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MODELING OF SOFT TISSUES DEFORMATION

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MODELING OF ORGAN DEFORMATION - WHY WE NEED IT FOR SURGICAL MANIPULATOR?

Surgical manipulator (or robot) has showed several successful applications to bone and brain surgeries. However, after them, we come to a barrier: most of other organs are globally translatable and locally deformable. We consider that it is safe to assume that the global transform is able to be suppressed by fixation of the organs in most of cases. However, we think that the local deformation is not negligible in number of cases. If the manipulator is full- or semi-automatic, we must adapt its control according to the deformation [2].

Nowadays several 3D spatial pointing devices are available. They offer the coordinate of sampled points on the surface, or inside of the organ if we allow invasion. However, it requires another technique to utilize information of sparse sampling points to the control of the robot.

The intraoperative imagings can supply the dense information. However, most of medical imaging methods need tens of seconds to even tens of minutes to obtain a set of 3D image. Thus, if we think of using the image measurement, we also have to consider the delay: an answer is the prediction of the deformation based on its dynamic/kinetic model.

STANDARD BIPHASIC MODELS OF SOFT TISSUES

Presently it is widely accepted that soft tissues are ***biphasic*** materials consisting of solid deformable porous matrix and penetrating fluid. Based on this concept, in 80's, the first biphasic models of soft cartilage tissues were reported ([4] and references cited therein).

Let's assume, that the 'very' soft tissue is a biphasic mixture of solid porous matrix and fluid. We also assume, that ***components are chemically inert*** and the compressibility of the tissue is a result of fluid flow through the pores only - ***the solid and fluid themselves are incompressible.*** This assumptions will allow as to use the special arrangement of the theory of mixtures for incompressible constituents. The governing equations of such biphasic continuum are as follows:

Continuity: $$\nabla(\phi^s v^s + \phi^f v^f) = 0 \quad (1)$$

Equilibrium: $$\nabla \sigma^\alpha + \Pi^\alpha = 0 \quad (2)$$

where:

ϕ^α - α phase content,
v^α - velocity of α phase,
σ^α - α phase Cauchy stress tensor,
Π^α - diffusive momentum exchange between phases.

When writing the equilibrium equation we neglected inertial body forces.

If we assume additionally, that the ***fluid is inviscid*** and that ***the diffusive momentum exchange is proportional to the relative velocity between phases***, the constitutive equations are as follows:

$$\sigma^s = -\phi^s p I + \sigma^s_E \quad (3)$$

$$\sigma^f = -\phi^f p I \quad (4)$$

$$\Pi^s = -\Pi^f = -\nabla\phi^f + K(v^f - v^f) \quad (5)$$

where:

p - apparent pressure,
K - diffusive drag coefficient function,
σ^s_E - Cauchy stress tensor of the solid phase.
I - the identity tensor,

The diffusive drag coefficient K is not in general constant. The investigations summarized in [4] suggest that K is strongly (exponentially) dependent on strain.

The accepted way to relate the stresses to the deformation is by means of the Helmholtz free energy:

$$\sigma^s_{E_{ij}} = \rho \frac{\partial U}{\partial E_{KL}} \frac{\partial x_i}{\partial X_K} \frac{\partial x_j}{\partial X_L} \quad (6)$$

where:

U - the Helmholtz free energy function of the solid phase,
ρ - apparent density of the solid phase,
$\frac{\partial x_j}{\partial X_L}$ - deformation gradient of solid phase,
E_{KL} - Green's strain tensor (relative to original configuration) of the solid phase.

This approach is based on the assumption, that the solid phase stress depends only on the current deformation. Therefore there is no energy dissipation in the solid, but ***the dissipation comes from interactions between phases only.*** As it will be shown later, this assumption is a very restrictive one and cannot be maintained, if the intended purpose is that of accurate modeling of soft tissue deformation behavior.

METHODS OF MODEL VALIDATION

Confined compression experiment

The confined compression experiment [4] (Fig.1) has become one of the standards for biphasic models validation and material constants determination. In this experiment, the tissue is confined in a cylindrical container, and compressed unidirectional through an ideally porous filter. The creep or stress relaxation phenomena can be observed and compared to the model predictions.

The confined compression test has been performed on various cartilage tissues and showed very good agreement with model predictions [4]. Sofar we've surveyed, no this kind of experiment conducted on 'very' soft tissues has been reported.

Unconfined compression experiment

In unconfined compression experiment (Fig. 2) the cylindrical sample of tissue is axially compressed between two impermeable

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platens. Figure 3 shows an experimental measurement from literature, which compressed cylindrical samples from human brain [3]. It showed a clear dependence of the stress values on the loading velocity.

COLLAPSE OF 'STANDARD' BIPHASIC MODEL

However, we found out that this arises a crisis for the so-called standard biphasic model that we've surveyed herein. It can be shown, that the ratio of the instantaneous stress (after sudden movement of the upper platen) to the equilibrium stress (after sufficiently long time following load application), as predicted by the biphasic theory, **cannot be larger than** $3/2(1+\nu) \in \langle 1, 1.5 \rangle$, where ν is the Poison's ratio of the solid phase. This poses a severe limit on the stress dependence on loading velocity. It is doubtful if the standard biphasic model could be maintained in the case of cartilage tissue. Obviously it cannot be accepted for very soft tissues, such as brain.

Why the described above the standard biphasic model describes excellently the tissue behavior in the confined compression experiment and simultaneously fails completely to account for the behavior under the unconfined compression conditions? In the confined compression test, the solid matrix moves through the stationary (or almost stationary) fluid. The solid's velocity is therefore equal to the relative velocity of phases. The velocity dependence of the solid matrix stresses can be overlooked, and accommodated by the suitable choice of the drag coefficient function K. In our opinion, the confined compression experiment is a 'bad' experiment [1].

Under the unconfined conditions, the relative velocity between phases is much smaller than that of the solid phase. The solid phase stress velocity dependence cannot be accounted for by tuning the frictional drag between phases and causes the inability of the standard biphasic model to describe tissue behavior.

CONCLUSIONS

The standard biphasic models of the soft tissues ***rely*** on the following simplifying assumptions:

- Tissue is a mixture of two immiscible, chemically inert constituents: an incompressible solid matrix and incompressible fluid,
- Solid matrix is non-dissipate; history independent, nemely, elastic,
- Fluid is non-dissipate,
- The only dissipation comes from frictional drag of relative motion between the phases,
- The diffusive momentum exchange between phases is proportional to their relative velocity.

The deformation behavior of the soft tissue is dominated by four nonlinear effects:

- The strain-dependent porosity effect,
- The strain-dependent permeability effect,
- The finite deformation effect,
- The stress dependence on velocity of loading effect.

The second simplifying assumption of the standard biphasic model - the elasticity of the solid matrix, conflicts with experimentally demonstrated velocity dependence of the stresses. Strictly considering, there is a possibility that the inconsistency was caused by the difference between brain and cartilage. However, if we study the behavior of brain, or other very soft tissues, we conclude that the pure elasticity assumption must be abandoned.

For computation, this obviously makes the analysis even difficult. Therefore, we suggest that we develop certain calibration means for the prediction stage of the deformation, which should be coupled with the history of the measured deformation.

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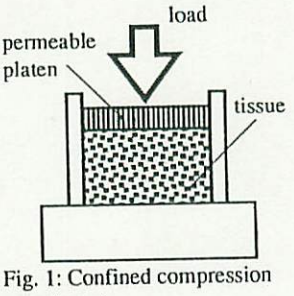

Fig. 1: Confined compression experiment.

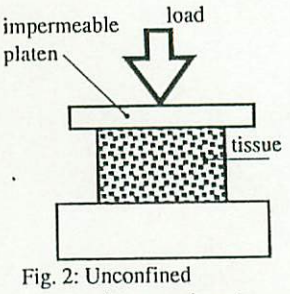

Fig. 2: Unconfined compression experiment.

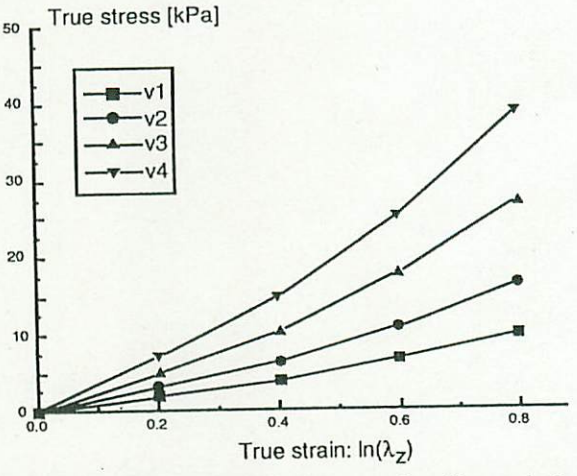

Fig. 3 Results of the unconfined compression of human brain tissue (cited from [3]). v1=-0.033 [1/s], v2=-0.33 [1/s], v3=-3.3 [1/s], v4=-16.5 [1/s]. The strong dependence on the strain rate is evident.