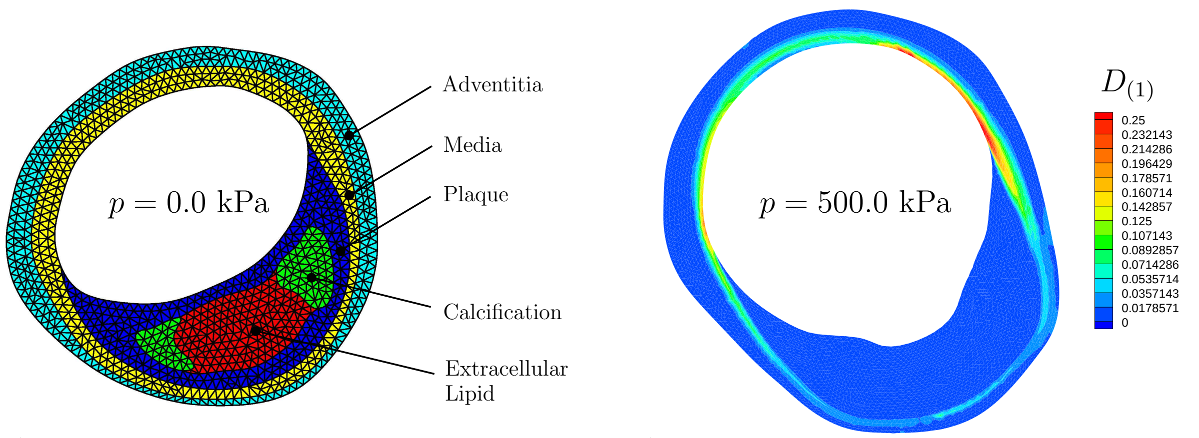
3 Damage in a Human Abdominal Aorta

As an example we simulate the deformation of a human abdominal aorta with a slight artherosclerotic plaque. The first step is the adjustment of the elastic material parameters to experiments in the physiological range of deformations in order to obtain the hyperelastic ground behavior, cf. [2]. The chosen material parameters for the Media and Adventitia are given in Table 1.

	c_1 [kPa]	ε [kPa]	γ [-]	α_1 [kPa]	α_2 [-]	α_3 [kPa]	α_4 [-]
Media	17.0	22.0	10.8	$9 \cdot 10^{14}$	20.5	17.0	1.8
Adventitia	7.0	22.0	10.8	25000.0	5.0	110.0	2.0

Table 1: Material parameters of the arterial wall

The components of the artherosclerotic plaque are treated as isotropic and modeled by the Neo-Hooke model; no damage evolution is assumed. For the material parameters describing the damage inside the arterial wall we choose $\gamma_1 = 0.9$, $\gamma_2 = 280.0$ kPa for the Media and $\gamma_1 = 0.9$, $\gamma_2 = 1000.0$ kPa for the Adventitia.



a) b)

Figure 1: a) Finite-Element mesh of unloaded configuration of the considered artery with particular components and b) overexpanded artery at an internal pressure of $p = 500.0$ kPa ≈ 3750.0 mmHg with distribution of damage $D_{(1)}$.

In Fig. 1 the considered Finite-Element mesh of a slightly deseased artery is illustrated. Internal pressure of at first $p = 180$ mmHg, which is regarded as the maximum value of the physiological load, is applied. This situation is defined as the referential damage state and now the damage evolution starts. The internal pressure is increased up to $p = 3750$ mmHg and for this loading condition the damage distribution inside the artery is depicted in Fig. 1. It should be noted that the calcification is treated as nearly rigid and the parameters for the other plaque components are rather arbitrarily chosen.

4 Conclusion

In this contribution the deformation of a human abdominal aorta with a slight artherosclerotic plaque has been simulated. The hyperelastic basic behavior has been represented by a polyconvex stored energy. Thereby, we satisfy a priori the Legendre-Hadamard condition and guarantee the existence of solutions of the boundary value problem. A thermodynamically consistent damage model has been applied to this energy, which is able to describe discontinuous damage inside the arterial wall considering a referential damage state. The parameters have been adjusted to the Media and Adventitia and a computer simulation of the overexpansion of an artery showed the performance of the model.

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