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A Model for Kidney Tissue Damage under High Speed Loading

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In a medical procedure to comminute kidney stones the patient is subjected to hypersonic waves focused at the stone. Unfortunately such shock waves also damage the surrounding kidney tissue. We present here a model for the mechanical response of the soft tissue to such a high speed loading regime.

The material model combines shear induced plasticity with irreversible volumetric expansion as induced, e.g., by cavitating bubbles. The theory is based on a multiplicative decomposition of the deformation gradient and on an internal variable formulation of continuum thermodynamics. By the use of logarithmic and exponential mappings the stress update algorithms are extended from small-strain to the finite deformation range. In that way the time-discretized version of the porous-viscoplastic constitutive updates is described in a fully variational manner.

By numerical experiments we study the shock-wave propagation into the tissue and analyze the resulting stress states. A first finite element simulation shows localized damage in the human kidney.

1 Introduction

In extracorporeal shock-wave lithotripsy (SWL) high intensity sound waves are generated outside the patient and focused onto the kidney stone. The compressive wave is thought to comminute the stone but it also causes shearing along its way in the kidney tissue. This may be one reason for the observed kidney injuries. Moreover, the compressive front is followed by a "tension tail", i.e., a negative pressure, see Figure 1a. In this way cavitation is induced, cf. [3]. Bubbles nucleate, expand up to several micrometer in size and finally collapse. During bubble expansion the surrounding vessels and capillaries dilate and may rupture causing damage in the kidney tissue material.

2 Theoretical Framework

The equilibrium response of the solids considered here is characterized by a free-energy density per unit undeformed volume $A = A(\boldsymbol{F}, \boldsymbol{F}^p, \varepsilon^p, \theta^p)$, where $\boldsymbol{F}$ is the deformation gradient, $\boldsymbol{F}^p$ and $\boldsymbol{F}^e = \boldsymbol{F}\boldsymbol{F}^{p-1}$ are the plastic and elastic components, respectively. The plastic deformation rate is assumed to obey the flow rule

$$\dot{\boldsymbol{F}}^p \boldsymbol{F}^{p-1} = \dot{\varepsilon}^p \boldsymbol{M} + \dot{\vartheta}^p \boldsymbol{N} \tag{1}$$

where here and above $\dot{\varepsilon}^p$ and $\dot{\vartheta}^p$ are scalar (internal) variables accounting for irreversible deformation subjected to the constraints

$$\dot{\varepsilon}^p \geq 0 \quad \text{and} \quad \dot{\vartheta}^p \geq 0. \tag{2}$$

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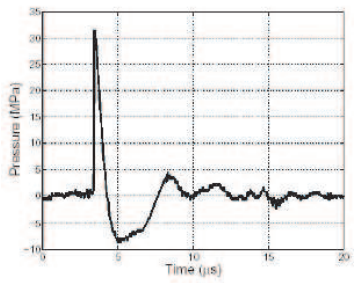
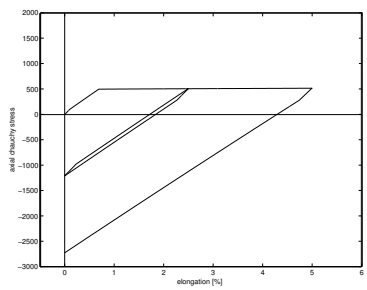
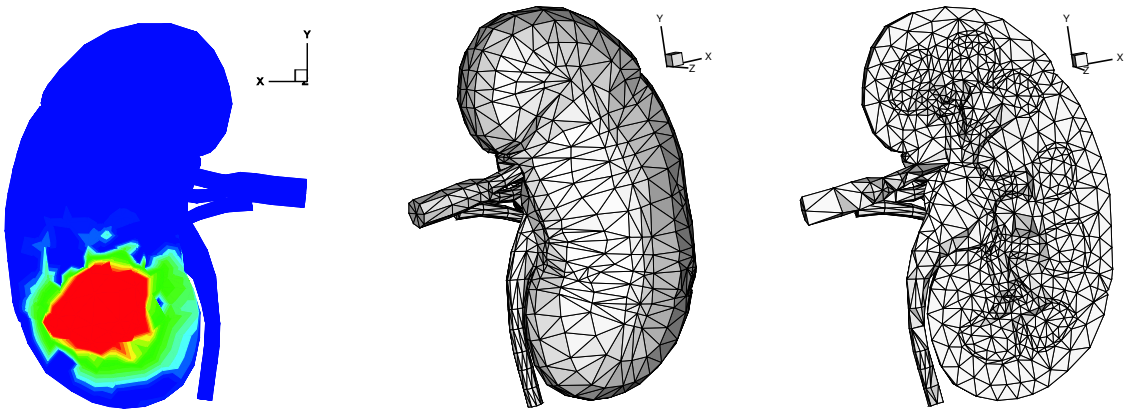


(a) Pressure vs. time in SWL



(b) Cauchy stress vs. elongation

Fig. 1 Typical SWL-pressure wave and stresses computed in two cycles of a uniaxial strain test.



(a) damaged kidney surface (b) finite element mesh (c) meridional section

Fig. 2 Computed field of irreversible volumetric expansion and anatomically correct finite element mesh of the kidney.

The tensors $\boldsymbol{M}$ and $\boldsymbol{N}$ set the direction of the deviatoric and volumetric plastic deformation rates and are to be determined as part of the solution. Motivated by experimental observations we restrict here to irreversibility in tension only, see Figure 1b.

The free-energy density is assumed to decompose additively into deviatoric and volumetric components of elastic and stored plastic energy densities, $W(\boldsymbol{F}^e)$ and $W^p(\varepsilon^p, \theta^p)$. The elastic strain-energy density is expressed in terms of the logarithmic elastic strain $\boldsymbol{\epsilon}^e = \frac{1}{2}\log(\boldsymbol{F}^{eT}\boldsymbol{F}^e)$. The deviatoric part of the dissipated energy function is solely a function of the effective deviatoric plastic strain ε^p, whereas the volumetric part of the dissipated energy function is attributed directly to the bubble growth. Using a simple dilute distribution of (empty) spherical bubbles we link the mechanism of bubble expansion to the macroscopic material parameter. The local volume fraction of bubbles is $f = N\frac{4}{3}\pi a^3$, where a is current average bubble radius and N is the referential bubble density. The infinitesimal initial and current material volumes V_0 and V are related through $V = \det \boldsymbol{F}\ V_0$. To express $\det \boldsymbol{F}^p$ in terms of the bubble radius we state

$$\det \boldsymbol{F}^p = V(\det \boldsymbol{F}^e V_0)^{-1} = (1 - f_0)/(1 - f). \tag{3}$$

Aside of conventional newtonian viscosity and rate sensitivity in the plastic deformations we consider the micro-inertia due to rapidly expanding bubbles as a dissipative energy term. The total micro-inertia attendant to the growth of the bubble ensemble is assumed to be the sum of the kinetic energies due to the expansion of each individual bubble. The latter can be computed readily in terms of the radius and may be transformed to fit in a variational time-discretization scheme.

3 Numerical Analysis

The flow rule is discretized in time using an exponential mapping technique, $\boldsymbol{F}^p_{n+1} = \exp\left(\Delta\varepsilon^p \boldsymbol{M} + \Delta\vartheta^p \boldsymbol{N}\right) \boldsymbol{F}^p_n$. We update the internal state variables, ε^p_{n+1} and ϑ^p_{n+1}, and simultaneously determine the incremental direction of plastic flow, $\boldsymbol{M}$ and $\boldsymbol{N}$, by recourse to the variational formulation of Ortiz and Stainier [2]. To this end, we introduce in every time interval $\Delta t = t_{n+1} - t_n$ an incremental objective function which includes the free energy of the material as well as the stored plastic energy, the micro-kinetic energy and the dual kinetic potential. These energy contributions compete among themselves to optimize the incremental objective function.

In our finite element computation we distinguish functional (damage sensitive) tissue and non-functional tissue (as, e.g., the ureter and main blood vessels) modelled with the presented material and a non-linear elastic material law, respectively. For time integration an explicit Newmark scheme is used. The kidney was subjected to a 1μs pressure wave modelled as distributed force on the boundary and then released. Figure 2a shows the computed field of irreversible volumetric expansion on the outside of the kidney. The localized damage correspondents well to the typical bruising observed in animal experiments [1].

References

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