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Journal of Biomechanics 41 (2008) 1458–1466

JOURNAL OF BIOMECHANICS

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A variational constitutive model for soft biological tissues

Tamer El Sayed[a], Alejandro Mota[a], Fernando Fraternali[b], Michael Ortiz[a,*]

[a]*Division of Engineering and Applied Science, California Institute of Technology, Pasadena, CA 91125, USA*
[b]*Department of Civil Engineering, University of Salerno, 84084 Fisciano (SA), Italy*

Accepted 18 February 2008

Abstract

In this paper, a fully variational constitutive model of soft biological tissues is formulated in the finite strain regime. The model includes Ogden-type hyperelasticity, finite viscosity, deviatoric and volumetric plasticity, rate and microinertia effects. Variational updates are obtained via time discretization and pre-minimization of a suitable objective function with respect to internal variables. Genetic algorithms are used for model parameter identification due to their suitability for non-convex, high dimensional optimization problems. The material behavior predicted by the model is compared to available tests on swine and human brain tissue. The ability of the model to predict a wide range of experimentally observed behavior, including hysteresis, cyclic softening, rate effects, and plastic deformation is demonstrated.

Keywords: Biological tissues; Variational; Ogden; Plasticity; Shear; Volumetric damage; Finite strain; Cavitation; Genetic algorithms; Hyper elasticity; Viscosity; Microinertia

1. Introduction

Soft biological tissues exhibit complex mechanical behavior, characterized by large strains, rate-sensitivity, hysteresis, solid/fluid behavior, residual stresses, and permanent deformation. They consist of cells, extracellular components, vascular network, and water. One important example is brain tissue, which exhibits extremely soft behavior and is often modeled using hyperelastic or hyperviscoelastic constitutive equations (Prange and Margulies, 2002; Miller and Chinzei, 1997, 2002; Miller et al., 2006; Meaney, 2003; Brands et al., 2002; Velardi et al., 2006). Hyperelastic or viscoelastic anisotropic models for arterial tissues have been proposed by Holzapfel, Ogden and Gasser in a series of recent studies (Holzapfel et al., 2000; Holzapfel, 2001; Gasser et al., 2006). Some authors have analyzed plasticity, hysteresis, permanent deformation and biphasic (solid/fluid) behavior of soft biological tissues (Bergström and Boyce, 2001; Gasser and Holzapfel, 2002; Franceschini et al., 2006). Variationally consistent finite viscoelastic models for rubber-like materials have been proposed by several authors (Govindjee and Reese, 1997; Reese and Govindjee, 1998; Fancello et al., 2006). It is often assumed that biological tissues exhibit mechanical *anisotropy*, due to the presence of reinforcing fibers in the extracellular matrix (Prange and Margulies, 2002; Meaney, 2003; Ogden, 2003; Gasser et al., 2006; Velardi et al., 2006). Nevertheless, some authors argue that "very soft tissue do not bear mechanical load and do not exhibit directional structure (provided that a large enough sample is considered ...)" (see Miller, 2005 and references therein).

Two of the major recognized causes of physiological damage to living tissues are tensile and shearing structural failures caused by relative motions within the tissues (Stoyanovski and Grozeva, 2005). For example, referring to head traumas, linear forces resulting from straight ahead acceleration–deceleration impact can be associated with focal lesions and tensile (cavitation) injuries. Those may occur at the site of contact (coup injury) or distant, usually opposite the site of contact (contre-coup injury) (Lubock and Goldsmith, 1980; Hardy et al., 1994; Nusholtz et al., 1996; Brennen, 2003; Johnson and Young,

*Corresponding author. Tel.: +1 626 395 4530.
E-mail addresses: tamer@caltech.edu (T. El Sayed), ortiz@aero.caltech.edu (M. Ortiz).

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doi:10.1016/j.jbiomech.2008.02.023

2006). Rotational forces produced by rotational acceleration–deceleration traumas can instead lead to shearing injuries in the brain parenchyma, between tissue planes of varying densities (diffuse axonal injury) (Strich, 1956; Adams and Graham, 1984; Perles and Rewcastle, 1967).

In view of all the above requirements in the modeling of soft biological tissues, herein we present a fully variational phenomenological constitutive model with the ability to capture all of the following:

- Rate and microinertia effects.
- Complex viscous behavior via a flexible number of viscoelastic mechanisms capable of representing finite viscosity.
- Hysteresis, deviatoric and volumetric plasticity.
- Strong non-linearity, different behavior in tension and compression, preconditioning and cyclic softening.
- Thermal softening and adiabatic heating via thermal updates.
- Void growth and shrinkage during the process of cavitation (El Sayed et al., 2007).

The rheological representation of the model consists of an elastoplastic network acting in parallel with several viscoelastic networks. Quasi-incompressible Ogden-type potentials govern the elastic behavior, both in the elastoplastic (which accounts for both deviatoric and volumetric plasticity) and viscoelastic branches. The Ogden model was chosen because it can reduce to either the Neo-Hookean model ($N=1$, $\alpha=2$), or the Mooney–Rivlin model ($N=2$, $\alpha_1=2$, $\alpha_2=-2$). This indeed shows that the Ogden model is a practical choice in this framework due to its generality and capability to reproduce simpler existing models, if needed, in such an easy fashion. Cyclic stress softening is reproduced through a combination of elastoplastic and viscoelastic responses. Deviatoric plasticity allows for representing shearing-type injuries, while volumetric plasticity is intended to reproduce cavitation injuries, being related to the expansion of spherical voids or bubbles in a plastically incompressible matrix (El Sayed et al., 2007). As the pressure reaches a critical value in tension, the material is allowed to yield and exhibit volumetric strain softening. Ortiz and Molinari (1992) analyzed the dynamic expansion of a single spherical void in an infinite rigid plastic medium under the action of remote hydrostatic tension. They reported that if the initial void radius increases by at least one order of magnitude, the void growth is dominated by microinertial effects, whereas the effect of rate dependence of the material and the plastic dissipative effects play a secondary role. High accelerations sustained by the material particles in the vicinity of voids result in significant inertial effects, particularly for materials with low strain-rate sensitivity (Molinari and Mercier, 2001). The presented model accounts for microinertia (see Section 2.4). This particular feature is utilized in the simulation of traumatic brain injury (El Sayed et al., 2007) where volumetric damage as a result of a high strain rate impact is evident. Viscous deformation of the viscoelastic networks allows one to reproduce transient deviatoric and volumetric damage to the tissue.The model is formulated in an isotropic framework and is intended to mimic the presence of reinforcing fibers through regional dependence of mechanical properties (Prange and Margulies, 2002).

The number of model parameters is a function of the number of active Ogden terms and relaxation mechanisms, and therefore a significant number of parameters may need to be identified. This requires the use of advanced techniques for the calibration of model parameters based on experimental data, hence we propose a procedure based on genetic algorithms (GA), which have been proved to be well suited for multimodal non-convex optimization (Schmitt, 2004). Several sets of experimental data are compared with model predictions, showing the ability of the model to reproduce the observed behavior of soft biological tissues. Monotonic and cyclic tests on brain tissue are examined, which involve complex behavior such as cyclic hysteresis, cyclic softening, rate effects, and plastic deformation.

The current constitutive model is applied to the finite element simulation of diffuse axonal injury and cavitation damage associated with traumatic brain injury in El Sayed et al. (2007).

2. Constitutive model

Let $\boldsymbol{F}$ denote the deformation gradient at an arbitrary point of the material, and let

$$\boldsymbol{F}=\boldsymbol{F}^e\boldsymbol{F}^p=\boldsymbol{F}^e_1\boldsymbol{F}^v_1=\cdots=\boldsymbol{F}^e_M\boldsymbol{F}^v_M \tag{2.1}$$

be its multiple multiplicative decomposition, where M is a positive integer that defines the number of viscoelastic (Maxwell-type) relaxation networks that the model possesses, and that act in parallel with an elastoplastic equilibrium network; $\boldsymbol{F}^e,\boldsymbol{F}^e_1,\ldots,\boldsymbol{F}^e_M$ are the elastic parts of $\boldsymbol{F}$; $\boldsymbol{F}^p$ is the plastic deformation gradient; and $\boldsymbol{F}^v_1,\ldots,\boldsymbol{F}^v_M$ are the viscous deformation gradients.

The thermo-mechanical behavior of the material derives from the additive potential

$$A=A^{ep}(\boldsymbol{F},\boldsymbol{F}^p,\boldsymbol{Z}^p,T)+A^{ve}(\boldsymbol{F},\boldsymbol{F}^v_i,\boldsymbol{Z}^v_i), \tag{2.2}$$

where A^{ep} and A^{ve} are elastoplastic and viscoelastic contributions, correspondingly; $\boldsymbol{Z}^p$ is a vector of plastic internal variables; $\boldsymbol{Z}^v_i$ are vectors of viscous internal variables ($i=1,\ldots,M$); and T is the absolute temperature.

The first Piola–Kirchhoff stress $\boldsymbol{P}$, and the thermodynamic forces $\boldsymbol{Y}^p$ and $\boldsymbol{Y}^v_i$ conjugate to $\boldsymbol{Z}^p$ and $\boldsymbol{Z}^v_i$ follow from

$$\boldsymbol{P}=\frac{\partial A}{\partial \boldsymbol{F}}, \tag{2.3}$$

$$\boldsymbol{Y}^p=-\frac{\mathrm{d}A}{\mathrm{d}\boldsymbol{Z}^p}, \tag{2.4}$$

$$\boldsymbol{Y}^v_i=-\frac{\mathrm{d}A}{\mathrm{d}\boldsymbol{Z}^v_i}. \tag{2.5}$$