An In-Silico Coupled Electromechanical Model to Identify Arrhythmogenic Patterns in Mitral Valve Prolapse

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Introduction

- **Background.** A subset of MVP (A-MVP) develops ventricular arrhythmias and SCD risk despite minimal regurgitation.

- **Signatures.** Inferior-lead T-wave inversion; modest QTc/Tp–Te prolongation; increased mechanical dispersion (MD); focal fibrosis on LGE in papillary/inferobasal LV.

- **Objective.** Build a patient-specific, coupled EP–EM model that *reproduces and identifies* arrhythmogenic patterns and enables an inverse retrieval of segmental substrate from ECG + CMR/LGE.

Methods

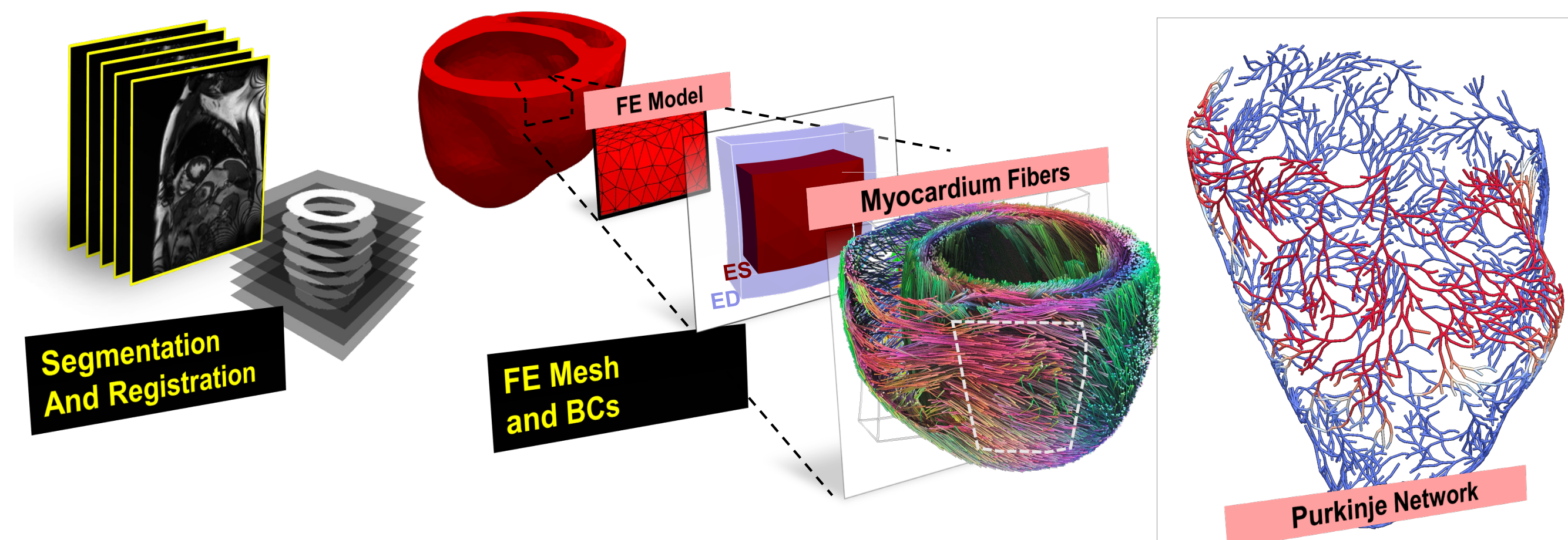### Overall patient-to-model pipeline.

Figure 1. Cine CMR → segmentation/registration → FE mesh/BCs → rule-based fibers → endocardial Purkinje network.

Cohort & input data.

- Cross-sectional MVP cohort: **n=20** (A-MVP=10, NA-MVP=10).
- Paired pre-/post-MV repair (MVRe): **n=8** (0–6 mo pre, ~30 mo post).
- ECG: resting 12-lead (emphasis on II/III/aVF);
- CMR: cine bSSFP for strain; LGE or T1 mapping.

Imaging & preprocessing.

- LV/RV segmentation → biventricular FE mesh; AHA-17 segments.
- Feature-tracking strain → TTP_s per segment → compute MD and late-systolic residual strain rate (LSRS).
- LGE mask $S(\mathbf{x})$ co-registered to mesh (papillary, inferobasal) and used as priors:

$$\mathbf{D}(\mathbf{x}) = \mathbf{D}_0(1 - \rho S), \qquad \mathrm{APD}(\mathbf{x}) = \mathrm{APD}_0(1 + \kappa S).$$

where $\mathbf{D}$ is electrical conductivity, ρ the reduction factor, APD the action potential duration, and κ its local prolongation scale.

Model equations (short form).

$$C_m \dot{V}_m = \nabla \cdot (\mathbf{D}\nabla V_m) - I_{\mathrm{ion}} + I_{\mathrm{stim}},$$

$$T_{\mathrm{active}} = f(v, \lambda), \quad \lambda = \sqrt{\mathbf{f}_0 \cdot \mathbf{C}\, \mathbf{f}_0}, \quad \nabla_x \cdot (\mathbf{FS}) + \mathbf{b}_0 = \rho_0 \ddot{\mathbf{u}}.$$

where V_m is transmembrane potential, $\mathbf{D}$ the conductivity tensor, T_{active} the active tension from voltage–stretch coupling, λ the fiber stretch ratio, and $\mathbf{S}$ the total stress tensor driving tissue mechanics.

Methods (contd.)

Pseudo-ECG.

$$\phi_u(t) = \frac{1}{4\pi G_{\mathrm{torso}}} \int_V \frac{(G_i \nabla \phi_i) \cdot \mathbf{r}}{|\mathbf{r}|^3} dV, \quad \mathbf{r} = \mathbf{x} - \mathbf{x}_a$$

- G_{torso}: scalar torso conductivity ($\approx 1\,\mathrm{S\,m^{-1}}$)
- G_i: intracellular conductivity tensor
- ϕ_i: intracellular potential
- $G_i\nabla\phi_i$: intracellular current density
- $\mathbf{x}$: source-element position
- $\mathbf{x}_a$: electrode position.

Wilson's central terminal
- $\mathrm{WCT} = \frac{1}{3}(\mathrm{RA} + \mathrm{LA} + \mathrm{LL})$
- $V_k = \phi_k - \mathrm{WCT}, \quad k = 1 \dots 6$

Lead
- I   LA – RA
- II LL – RA
- III LL – LA
- aVF LL – 1/2 (RA + LA)
- aVL LA – 1/2 (LL + LL)
- aVF LL – 1/2 (RA + LL)
- aVR RA – 1/2 (LA + LL)

Figure 2. Schematic of lead geometry and lead-field weighting used to synthesize pseudo-ECG.

Mechanical dispersion (cine FT).

$$TTP_s = \arg\max_t \varepsilon_{cc,s}(t), \qquad \mathrm{MD} = \mathrm{SD}(TTP_1, \dots, TTP_{17}).$$

Inverse problem

$$\min_{\boldsymbol{\theta}} \sum_\ell \left\| E_{\mathrm{sim}}^{(\ell)}(\boldsymbol{\theta}) - E_{\mathrm{obs}}^{(\ell)} \right\|^2 + \alpha \left\| \mathbf{TTP}_{\mathrm{sim}}(\boldsymbol{\theta}) - \mathbf{TTP}_{\mathrm{CMR}} \right\|^2 + \beta\, \mathrm{TV}(\mathrm{APD}(\boldsymbol{\theta})) + \gamma\, \Phi(\mathrm{APD}; S(\mathbf{x})),$$

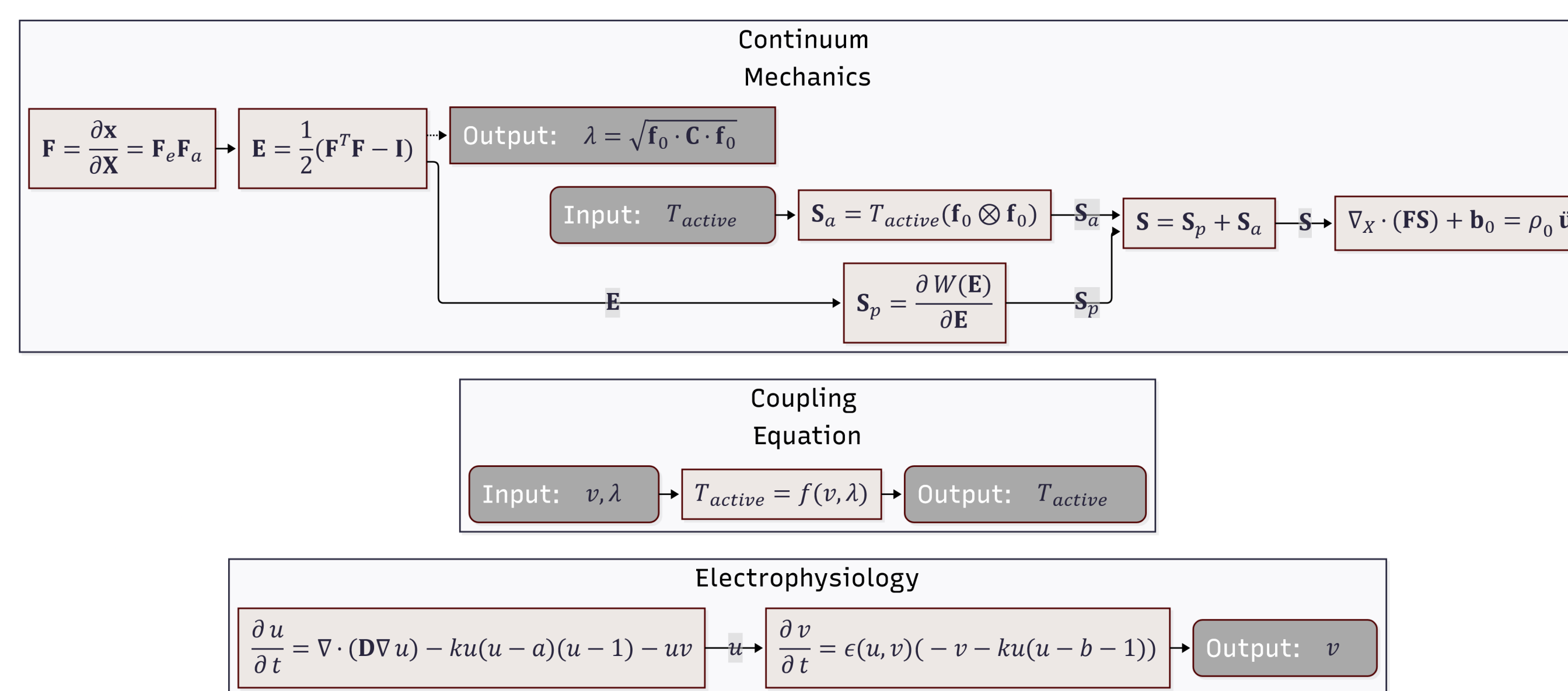where Φ is an LGE-weighted smoothness prior and ℓ emphasizes II/III/aVF.

Figure 3. Electromechanical coupling workflow and information flow between EP, coupling law, and mechanics.

Results

- **Baseline (NA-MVP):** parameters tuned to match CMR regional strain; pseudo-ECG with normal inferior-lead polarity and physiologic QTc.

- **A-MVP remodeling:** regional APD prolongation and local Purkinje delay near papillary insertions with optional LGE-guided conductivity/active-tension changes.
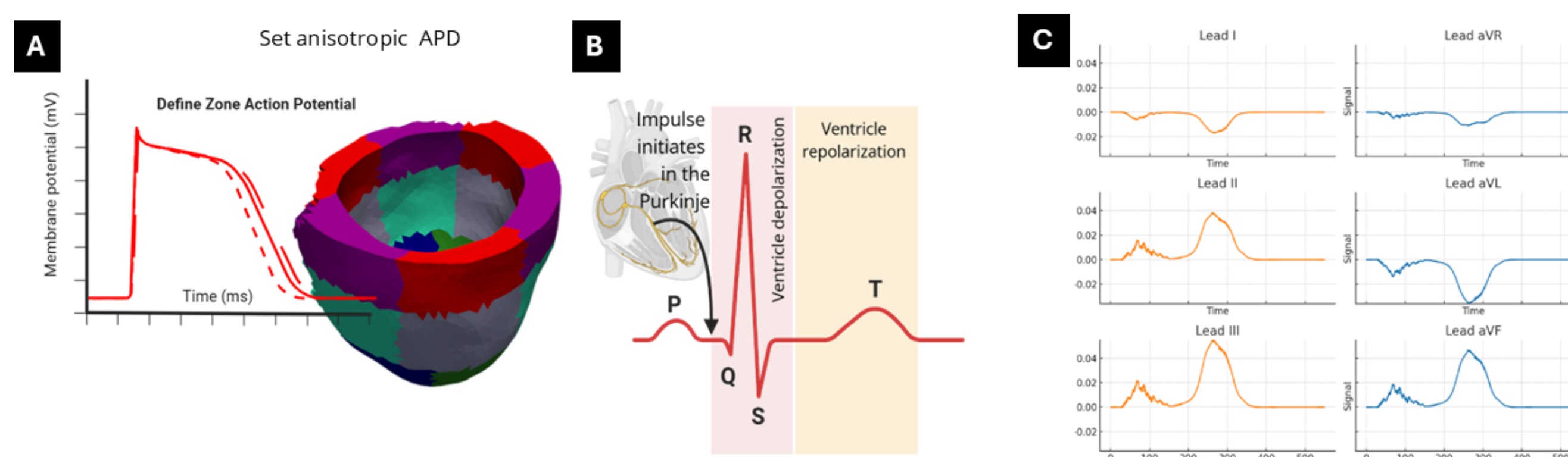

Figure 4. **Representative outputs from the remodeling experiment.** (A) Example regional APD modulation applied to the LV model. (B) Purkinje-initiated activation schematic used for pseudo-ECG generation. (C) Synthetic 12-lead ECG showing lead-specific manifestations; inferior leads (II/III/aVF) invert and Tp–Te modestly increases after remodeling.
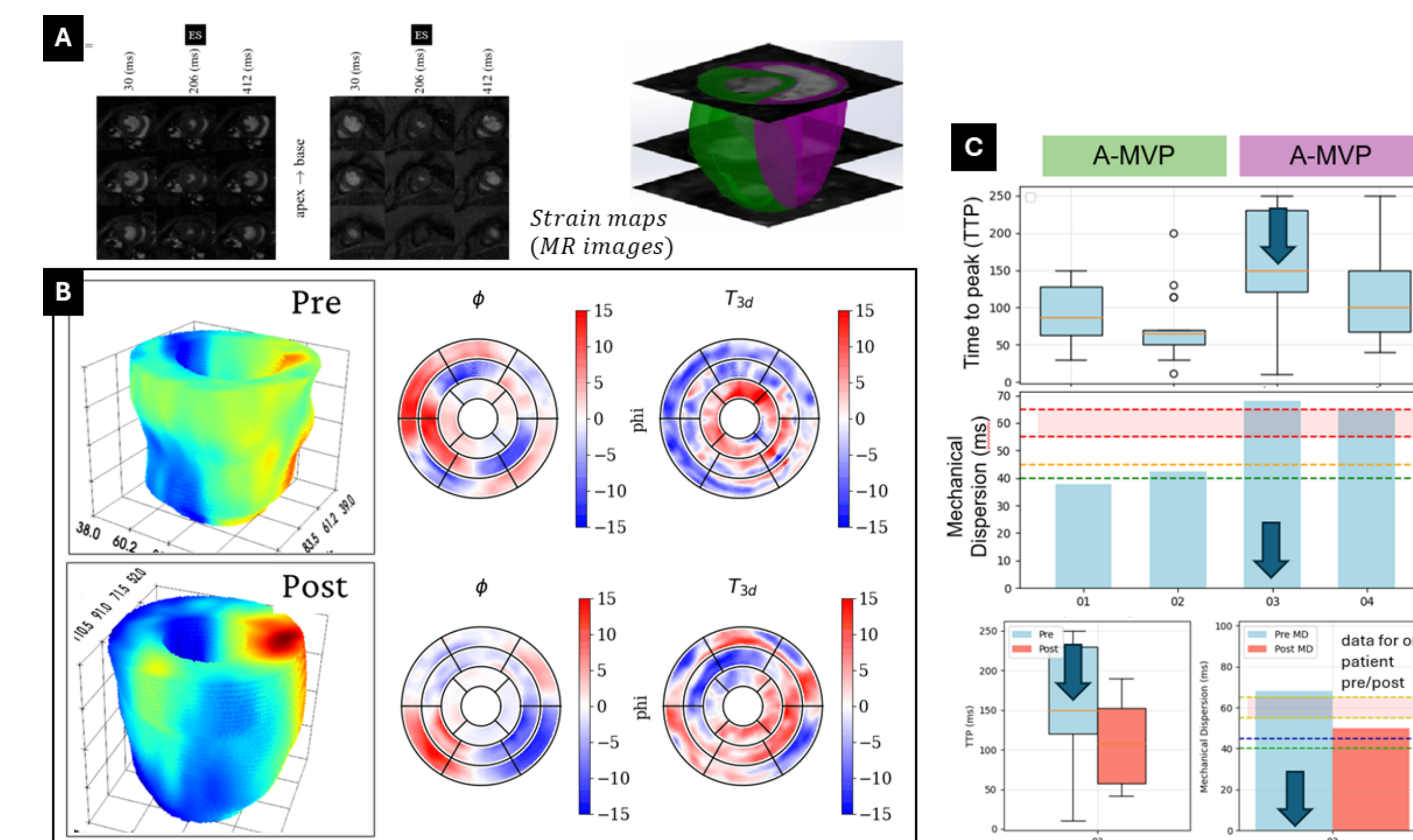
Results (contd.)

Figure 5. Patient-specific LV model: CMR→mesh, rule-based helix-angle fibers, and endocardial His–Purkinje network.

Discussion

- Simulations with regional APD prolongation and localized Purkinje delays can reproduce several hallmark A-MVP ECG/mechanics features in this modeling framework.

- LGE-informed parameter changes are treated as priors; their specific electrical and mechanical effects should be interpreted cautiously and verified on a larger cohort.

Conclusion

A coupled, patient-specific EP–EM framework calibrated to ECG and cine/LGE reproduces key A-MVP signatures in silico and *may help* identify segmental patterns relevant to risk stratification and post-repair tracking. Additional subjects and calibration/validation are planned.

References

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3) Holzapfel & Ogden (2009) — Myocardial constitutive modeling; monodomain/forward ECG basics.

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