NONLINEAR 3D FE BIOMECHANICAL ANALYSIS OF THE TRABECULATED EMBRYONIC CHICK HEART UNDER CARDIAC CYCLE LOADING

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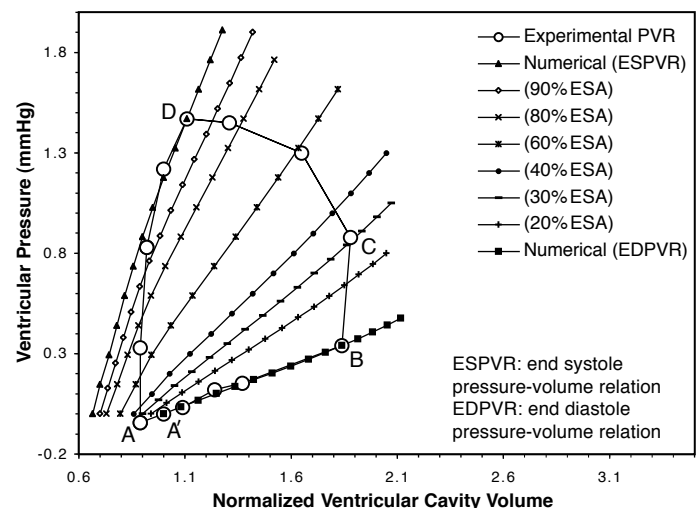Rochester, NY

INTRODUCTION

The wall of the tubular embryonic chick heart begins to trabeculate at Hamburger-Hamilton stage 16 (HH16) and by stage HH21 a fine trabecular network of column-like structures is fully developed. Trabeculation was initially regarded only as a mechanism to increase the delivery of oxygen and nutrients to the working ventricles in the absence of vascular system. However, recent experimental observations suggest that trabeculae in the embryonic heart likely have the role of contractile elements and contribute substantially to the stiffness of the ventricular wall [1]. We perform a systematic finite element (FE) study in order to investigate the impact of ventricular trabeculation on the mechanics of a HH21 undergoing a complete cardiac cycle. We use on a nonlinear hyperelastic 3D FE formulation with active and passive muscle properties and a local-global multiscale approach introduced in [2]. The construction of the image-based 3D mesh for global analysis (the *standard* model) and its calibration are discussed in [3]. In this paper, we first determine appropriate HH21 blood pressure and muscle activation intensity values based on experimental pressure-volume data. Then we simulate a complete cardiac cycle using the calibrated standard model. Finally we validate the FE simulation by comparing the predicted epicardial strains with those obtained through video-based motion tracking and discuss the simulation results.

CARDIAC CYCLE LOADING

The pressure- and activation-time curves needed to simulate the complete cardiac cycle are derived from numerical and experimental data following the procedure introduced by Taber [4]. We first contract the standard model at a specific intensity of muscle activation, e.g., 20% of the end-systole activation (20% ESA in Fig.1). Then we gradually apply internal pressure and record the cavity volume as a function of the applied pressure, thus generating an isochronal pressure-volume curve (Fig. 1). This process is repeated for each desired activation intensity. We appropriately scale the active material parameters under the assumption that the stiffness of the active fibers is proportional to the muscle activation intensity. The intersection point between a computed isochronal P-V curve and the experimental P-V loop yields the ventricular pressure load corresponding to specific activation intensity. Finally, by correlating this pressure/activation data with the experimental ventricular pressure-time curve in [5] we construct the activation-time curve. The pressure- and activation-time curves allow us to precisely correlate the FE simulation results at different load steps to time-dependent experimental epicardial strains obtained through motion tracking.

We apply the obtained pressure and activation loading to the standard FE model and simulate a complete cardiac cycle of a HH21 heart. Computed Green-Lagrange (GL) strains distributions at end-diastole and end-systole are shown in Figs. 2 and 3.

Figure 1. Isochronal pressure-volume curves. (A': zero pressure loading, B: end-diastole and start of isovolumetric contraction, C: start of ejection, and D: end-systole).

RESULTS AND DISCUSSION

We experimentally measure the epicardial strains within a small region on the right side of HH21 ventricle through a video-based motion tracking analysis. Figure 4 shows computed and experimentally measured GL strains at similar epicardial locations on the right side of the ventricle. The computed circumferential strain agrees well with the experimental data. Both FE simulation and motion tracking report a small shear strain. The initial increase in longitudinal strain during isovolumetric contraction can be observed in both FE simulation and motion tracking results. However, the predicted maximal longitudinal strain at start of ejection is about twice as large as the experimental one, indicating that the current FE modeling may under-estimate the longitudinal stiffness of ventricular wall at this specific location. Considering that these two sets of strain results are based on two different hearts, and that there are substantial mathematical simplifications for both FE modeling and motion tracking, we regard the accuracy of our FE simulation of a functioning HH21 heart as acceptable, in particular for the current qualitative investigation on the mechanics of the trabeculated heart and the functional roles of trabeculated myocardium.

The FE simulation predicts a relatively uniform strain distribution in the compact layer. This homogeneous epicardial deformation is probably the outcome of the balance between the material property and the thickness of the ventricular wall. Despite of a uniform epicardial deformation, the trabecular layer features a significant non-uniformity in both strain and stress distributions. We speculate that the heterogeneous stress and strain fields in the trabecular layer might contribute to the continuous remodeling of the trabeculated myocardium. Results show that a strong stress discontinuity exists at the material interface of the compact and the trabecular layers. This transmural stress discontinuity might be partly responsible for the formation at later developmental stages of smaller and finer trabecular cavities - and hence a stiffer trabeculated myocardium - next to the compact layer.

ACKNOWLEDGMENTS

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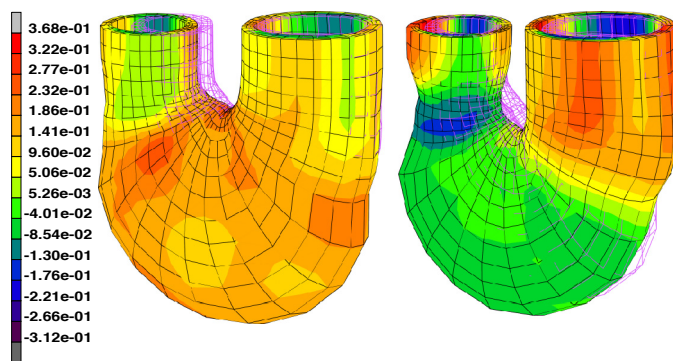
Figure 2. Circumferential epicardial GL strains (a) at end-diastole and (b) at end-systole.
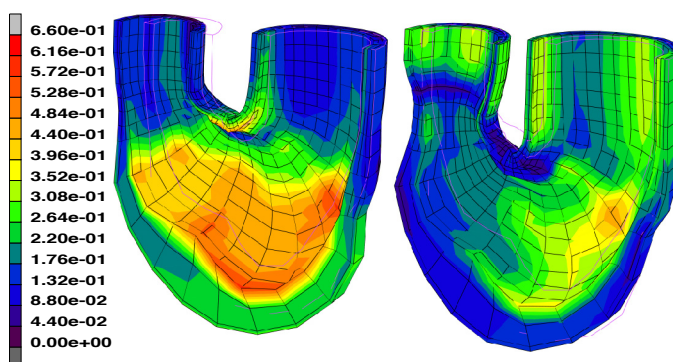

Figure 3. Equivalent total GL strains (a) at end-diastole and (b) at end-systole.
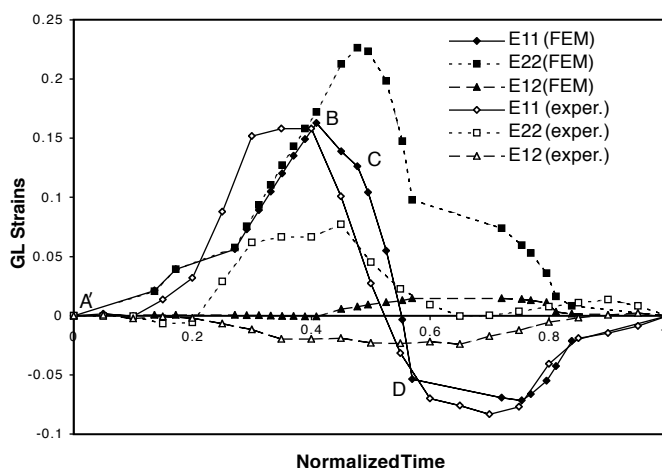

Figure 4. Comparison of epicardial GL strains from FE simulation and from motion tracking. E11, E22, and E12 denote circumferential, longitudinal, and shear strain, respectively.