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A WHOLE HEART MODEL WITH FINITE GROWTH FOR HYPERTENSION-INDUCED PATHOLOGIES

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INTRODUCTION. Heart failure (HF) is a leading cause of morbidity and has a 5-year mortality rate of 50%. Up to 70% of all HF conditions are of ischemic origin and all systolic HF conditions involve some degree of myocardial ischemia, in which there is a mismatch between coronary blood supply and myocardial demand. The incidence of advanced HF continues to increase despite considerable progress in medical management and the more recent development of devices for treating HF. For end-stage HF, heart transplantation or mechanical assist devices are the only viable options. Although current devices and therapies are useful, early diagnosis is most effective for long-term outcome. Clearly, early diagnosis and prediction of propensity to HF can stratify susceptible patients for preventative medical therapy, including treatment of co-morbidities, to combat this health care epidemic [1].
In this work, we investigate the role of multiple growth mechanisms in disease progression and ventricular mechanics. More precisely, we study longitudinal and transverse growth in systemic and pulmonary hypertension-induced hypertrophy. For this, we use the finite growth theory [2], [3], and the Living Heart Project's whole heart model [4].

METHODS. In finite growth theory, the full transformation of the considered body is decomposed into growth-only and purely elastic transformations [2], [3]. In practice, this is achieved by multiplicatively decomposing the total deformation gradient into growth and elastic parts. The elastic part is plugged into a standard cardiac mechanical model: it is first decomposed into volumetric and isovolumic parts, the former being associated to a large bulk modulus to insure quasi-incompressibility, and the later part to a transversely isotropic Fung strain energy potential [5], [6]. The growth part then obeys an additional evolution law. We study two different kinematics: (i) fiber, or longitudinal, growth, which corresponds to an in-series deposition of sarcomeres; and (ii) transverse growth, which corresponds to an in-parallel deposition. The growth kinetics is always driven by maximal fiber stretch at end-diastole.

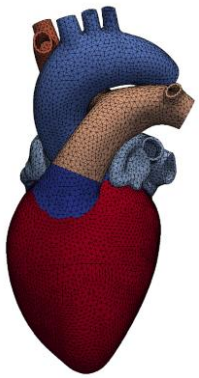

Figure 1.Whole heart finite element model.

The whole heart model used here was created from normal human computed tomography and magnetic resonance data [4]. It contains the four cardiac chambers, and main blood vessels. The geometry is discretized using linear tetrahedrons. The mesh is show in Figure 1. A normal myofiber distribution was defined onto the mesh [4].

The mesh is first loaded to a "normal" end-diastolic state: 5 mmHg in the left ventricle and left atria, and 2 mmHg in the right ventricle and right atria. This corresponds to a 60% ejection fraction and balanced left ventricular and right ventricular stroke volumes. This also defines a "normal" fiber stretch field, which will be used as threshold in the growth steps. To study the effect of systemic and pulmonary hypertension, we then overload either the left or right side of the heart, and activate the growth model.

RESULTS. Figure 2 illustrates, in the case of pulmonary hypertension, the different growth patterns obtained with longitudinal and transverse growth. The evolution of chamber and wall volumes during growth is also represented in Figure 2.

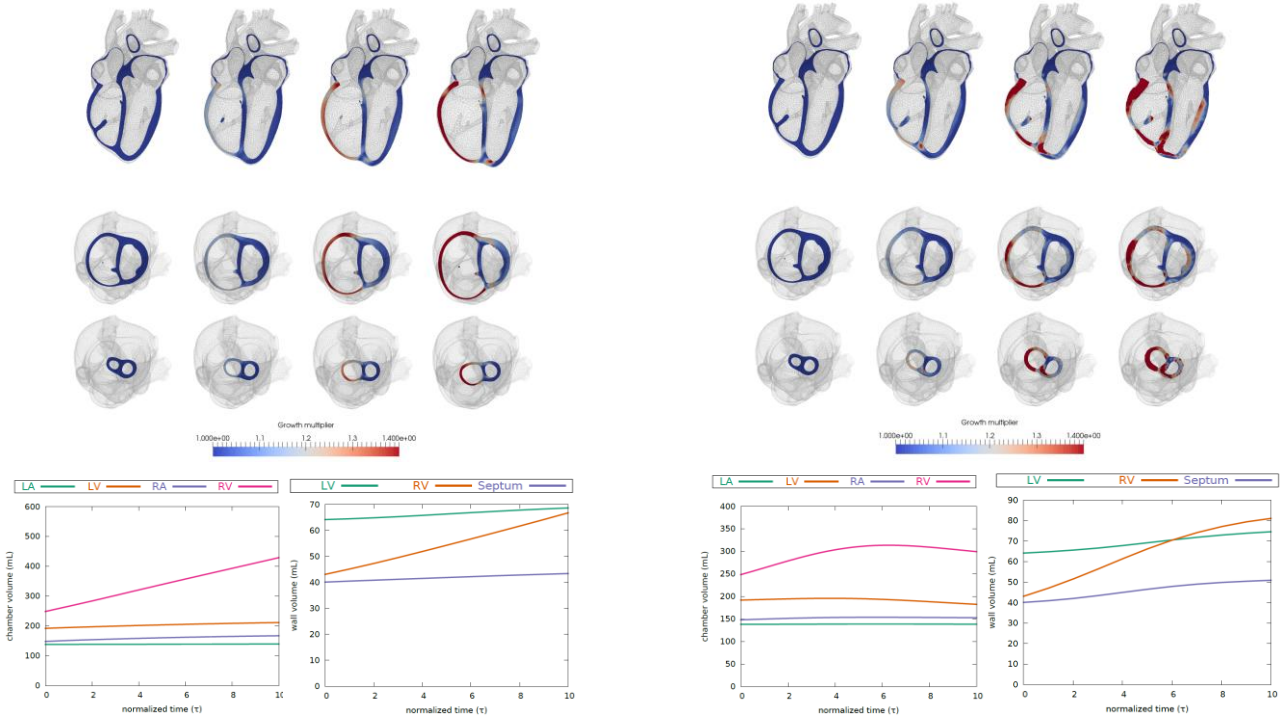

Figure 2.Longitudinal (left) and transverse (right) growth in pulmonary hypertension-induced hypertrophy.Top: evolution of the growth state variable. Bottom: evolution of chamber (left) and wall volumes (right).

DISCUSSION. Our model is able to reproduce the main features of cardiac hypertrophy, such as reverse septum and left ventricular "D-shape" in pulmonary hypertension. It also shows different time behaviors, with non-saturating longitudinal growth and saturating transverse growth. Once better informed with experimental data, our model will have the potential to reliably predict a variety of eccentric and concentric growth phenomena, thus providing the missing link between biology, mechanics and medicine in heart failure.

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