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A biphasic Finitee-Element-Model for Sinusoidal Liver Perfusion Remodeling

Liver resection can lead to focal outflow obstruction due to transection of hepatic veins. Outflow obstruction may cause additional damage to the small remnant liver. Drainage of the obstructed territories is reestablished via dilatation of sinusoids. Subsequently sinusoidal canals are formed draining the blood from the obstructed territory to the neighboring unobstructed territories. We raised the phenomenological hypothesis that the blood pressure gradient is the main driving force for the formation of sinusoidal vascular canals. Based on the theory of porous media we generated a biphasic mechanical model to describe this vascular remodeling process in relation to the variable pressure gradient. Therefore, we introduced a transverse isotropic permeability relation as well as an evolutional optimization rule to describe the relationship between pressure gradient and the direction of the sinusoidal blood flow in the fluid phase. As a next step, we developed a framework for the calculation concept including the representation of the governing weak formulations. The governing equations of the model are developed on the basis of a consistent thermo-mechanical approach including the momentum and mass balances of both solid and fluid phases. The mathematical concept describes the motion of the solid phases coupled by the fluid transport due to pressure development. The theoretical formulations are implemented into the finite element code FEAP. Then, we examined a representative numerical example with simulation of the blood flow under both conditions, the physiological situation as well as after outflow obstruction. We based our simulation on the concept of mechanical-induced remodeling. We incorporated the fluid directly into the model as a mixture together with the solid. We hypothesized that the reorientation of the sinusoidal flow and the remodeling of the sinusoidal structure depends mainly on the fluid pressure and the fluid pressure gradient caused by the outflow obstruction. We tested this hypothesis with a numerical simulation and compared the results to the experimental findings. As we did not implement liver resection in the mathematical model presented here, but concentrated on focal outflow obstruction only, liver growth (=regeneration) was not addressed. Doing so, we were able to reproduce numerically the experimentally observed process of reestablishing hepatic venous drainage via redirection of blood flow and formation of new vascular structures in respect to the fluid flow. The calculated results support the hypothesis that the reorientation of blood flow mainly depends on the pressure gradient. Further investigations are needed to determine the micromechanical influences on the reorientation of the sinusoids.