Supplementary Material

Material property alterations for HFpEF phenotypes: A numerical study of subject-specific porcine models

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1. Supplementary Tables and Videos

Supplementary Video S1: Maximum principal strain (%) distribution in the four models throughout the last cardiac

Table S1: Comparison of the circulatory system parameters.

	Case 1		*Case 2*	
	Normal	**HFpEF**	**Normal**	**HFpEF**
Aortic valve resistance	1	4	2	3
Systemic resistance	8	160	18	160
Venous resistance	132	100	75	55

2. Additional details for "Klotz" curve configuration

The "Klotz" curve can be configured using a single known scaling point to analytically predict the end-diastolic pressure-volume relations, as suggested in the original paper of Klotz et al. (2006). Volumes at the pressure of 0 mmHg (V_0) and 30 mmHg (V_{30}), are computed from the scaling point:

$$V_0 = V_m(0.6 - 0.006P_m) \quad ; \quad V_{30} = V_0 + \frac{V_m - V_0}{0.302P_m^{0.358}}$$

where V_m, P_m are the volume and pressure of the scaling point. The resultant V_{30} can then be used to calculate the coefficients α and β:

$$\alpha = 30/V_{30}^{\beta} \quad ; \quad \beta = \frac{\log(P_m/30)}{\log(V_m/V_{30})}$$

Finally, the entire end-diastolic pressure volume relation can be expressed as $P = \alpha V^{\beta}$.

3. Additional details for active contraction

The constitutive equations of the active stress are (Guccione et al., 2001):

$$\sigma_{af(t,E_{ff})} = \frac{T_{max}}{2} \frac{Ca_0^2}{Ca_0^2 + ECa_{50}^2(E_{ff})} (1 - \cos(\omega(t, E_{ff})))$$

$$ECa_{50}^2 = \frac{(Ca_0)_{max}}{\sqrt{\exp[(B(l-l_0)]-1}} \quad ; \quad l = l_R\sqrt{2E_{ff}+1}$$

$$\omega = \begin{cases} \pi \frac{t}{t_0} & \text{when} \quad 0 \le t \le t_0 \\ \pi \frac{t - t_0 + t_r}{t_r} & \text{when} \quad t_0 \le t \le t_0 + t_r \\ 0 & \text{when} \quad t \ge t_0 + t_r \end{cases} \quad ; \quad t_r = ml + b$$

where T_{max} is the isometric tension of the largest sarcomere length with the highest calcium concentration multiplied by a calcium concentration determining term and a contraction timing governing term, both of which are dependent on sarcomere length *l*. The timing of contractile function corresponds with the heart rate and timing of the cardiac cycle, which is imposed by a time (*t*) dependent modulus function.

For calibration purposes, a consistent alteration in contractility was facilitated by altering the T_{max}, changing simultaneously both the total contractile force of the tissue as well as the aforementioned parameters related to active behavior.

The model was initialized with the passive material properties previously calibrated. Then, the active calibration followed methods from previous studies (Genet et al., 2014; Guccione and McCulloch, 1993; Sack et al., 2016). The pressure in the LV was raised to EDP, increasing the LV volume more than the EDV that was used for the calibration of the passive calibration. Next, the active tension was elevated to enforce systolic contraction and thereby reducing LV volume. The T_{max}, which governs the systolic contraction, was scaled iteratively using a gradient descent algorithm to match the end-systolic volume obtained from the simulation to the animal study measurement.

Supplementary references

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