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MODELING AND NUMERICAL SIMULATION OF KIDNEY DAMAGING SIDE EFFECTS IN SHOCK WAVE LITHOTRIPSY

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ABSTRACT

An example for a classical medical procedure with damaging side effects on soft tissue is the non-invasive comminution of kidney stones during Shock-Wave-Lithotripsy (SWL). Even though this procedure is widely applied, the physics behind this medical treatment, in particular the question of how the injuries in the surrounding kidney tissue arise, is still under investigation. We contribute to the solution of this problem with a constitutive model for a soft elastic material with a certain time and load dependent porosity. The specific phenomena of cavitation induced oscillating bubbles is modeled as an evolution of spherical pores within the soft tissue. By means of large scale finite element analyzes (FEA) we study the shock-wave propagation into the kidney tissue and analyze the resulting stress states. The numerical simulations predict localized damage in the human kidney in the same regions as in animal experiments.

During a SWL treatment a number of high intensity pressure waves (shock waves) are generated outside the patient and focused on the stone within the kidney. Compressive waves with a typical peak of 30-80 MPa induce cracks and, by internal reflection, fragmentation of the stone. However, along its way the focused wave causes shearing in the kidney tissue which may be *one* reason for the observed kidney injuries. The compressive shock front is followed by a "tension tail", i.e., a negative pressure up to 10 MPa. In this way bubble cavitation is induced which is thought to assist in stone fragmentation. Bubbles nucleate, expand up to several micrometer in size and finally collapse. During bubble expansion the surrounding vessels and capillaries dilate and may rupture. This mechanism is considered to be *another* reason for the kidney injuries.

In our numerical simulation of SWL we do not analyze the micro-histological changes of the kidney tissue in detail, this would require an other scale of modeling. Instead we summarize the morphological lesions like hemorrhage, rupture of small arteries and tearing of peri-tubular capillaries, as damage in the sense of irreversible deformations. The basic idea is to model the tissue like a general (two-parametric) elastic material with (initially very small) bubbles and to study the oscillation of the bubbles of different sizes. These oscillations result from an interplay of elastic, surface and kinetic energy contributions with

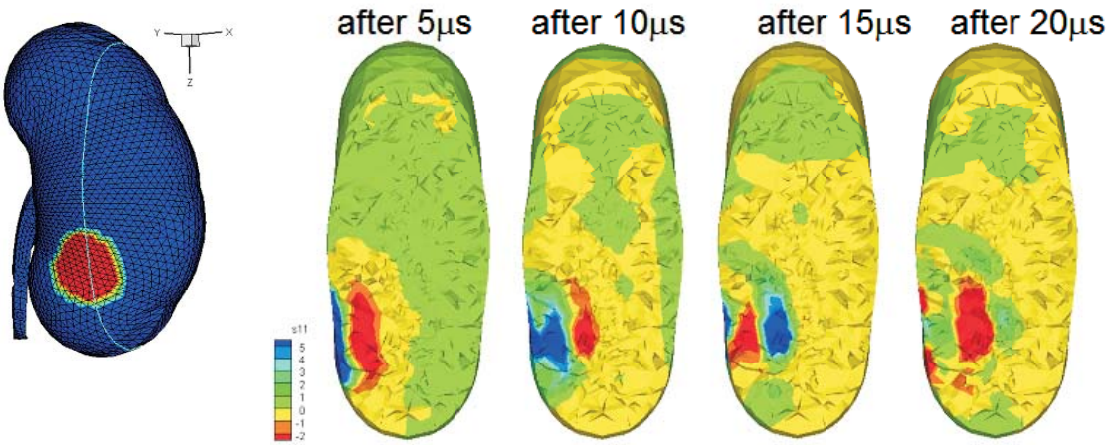

Figure 1: ESWL impulse traveling through the kidney (blue: pressure, red: tension).

the applied shock wave loading. From the results we derive a scalar criteria (in the sense of an internal variable formulation) which accounts for the macroscopic irreversible effects. The equilibrium response of the tissue is characterized by a (time-discretized) effective energy function which includes elasticity, dissipated energy, micro-inertia and viscosity. These contributions compete among themselves and the optimal internal process is that one which minimizes the effective energy function, see [3] for details.

For FEA we built anatomical correct two- and three-dimensional meshes of a human kidney using geometrical data from image processing. The kidney is modeled without a stone. This approach is backed by experimental studies, cf. [1]. We distinguish several regions of functional tissue, each with different sensitivity and elasticity. In contrast to former results, cf. [4], the kidney is now embedded in an acoustic material to simulate the surrounding body tissue. By large scale simulations we study the shock-wave propagation into the kidney tissue, adapt unknown material parameters and analyze the resulting stress states. Figure 1 shows the stress propagating into the kidney with magnitudes of 2-5MPa. These values corresponds very well to observations of [2] where a peak negative pressure of 1.5-3.5MPa is considered to be the threshold for damage caused by SWL. The FE simulations predict localized damage in the human kidney in the same regions as observed in animal experiments. Furthermore, the numerical results suggest that in first instance the pressure amplitude of the shock wave impulse (and not so much its exact time-pressure profile) is responsible for damaging the kidney tissue.

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