Biomechanics of Soft Tissue

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10.11.1 VALIDITY

An efficient constitutive formulation approximates all types of soft tissues with a reasonable accuracy over a large strain range. We request a simple constitutive equation with only a few material parameters involved that allow for an “explanation” of the material response of tissues in terms of their structure. In addition, we request that the constitutive formulation is fully three-dimensional and consistent with both mechanical and mathematical...
requirements, applicable for arbitrary geometries, and suitable for use within the context of finite element methods in order to solve complex initial boundary-value problems.

The presented general model is a fully three-dimensional material description of soft tissues for which nonlinear continuum mechanics is used as the fundamental basis [10, 18]. It has the special feature that it is based partly on histological information (i.e., the microscopic structure of organs and tissues). The general model describes the highly nonlinear and anisotropic behavior of soft tissues as composites reinforced by two families of collagen fibers. The constitutive framework is based on the theory of the mechanics of fiber-reinforced composites [26] and is suitable to describe a wide variety of physical phenomena of soft tissues. The performance and the physical mechanism of the model are presented in Reference [11]. As a representative example, the general model for soft tissues is specified to predict the mechanical response of healthy and young arteries under physiological loading conditions [12]. The model neglects active components, i.e., contracting elements with biochemical energy supply which are controlled by biological mechanisms, and is concerned with the description of the passive state of arteries.

The models are suitable for predicting the anisotropic elastic response of soft tissues in the large strain domain. A suitable constitutive and numerical model that is general enough to describe the finite viscoelastic domain is documented in Reference [11]. The presented models do not consider acute and long-term changes in geometry and/or the mechanical response of tissues due to, for example, drugs, aging, and disease. When soft tissues are subjected to loads that are beyond their physiological range, the load-carrying fibers of the tissue slip relative to each other. In clinical procedures tissues may undergo irreversible (plastic) deformations [12] which are of medical importance. Constitutive equations for describing plastic deformations of, for example, arteries are proposed in References [8, 27].

10.11.2 BACKGROUND ON THE STRUCTURE OF SOFT TISSUES: COLLAGEN AND ELASTIN

What do we mean by soft tissues? A primary group of tissue which binds, supports, and protects our human body and structures such as organs is soft connective tissue. In contrary to other tissues, it is a wide-ranging biological material in which the cells are separated by extracellular material. Connective tissues may be distinguished from hard (mineralized) tissues such as bones for their high flexibility and their soft mechanical properties. This article
discusses connective tissue from the point of view of material science, biomechanics, and structural engineering (for more details, see, for example, Reference [6], Chapter 7).

Examples of soft tissues are tendons, ligaments, blood vessels, skins, or articular cartilages, among many others. Tendons are muscle-to-bone linkages that stabilize the bony skeleton (or produce motion), and ligaments are bone-to-bone linkages that restrict relative motion. Blood vessels are prominent organs composed of soft tissues which have to distend in response to pulse waves. The skin is the largest single organ (16% of the human adult weight). It supports internal organs and protects our body. Articular cartilages form the surface of body joints (which is a layer of connective tissue with a thickness of 1–5 mm), distribute loads across joints, and minimize contact stresses and friction.

Soft connective tissues of our body are complex, fiber-reinforced, composite structures. Their mechanical behavior is strongly influenced by the concentration and structural arrangement of constituents such as collagen and elastin, the hydrated matrix of proteoglycans, and the topographical site and respective function in the organism.

10.11.2.1 Collagen

Collagen is a protein which is a major constituent of the extracellular matrix of connective tissue. It is the main load-carrying element in a wide variety of soft tissues and is very important to human physiology (for example, the collagen content of the [human] Achilles tendon is about 20 times that of elastin).

Collagen is a macromolecule with a length of about 280 nm. Collagen molecules are linked to each other by covalent bonds that build collagen fibrils. Depending on the primary function and the requirement of strength of the tissue, the diameter of collagen fibrils varies (the order of magnitude is 1.5 nm [17]). In the structure of tendons and ligaments, for example, collagen appears as parallel-oriented fibers [1], whereas many other tissues have an intricate disordered network of collagen fibers embedded in a gelatinous matrix of proteoglycans.

More than 12 types of collagen have been identified [17]. The most common collagen is type I, which can be isolated from any tissue. It is the major constituent in blood vessels. The rodlike shape of the collagen molecule comes from three polypeptide chains which are composed in a right-handed triple-helical conformation. Most of the collagen molecule consists of three amino acids: glycine (33%), which enhances the stability of the molecule, proline (15%), and hydroxyproline (15%) [23].
The intramolecular crosslinks of collagen gives the connective tissues the strength which varies with age, pathology, etc. (for a correlation between the collagen content in the tissue, % dry weight, and its ultimate tensile strength, see Table 10.11.1. The function and integrity of organs are maintained by the tension in collagen fibers. They shrink upon heating breakdown of the crystalline structure (at 65°C, for example, mammalian collagen shrinks to about one-third of its initial length, [6], p. 263).

10.11.2.2 Elastin

Elastin, like collagen, is a protein which is a major constituent of the extracellular matrix of connective tissue. It is present as thin strands in soft tissues such as skin, lung, ligamenta flava of the spine, and ligamentum nuchae (the elastin content of the latter is about five times that of collagen).

The long flexible elastin molecules build up a three-dimensional (rubber-like) network, which may be stretched to about 2.5 times the initial length of the unloaded configuration. In contrast to collagen fibers, this network does not exhibit a pronounced hierarchical organization. As for collagen, 33% of the total amino acids of elastin consists of glycine. However, the proline and hydroxyproline contents are much lower than in collagen molecules.

The mechanical behavior of elastin may be explained within the concept of entropic elasticity. As for rubber, the random molecular conformations, and hence the entropy, change with deformation. Elasticity arises through entropic straightening of the chains, i.e., a decrease of entropy, or an increase of internal energy (see, for example, References [9]). Elastin is essentially a linearly elastic material (tested for the ligamentum nuchae of cattle). It displays very small relaxation effects (they are larger for collagen).

<table>
<thead>
<tr>
<th>Material</th>
<th>Ultimate tensile strength (MPa)</th>
<th>Ultimate tensile strain (%)</th>
<th>Collagen (% dry weight)</th>
<th>Elastin (% dry weight)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tendon</td>
<td>50–100</td>
<td>10–15</td>
<td>75–85</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Ligament</td>
<td>50–100</td>
<td>10–15</td>
<td>70–80</td>
<td>10–15</td>
</tr>
<tr>
<td>Aorta</td>
<td>0.3–0.8</td>
<td>50–100</td>
<td>25–35</td>
<td>40–50</td>
</tr>
<tr>
<td>Skin</td>
<td>1–20</td>
<td>30–70</td>
<td>60–80</td>
<td>5–10</td>
</tr>
<tr>
<td>Articular cartilage</td>
<td>9–40</td>
<td>60–120</td>
<td>40–70</td>
<td>—</td>
</tr>
</tbody>
</table>
10.11.3 GENERAL MECHANICAL
CHARACTERISTICS OF SOFT TISSUES

Before describing a model for soft tissues, it is beneficial and instructive to gain some insight into their general mechanical characteristics. Soft tissues behave anisotropically because of their fibers, which tend to have preferred directions. In a microscopic sense they are nonhomogeneous materials because of their composition. The tensile response of soft tissue is nonlinear stiffening, and tensile strength depends on the strain rate. In contrast to hard tissues, soft tissues may undergo large deformations. Some soft tissues show viscoelastic behavior (relaxation and/or creep), which has been associated with the shear interaction of collagen with the matrix of proteoglycans [16] (the matrix provides a viscous lubrication between collagen fibrils).

In a simplified way we explain here the tensile stress-strain behavior for skin, an organ consisting mainly of connective tissues, which is representative of the mechanical behavior of many (collagenous) soft connective tissues. For the connective tissue parts of the skin the three-dimensional network of fibers appears to have preferred directions parallel to the surface. However, in order to prevent out-of-plane shearing, some fiber orientations also have components out of plane.

Figure 10.11.1 shows a schematic diagram of a typical J-shaped (tensile) stress-strain curve for skin. This form, representative for many soft tissues,
differs significantly from stress-strain curves of hard tissues or from other types of (engineering) materials. In addition, Figure 10.11.1 shows how the collagen fibers straighten with increasing stress.

The deformation behavior for skin may be studied in three phases: I, II, and III.

**Phase I.** In the absence of load the collagen fibers, which are woven into a rhombic-shaped pattern, are in relaxed conditions and appear wavy and crimped. Unstretched skin behaves approximately isotropically. Initially low stress is required to achieve large deformations of the individual collagen fibers without requiring stretch of the fibers. In phase I the tissue behaves like a very soft (isotropic) rubber sheet, and the elastin fibers (which keep the skin smooth) are mainly responsible for the stretching mechanism. The stress-strain relation is approximately linear, and the elastic modulus of skin in phase I is low (0.1–2.0 MPa).

**Phase II.** In phase II, as the load is increased, the collagen fibers tend to line up with the load direction and bear loads. The crimped collagen fibers gradually elongate and interact with the hydrated matrix. With deformation the crimp angle in collagen fibrils leads to a sequential uncrimping of fibrils. Note that the skin is normally under tension in vivo.

**Phase III.** In phase III, at high tensile stresses, the crimp patterns disappear and the collagen fibers become straighter. They are primarily aligned with one another in the direction in which the load is applied. The straightened collagen fibers resist the load strongly, and the tissue becomes stiff at higher stresses. The stress-strain relation becomes linear again. Beyond the third phase the ultimate tensile strength is reached and fibers begin to break.

The mechanical properties of soft tissues depend strongly on the topography, risk factors, age, species, physical and chemical environmental factors such as temperature, osmotic pressure, and pH, and the strain rate. The material properties are strongly related to the quality and completeness of experimental data, which come from in vivo or in vitro tests having the aim of mimicking real loading conditions. Therefore, to present specific values for the ultimate tensile strength and strain of a specific tissue is a difficult task. Nevertheless, Table 10.11.1 attempts to present ranges of values of mechanical properties and collagen and elastin contents (% dry weight) in some representative organs mainly consisting of soft connective tissues.

**10.11.4 DESCRIPTION OF THE MODEL**

At any referential position $X$ of the tissue we postulate the existence of a Helmholtz free-energy function $\Psi$. We assume the decoupled form

$$\Psi = U(X; J) + \tilde{\Psi}(X; \tilde{C}, A_1, A_2)$$

where $U$ is a purely *volumetric* (dilatational) contribution and $\bar{\Psi}$ is a purely *isochoric* (volume-preserving) contribution to the free energy $\Psi$. Here $\bar{\mathbf{C}} = \tilde{\mathbf{F}}^T \tilde{\mathbf{F}}$ denotes the modified right Cauchy-Green tensor and $\tilde{\mathbf{F}} = J^{-1/3} \mathbf{F}$ is the unimodular (distortional) part of the deformation gradient $\mathbf{F}$, with $J = \det \mathbf{F} > 0$ denoting the local volume ratio. In addition, in Eq. 1, $\{A_1, A_2\}$ is a set of two (second-order) tensors which characterize the anisotropic properties of the tissue at any $X$. The structure tensors $A_1$ and $A_2$ are defined as the tensor products $a_{0x} \otimes a_{0x}$, where $a_{0x}$, $x = 1, 2$, are two unit vectors characterizing the orientations of the families of collagen fibers in the (undeformed) reference configuration of the tissue (see Figure 10.11.2).

Since most types of soft tissues are regarded as incompressible (for example, arteries do not change their volume within the physiological range of deformation [2]), we now focus attention on the description of their isochoric deformation behavior characterized by the energy function $\bar{\Psi}$. We suggest the simple additive split

$$
\bar{\Psi} = \bar{\Psi}_{\text{iso}}(X; \bar{I}_1) + \bar{\Psi}_{\text{aniso}}(X; \bar{I}_1^*, \bar{I}_2^*)
$$

(2)

of $\bar{\Psi}$ into a part $\bar{\Psi}_{\text{iso}}$ associated with isotropic deformations and a part $\bar{\Psi}_{\text{aniso}}$ associated with anisotropic deformations. This is sufficiently general to capture the salient mechanical feature of soft tissue elasticity as described in Section 10.11.3 (a more general constitutive framework is presented in References [8, 11, 12]). In Eq. 2 we used $\bar{I}_1 = \bar{\mathbf{C}} : \mathbf{I}$ for the first invariant of tensor $\bar{\mathbf{C}}$ (it is the second-order unit tensor), and the definitions

$$
\bar{I}_1^* (\bar{\mathbf{C}}, a_{01}) = \bar{\mathbf{C}} : A_1, \quad \bar{I}_2^* (\bar{\mathbf{C}}, a_{02}) = \bar{\mathbf{C}} : A_2
$$

(3)

of the invariants, which are stretch measures for the two families of collagen fibers (see, for example, References [10, 26]). The invariants $\bar{I}_1^*$ and $\bar{I}_2^*$ are squares of the stretches in the directions of $a_{01}$ and $a_{02}$, respectively. Isotropy is described through the invariant $\bar{I}_1$ and anisotropy through $\bar{I}_1^*$ and $\bar{I}_2^*$.

**FIGURE 10.11.2** Arrangement of collagen fibers in the reference configuration characterized by two unit vectors $a_{01}$, $a_{02}$ at position $X$. 


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Since the (wavy) collagenous structure of tissues is not active at low stresses (it does not store strain energy), we associate $\Psi_{iso}$ with the mechanical response of the noncollagenous matrix of the material (which is less stiff than its elastin fiber constituent). To determine the noncollagenous matrix response, we propose to use the isotropic neo-Hookean model according to

$$\Psi_{iso} = \frac{c}{2} (\mathbf{I}_1 - 3),$$

where $c > 0$ is a stresslike material parameter. However, to model the (isotropic) noncollagenous matrix material any Ogden-type elastic material may be applied [18].

According to morphological findings at highly loaded tissues, the families of collagen fibers become straighter and the resistance to stretch is almost entirely due to collagen fibers (the tissue becomes stiff). Hence, the strain energy stored in the collagen fibers is taken to be governed by the polyconvex (anisotropic) function

$$\Psi_{aniso} = \frac{k_1}{2k_2} \left\{ \exp \left[ k_2 (\mathbf{I}_1^* - 1)^2 \right] - 1 \right\} + \frac{k_3}{2k_4} \left\{ \exp \left[ k_4 (\mathbf{I}_2^* - 1)^2 \right] - 1 \right\}$$

where $k_1 > 0, k_3 > 0$ are stresslike material parameters and $k_2 > 0, k_4 > 0$ are dimensionless parameters. According to Eqs. 2, 4, and 5, the collagen fibers do not influence the mechanical response of the tissue in the low stress domain. Because of the crimp structure of collagen fibers, we assume that they do not support compressive stresses, which implies that they are inactive in compression. Hence the relevant part of the anisotropic function (Eq. 5) is omitted for this case. If, for example, $\mathbf{I}_1^* > 1$ and $\mathbf{I}_2^* > 1$, then the soft tissue responds similarly to a rubberlike (purely isotropic) material described by the energy function (Eq. 4). However, in extension, that is when $\mathbf{I}_1^* > 1$ or $\mathbf{I}_2^* > 1$, and the collagen fibers are active and energy is stored in the fibers.

Equation 1 enables the Cauchy stress tensor, denoted $\sigma$, to be derived in the decoupled form

$$\sigma = \sigma_{vol} + \mathbf{\tilde{\sigma}} \quad \text{with} \quad \sigma_{vol} = p \mathbf{I}, \quad \mathbf{\tilde{\sigma}} = 2J^{-1} \text{dev} \left( \hat{F} \frac{\partial \Psi}{\partial \mathbf{C}} \hat{F}^T \right)$$

with the volumetric contribution $\sigma_{vol}$ and the isochoric contribution $\mathbf{\tilde{\sigma}}$ to the Cauchy stresses. In the stress relation (Eq. 6), $p = dU/dJ$ denotes the hydrostatic pressure and $\text{dev}(\bullet)$ furnishes the deviatoric operator in the Eulerian description. The operator is defined as $\text{dev}(\bullet) = (\bullet) - \frac{1}{3}(\bullet : \mathbf{I}) \mathbf{I}$, so that $\text{dev}(\bullet) : \mathbf{I} = 0$.

Using the additive split (Eq. 2) and particularizations (Eqs. 4 and 5), we get with the third part of Eq. 6 an explicit constitutive expression for the
isochoric behavior of soft connective tissues in the Eulerian description, i.e.,

\[ \bar{\sigma} = c \text{ dev } \bar{b} + \sum_{x=1}^{2} 2 \bar{\Psi}_x \text{ dev } (a_x \otimes a_x) \]  

(7)

where \( \bar{b} = \bar{F} \bar{F}^T \) denotes the modified left Cauchy-Green tensor,

\[ \bar{\Psi}_1 = \frac{\partial \Psi_{\text{aniso}}}{\partial I_1} = k_1(I_1^* - 1) \exp \left[ k_2(I_1^* - 1)^2 \right] \]  

(8)

\[ \bar{\Psi}_2 = \frac{\partial \Psi_{\text{aniso}}}{\partial I_2} = k_3(I_2^* - 1) \exp \left[ k_4(I_2^* - 1)^2 \right] \]  

(9)

are (scalar) response functions, and \( a_x = \bar{F} a_{0x} \), \( x = 1, 2 \), are the Eulerian counterparts of the unit vectors \( a_{0x} \).

The specific form of the proposed constitutive equation (Eq. 7) requires the five material parameters \( c, k_1, k_2, k_3, k_4 \) whose interpretations can be partly based on the underlying histological structure, i.e., matrix and collagen of the tissue. Note that in Eq. 7, orthotropic \((k_1 = k_3, k_2 = k_4)\), transversely isotropic \((k_1 = 0 \text{ or } k_3 = 0)\), and isotropic hyperelastic descriptions \((k_1 = k_3 = 0)\) at finite strains are included as special cases.

### 10.11.5 REPRESENTATIVE EXAMPLE: A MODEL FOR THE ARTERY

In this section we describe a model for the passive state of the healthy and young artery (no pathological changes in the intima, which is the innermost arterial layer frequently affected by atherosclerosis) suitable for predicting three-dimensional distributions of stresses and strains under physiological loading conditions with reasonable accuracy. It is a specification of the constitutive framework for soft tissues stated in the previous section. For an adequate model of arteries incorporating the active state (contraction of smooth muscles), see Reference [22]. For a detailed study of the mechanics of arterial walls, see the extensive review in Reference [13].

Experimental tests show that the elastic properties of the media (middle layer of the artery) and adventitia (outermost layer of the artery) are significantly different [31]. The media is much stiffer than the adventitia. In particular, in the unloaded configuration the mean value of Young's modulus for the media, for several pig thoracic aortas, is about an order of magnitude higher than that of the adventitia [32]. In addition, the arterial layers have different physiological tasks, and hence the artery is modeled as a thick-walled elastic circular tube consisting of two layers corresponding to the...
media and adventitia. In a young nondiseased artery the intima (innermost layer of the artery) exhibits negligible wall thickness and mechanical strength.

Each tissue layer is treated as a composite reinforced by two families of collagen fibers which are symmetrically disposed with respect to the cylinder axis. Hence, each tissue layer is considered as cylindrically orthotropic (already postulated in the early work [20]) so that a tissue layer behaves like a so-called balanced angle-ply laminate. We use the same forms of strain energy functions (Eqs. 4 and 5) for each tissue layer (each layer responds with similar mechanical characteristics) but use a different set of material parameters. Hence, Eq. 2 takes on the specified form

$$\Psi_M = \frac{c_M}{2}(\bar{I}_1 - 3) + \frac{k_{1M}}{2k_{2M}} \sum_{z=1}^{2} \left\{ \exp \left[ k_{2M}(\bar{I}_z^*-1)^2 \right] - 1 \right\}$$

$$\Psi_A = \frac{c_A}{2}(\bar{I}_1 - 3) + \frac{k_{1A}}{2k_{2A}} \sum_{z=1}^{2} \left\{ \exp \left[ k_{2A}(\bar{I}_z^*-1)^2 \right] - 1 \right\}$$

We end up with a two-layer model incorporating six material parameters, three for the media $M$, i.e., $c_M, k_{1M}, k_{2M}$, and three for the adventitia $A$, i.e., $c_A, k_{1A}, k_{2A}$.

The invariants associated with the anisotropic parts of the two tissue layers are defined by $\bar{I}_{ij} = \hat{C} : A_{ij}$ and $\bar{I}_{2j} = \hat{C} : A_{2j}, j = M, A$. The structure tensors $A_{ij}, A_{2j}$ are given by

$$A_{ij} = a_{01j} \otimes a_{01j}, \quad A_{2j} = a_{02j} \otimes a_{02j}, \quad j = M, A.$$  

Employing a cylindrical coordinate system, the components of the unit (direction) vectors $a_{01j}$ and $a_{02j}$ read in matrix notation

$$[a_{01j}] = \begin{bmatrix} \cos \beta_j \\ \sin \beta_j \end{bmatrix}, \quad [a_{02j}] = \begin{bmatrix} \cos \beta_j \\ -\sin \beta_j \end{bmatrix}, \quad j = M, A,$$

and $\beta_j, j = M, A$, are the angles between the collagen fibers and the circumferential direction in the media and adventitia (see Figure 10.11.3). Small components of the (collagen) fiber orientation in the radial direction, as, for example, reported for human brain arteries [5], are neglected.

### 10.11.5.1 Residual Stresses

It has been known for some years that arteries which are excised from the body and not subjected to any loads are not stress-free (or strain-free) [28]. If, for example, the media and adventitia are separated and cut in a radial
direction, the two arterial layers will spring open to form open (stress-free) sectors, which, in general, have different opening angles (see, for example, the experimental studies [29] for bovine specimens). In general, the residual stress state is very complex, and residual stresses (strains) in the axial direction may also occur. Note that residual stresses result from growth and remodeling mechanisms [21, 24].

By considering the arterial layers as circular cylindrical tubes, we may characterize the reference (stress-free) configuration of one arterial layer as a circular sector, as shown in Figure 10.11.4. For each arterial layer of the blood vessel a certain opening angle $\alpha$ can be found by experimental methods. The importance of incorporating residual stresses associated with the load-free

![Figure 10.11.3](image1.png)

**FIGURE 10.11.3** Load-free configuration of an idealized artery modeled as a thick-walled circular tube consisting of two layers, the media and adventitia.

![Figure 10.11.4](image2.png)

**FIGURE 10.11.4** Cross-sectional representation of one arterial layer at the reference (stress-free) and load-free (stressed) configurations.
(but stressed) configuration into the computation has been emphasized in, for example, References [4, 12]. Consideration of residual strains has a strong influence on the global pressure and radius response of arteries and also on the stress and strain distributions across the deformed arterial wall. For analytical studies of residual stresses see, for example, the works [14, 22], which contain further references.

Therefore, it is essential to incorporate the residual stresses inherent in many biologic tissues. One possible approach to consideration of the influence of residual stresses on the overall three-dimensional stress behavior is to measure the strain energy from the load-free (stressed) configuration and to include the residual stresses [19]. Another approach is to start with the energy function relative to the stress-free (and fixed) configuration, as assumed in the presented models, and determine the deformation required to reach the load-free (stressed) configuration. Figure 10.11.4 shows the cross-sectional representation of one arterial layer at the load-free configuration obtained from the reference configuration by pure bending.

With the condition of incompressibility, the radius $r$ of an arterial layer in the load-free configuration may be computed from the radius $R$ of the associated reference configuration as [12]

$$r = \sqrt{\frac{R^2 - R_i^2}{k \lambda_z} + r_i^2}, \quad k = \frac{2\pi}{2\pi - \alpha}$$

where $r_i, R_i$ are the internal radii associated with the two configurations. The (constant) axial stretch is denoted by $\lambda_z$, and the parameter $k$ is a convenient measure of the tube opening angle in the stress-free configuration.

10.11.6 IDENTIFICATION OF THE MATERIAL PARAMETERS

Preferred directions in soft tissues are well specified by the orientation of prolate cell nuclei. They can be identified in microphotographs of appropriately stained histological sections. By visual inspection there exists a high directional correlation between smooth muscle cells and collagen fibers. Hence, the bell-shaped distribution of collagen fiber orientations may be obtained from an image-processing analysis of stained histological sections. The angle $\beta$ (and thus the unit vectors $a_{01}, a_{02}$) may be identified as the mean value of the corresponding statistical distribution.

Values of the material parameters associated with the model for soft tissues are then obtained by fitting the equations to the experimental data of the soft tissue of interest by using standard nonlinear fitting algorithms, such as the

\[\text{Holzapfel}\]
Levenberg–Marquardt algorithm. If the mean values of the orientation of cell nuclei (collagen fiber) may not be identified experimentally, it is suggested to treat the collagen fiber orientations as additional (phenomenological) “material” parameters.

10.11.7 HOW TO USE THE MODEL

The energy functions are well suited for use in nonlinear finite element software, which enables complex boundary-value problems to be solved. Aspects of finite element implementation and numerical analysis of the model are presented in Reference [11]. Furthermore, computations may be carried out with some of the commercially available mathematical software packages such as Mathematica or Maple, which allow symbolic computation. Based on Mathematica, in Reference [12] a numerical technique for solving the bending, axial extension, inflation, and torsion problem of an artery is described.

10.11.8 TABLE OF PARAMETERS

Values of the parameters correspond to the Eqs. 10 and 11 and are given for a representative carotid artery from a rabbit (experiment no. 71 in Reference [7]). The material parameters \( c \), \( k_1 \), \( k_2 \) and the (mean) values of collagen fiber angles \( \beta \) are summarized in Table 10.11.2.

In the adventitia many collagen fibers run closer to the axial direction of the artery, whereas in the media the collagen fibers tend to run around the circumference. The fiber angles \( \beta \) are meant to be associated with the reference (stress-free) configuration, and we assume that they are the same in the load-free (stressed) configuration. The difference in fiber angle which occur due to bending to the load-free configuration (see Figure 10.11.4) is small, so that it has a negligible influence on the stress–strain analysis of arteries.

<table>
<thead>
<tr>
<th>TABLE 10.11.2 Parameters for a Carotid Artery from a Rabbit (Experiment no. 71 in Reference [7]) in reference to Eqs. 10 and 11</th>
</tr>
</thead>
<tbody>
<tr>
<td>Media</td>
</tr>
<tr>
<td>( c_M = 3.0000 ) (kPa)</td>
</tr>
<tr>
<td>( k_{1M} = 2.3632 ) (kPa)</td>
</tr>
<tr>
<td>( k_{2M} = 0.8393 ) (–)</td>
</tr>
<tr>
<td>( \beta_M = 29.0^\circ )</td>
</tr>
</tbody>
</table>
By using a wall thickness of 0.39 mm (adopted from Reference [3]) and assuming that the media occupies two-thirds of the arterial wall thickness, the parameters in Table 10.11.2 predict the characteristic orthotropic behavior of a carotid artery under combined bending, inflation, axial extension, and torsion, as documented in Reference [12].

REFERENCES


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