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DIFFERENTIAL GROWTH-INDUCED RESIDUAL STRESS IN ARTERIES AND THE HEART

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Residual strain and stress are present in living tissues, as evidenced by the classical opening angle experiment on the arterial or heart wall [1]. As such, the unloaded configuration is usually not the reference configuration required for any continuum mechanics computation (see Figure 1). These residual stresses are induced by differential growth and friction-like behavior on the cellular level, and can have a significant impact on the mechanical response of the tissues [1], [2].

In this work, we propose a computational method to create patient-specific residual strain and stress fields, with application to the human heart. From a mechanical standpoint, a residual stress field is an auto-balanced (externally and internally, *i.e.*, divergence-free) stress field, present in the structure when no loading is applied. The method relies on finite growth theory, which is used to induce residual stress in a patient-specific geometry (see Figure 2). The second ingredient of the method is a fixed point algorithm, which is used to find, for a given level of residual stress, the stress-free reference configuration that correlates with the *in vivo*-aquired patient-specific geometry after residual stress are introduced.

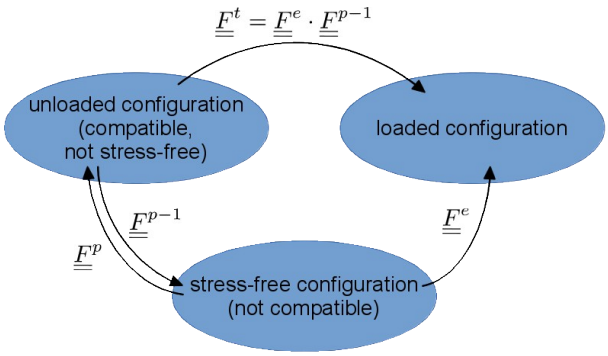

Figure 1. Principle of the mechanics of prestrained biological tissues.

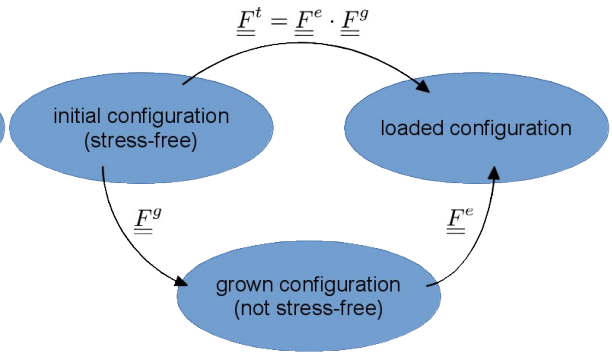

Figure 2. Principle for the mechanics of finite growth.

We first illustrate our method on a simple idealized artery. The artery is first loaded with some internal pressure, grown for some time, and then unloaded (see Figure 3). Because of growth, the final configuration is different from the original one, and contains residual strain and stress, as evidenced by a virtual opening angle experiment (see Figure 3). Figure 4 shows the influence of the amount of growth on the opening angle.

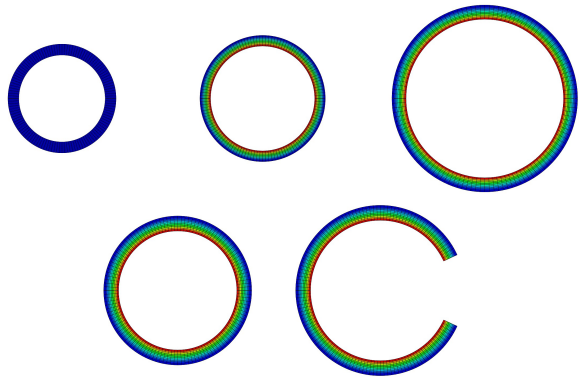

Figure 3. Growth-induced residual stresses and virtual opening angle experiment on an artery.

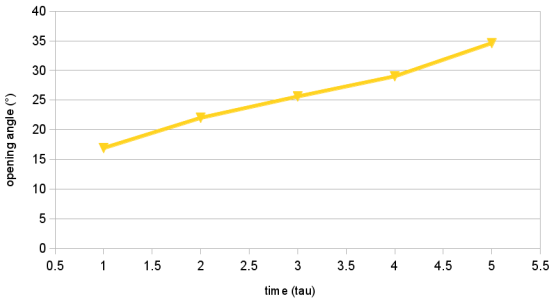

Figure 4. Opening angle as a function of growth time.

We then illustrate our method on a patient-specific left-ventricular heart geometry, developed in [3]. Similar to the artery, the heart is loaded, grown, and then unloaded. Using fixed point iterations, the initial geometry is modified so that the final geometry, which includes prestrain, matches the original MRI geometry. Ventricular pressure load can be easily added to the algorithm, to account for the fact that any MRI geometry, even when built very early in diastole, is never perfectly unloaded. The ventricle is then sliced and cut open to highlight the presence of residual stress (see Figure 5). We use opening angle experimental results to calibrate our model.

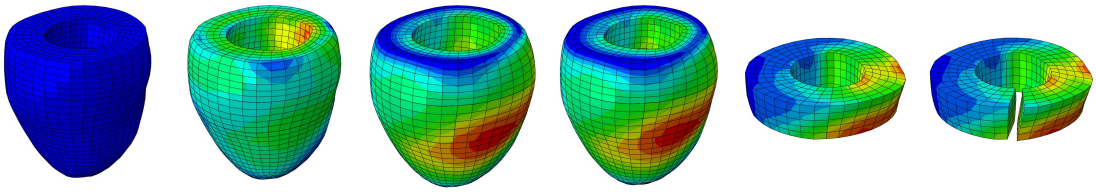

Figure 5. Growth-induced residual stresses and virtual opening angle experiment on subject-specific left ventricle.

We use our method to study the effect of prestrain on ventricular mechanical response. Our work suggests that residual strain and stress play a significant role in cardiac mechanics, and as such have direct implications in cardiac biomedical engineering.

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