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MODELLING MYOCARDIAL INFARCTION IN THE HEART

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Understanding the healing and remodelling processes induced by myocardial infarction (MI) of the heart is important and the mechanical properties of the myocardium post-MI can be indicative for effective treatments aimed at avoiding eventual heart failure.

We present two different modelling approaches for MI. Since MI remodelling is a multiscale feedback process between the mechanical loading and cellular adaptation, our first approach is based on an agent-based model [1], in which we describe collagen remodelling by fibroblasts regulated by chemical and mechanical cues after acute MI, and upscaling into a finite element 3D left ventricular model. We model the dispersed collagen fibre structure using the angular integration method, and have incorporated a collagen fibre tension-compression 'switch' in the LV model. This enables us to study the scar healing (collagen deposition, degradation and reorientation) of a rat heart post-MI. Our results, in terms of collagen accumulation and alignment, compare well with published experimental data. In addition, we show that different shapes of the MI region can affect the collagen remodelling, and in particular, the mechanical cue plays an important role in the healing process.

Our second approach [2] is patient-specific image-based modelling of myocardial mechanics following an acute-MI. We develop a finite element model of a human LV with MI morphologies derived directly from Late-Gadolinium enhanced (LGE) magnetic resonance (MR) images. The LV geometry is reconstructed from in vivo short-/long-axis cine images of a patient after acute-MI. A linear relationship between LGE intensity and myocardial passive stiffness and contractility is assumed. We approximate the LV end-diastolic pressure with a population-based value (15 mmHg) and assume that the LV peak systolic pressure is the same as the cuff-measured value. The full scar region (normalized LGE intensity > 0.95) is considered to be passive and 50 times stiffer compared to the remote functional myocardium. To calibrate the passive stiffness, we inversely determine the eight unknown material parameters by matching the strain measurements estimated from MR images and the LV end-diastolic volume using a multi-step optimisation approach. We further match the LV EDP-EDV curve published previously. The myocardial contractility is determined by matching the systolic function, including the systolic peak circumferential strain and LV ejection fraction. We infer from our model that the myocardial contractility of this patient is around 180.8 kPa, which is within the range of previously published human values. Finally, patient-specific myofibre stress distributions at end-diastole and end-systole are discussed in relation to the LV adverse remodelling.

REFERENCES

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