"Computational Simulation of Hemodynamic-Driven Growth and Remodeling of Embryonic Atrioventricular Valves"

Biomechanics and Modeling of Mechanobiology

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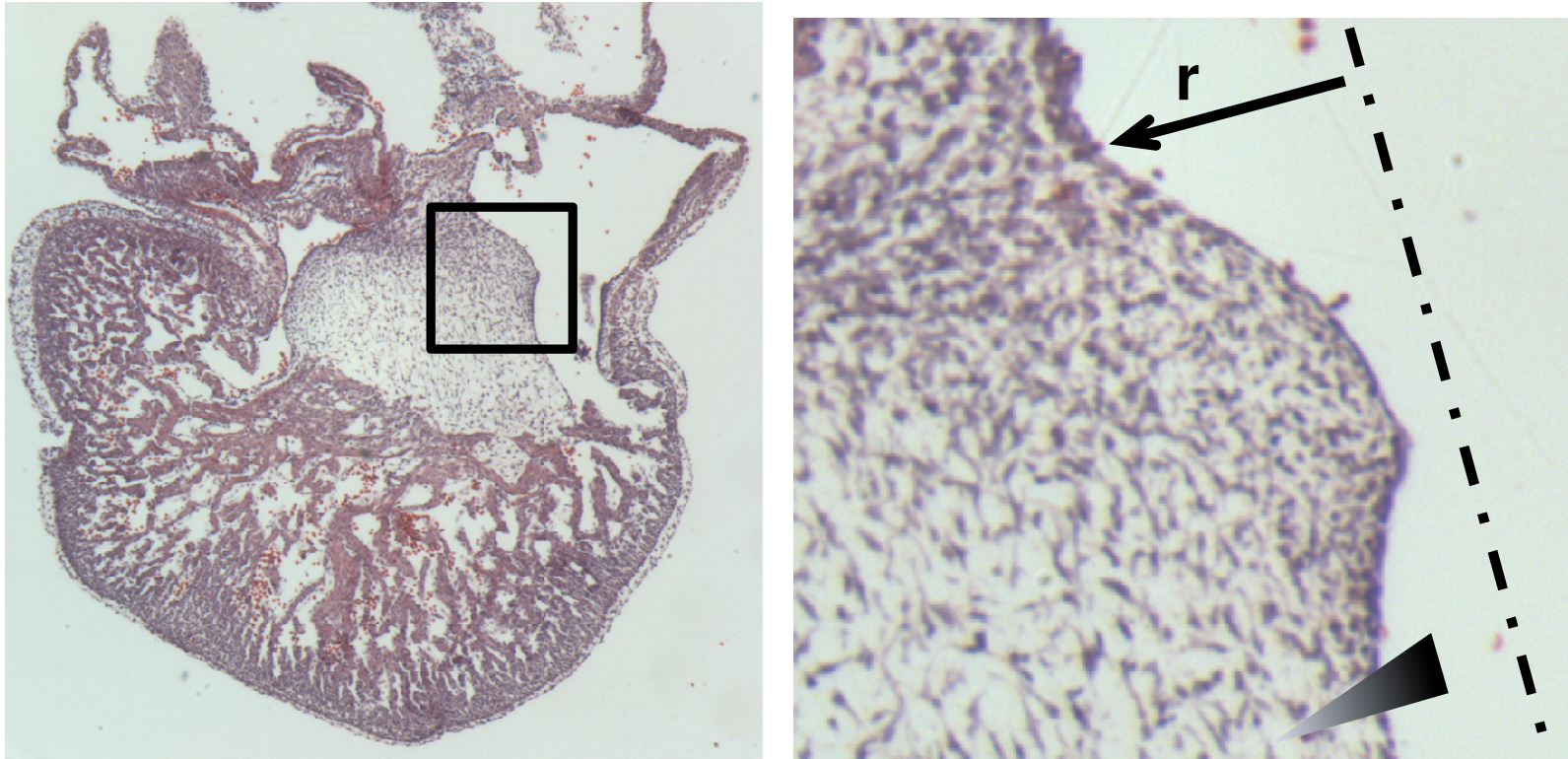306 Weill Hall, Cornell University, Ithaca, NY 14853
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Fig. S1 Increase cell density near the valve surface supports a gradient in the growth rate parameter. Representative histology image of HH27 left AV valves stained with Weigert's hematoxylin. Magnification: 94x, 150x
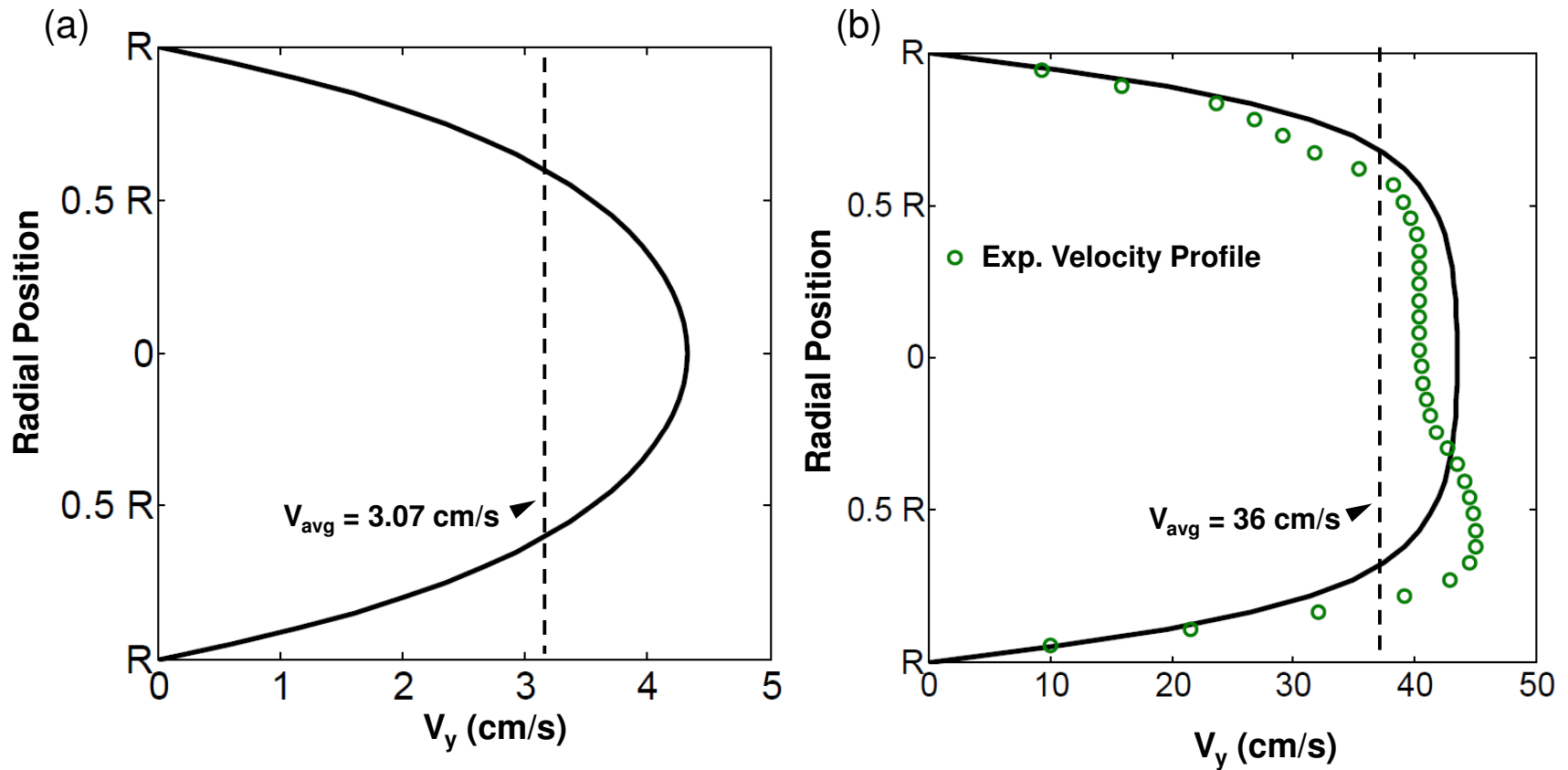
Fig. S2 Average velocity over the cardiac cycle generates low Reynolds flow (Re = 5). a) Parabolic velocity profile at AV orifice under average flow condition, with approximated 3.07 cm/s spatial average. **b)** Plug flow velocity profile across the AV orifice when experimental peak velocity used in simulation. Plug flow agrees with calculated velocity profile using innate AV orifice geometry (Yalcin et al 2011). This indicates that the idealized initial geometry of this model is an appropriate approximation. (Re = 65)
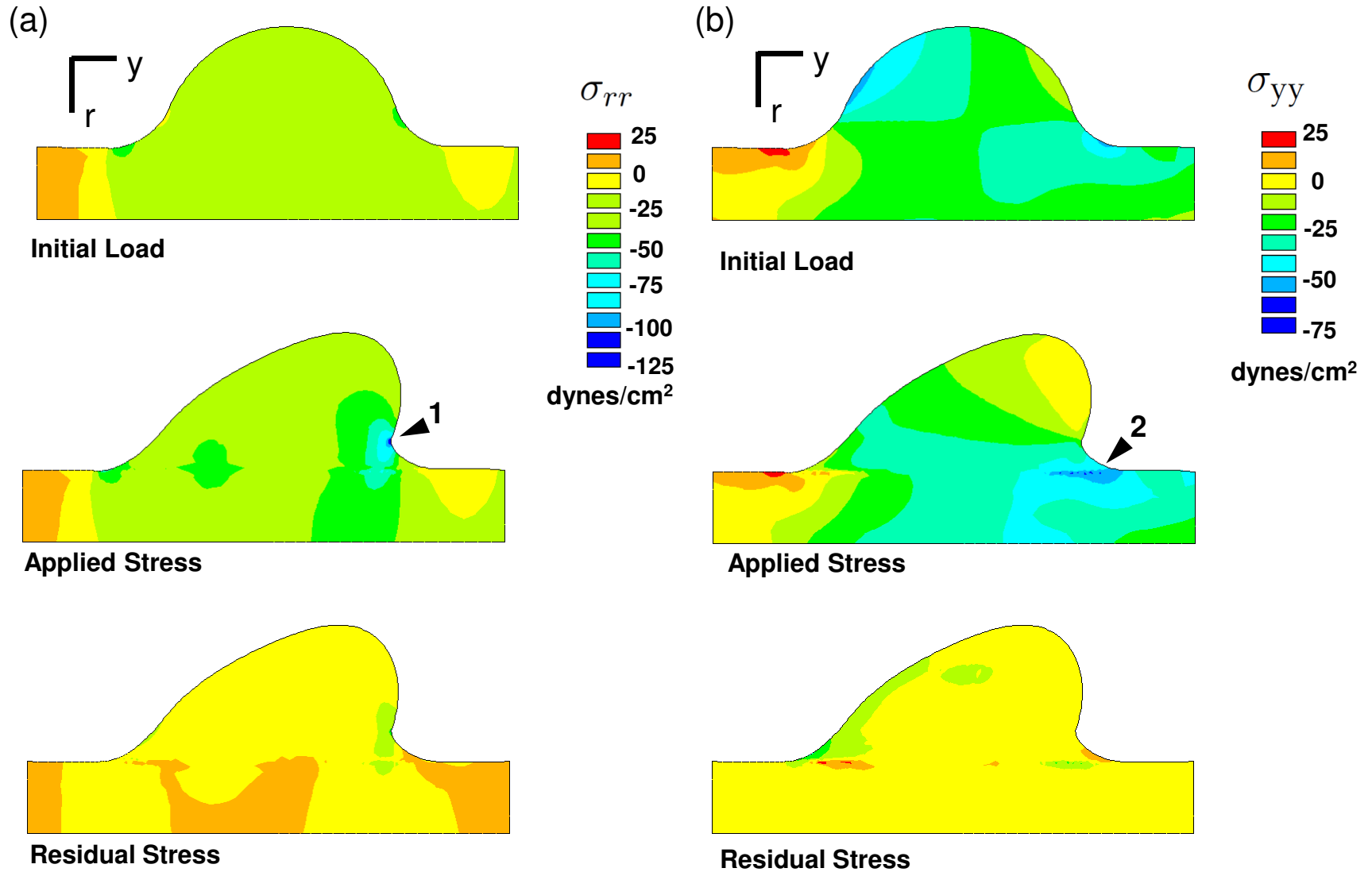
Fig. S3 Growth induced residual stress in the radial and axial directions.   a) Radial stress. The residual stress in this direction is minor when compared to the applied stress state values. Significant compressive radial stress occurs at the cushion/myocardial interface on the flow exit side (arrow 1). **b)** Axial Stress. Residual stress is insignificant when compared to the applied stress, but stress concentrations do exist along the cushion/myocardium interface (arrow 2).
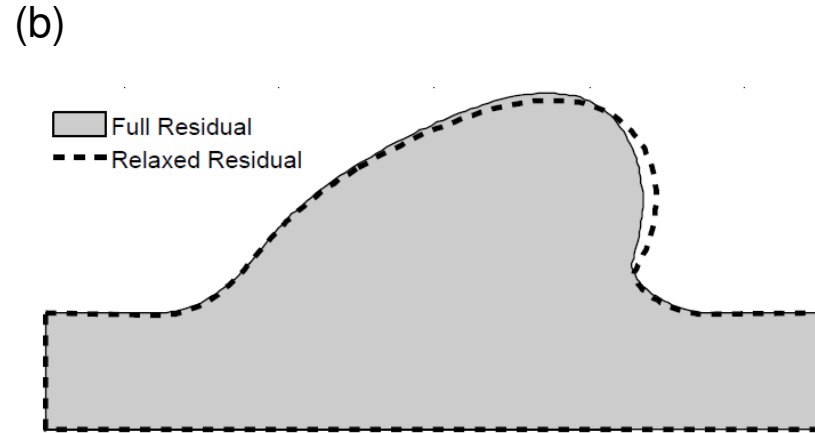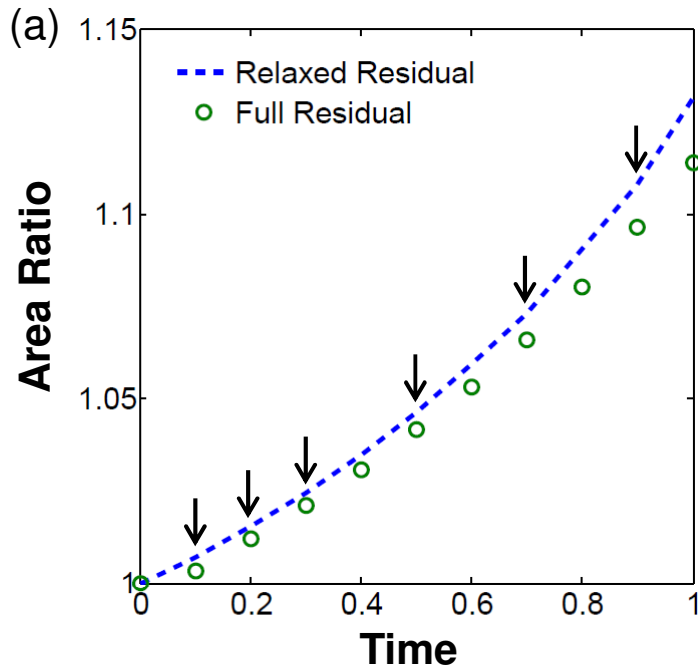
Fig. S4 Residual stress alters AV cushion evolution. a) Plot of normalized cushion area vs. normalized simulation time. Arrows denote the reset of the stress-free reference configuration to the unloaded evolved state, which in effect eliminates the residual stresses. The "relaxed" residual model increased the cross-sectional area at a faster rate than the simulation with residual stress maintained. **b)** Reduced residual stress stimulated evolution of more tissue in the direction of blood flow. Simulation details: Full Load, $\sigma^* = -\Delta P/2$