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.

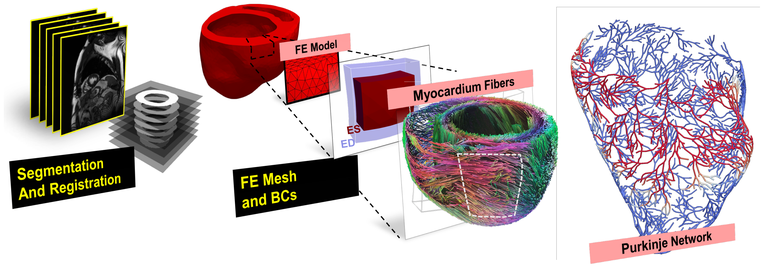

Figure 1. Cine CMR → segmentation/registration → FE mesh/BCs → rule-based fibers → endocardial Purkinje network.

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).

$$C_m \dot{V}_m = \nabla\cdot(\mathbf{D}\nabla V_m) - I_{\mathrm{ion}} + I_{\mathrm{stim}},$$
$$T_{\mathrm{active}} = f(v,\lambda),\ \lambda = \sqrt{\mathbf{f}_0\cdot\mathbf{C}\,\mathbf{f}_0}, \quad \nabla_x\cdot(\mathbf{FS}) + \mathbf{b}_0 = \rho_0\ddot{\mathbf{u}}.$$

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.

- $\phi_h(t) = \frac{1}{4\pi\sigma_{torso}}\int_V \frac{(G_i\nabla\phi_i)\cdot\tau}{\|\tau\|^3}\,dV$, $\mathbf{r} = \mathbf{x} - \mathbf{x}_a$
  - σ_{torso}: scalar torso conductivity (≈ 1 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
  - WCT $= \frac{1}{3}$(RA + LA + LL)
  - $V_k = \phi_{V_k} -$ WCT, $k = 1\ldots6$

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

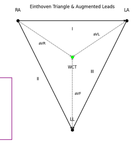

Figure 2. Schematic of lead geometry and lead-field weighting used to synthesize pseudo-ECG.

Mechanical dispersion (cine FT).

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

Inverse problem

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

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

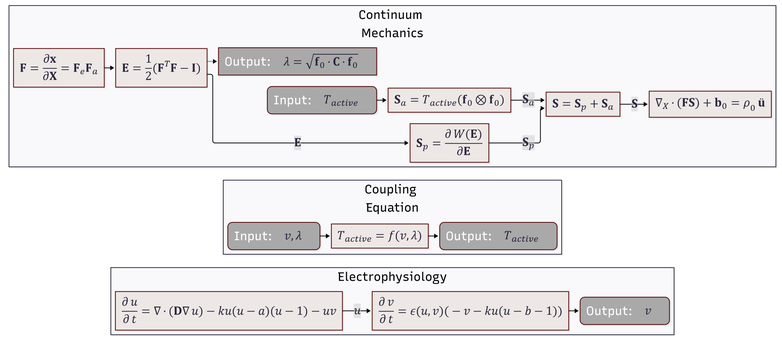

Figure 3. Electromechanical coupling workflow and information flow between EP, coupling law, and mechanics.

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.

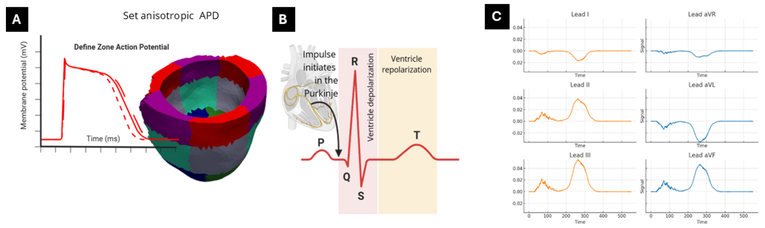

Figure 4. **Representative outputs from the remodeling experiment.** (A) Example regional APD modulation applied to the LV model. (B) Purkinje-initiated activation schematic used for pseudo-ECG generation. (C) Synthetic 12-lead ECG showing lead-specific manifestations; inferior leads (II/III/aVF) invert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

Results *(contd.)*

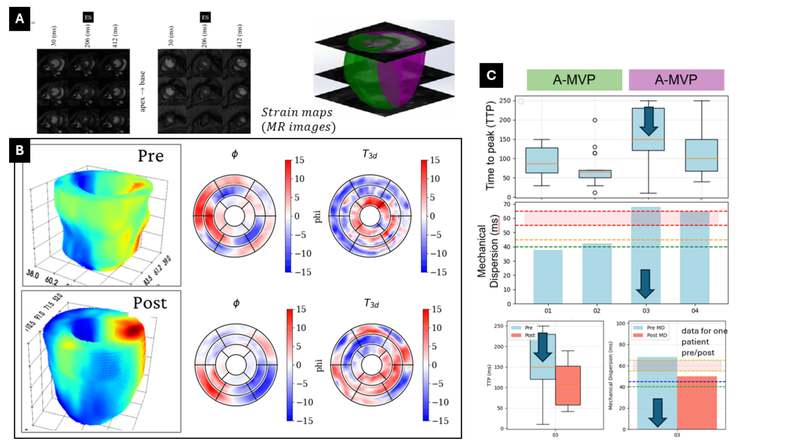

Figure 5. Patient-specific LV model: CMR→mesh, rule-based helix-angle fibers, and endocardial His–Purkinje network.

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

1) Essayagh B, et al. *J Am Coll Cardiol* (2020) — Arrhythmic MVP phenotype and risk.
2) Haugaa KH, et al. *JACC Imaging* (2010) — Mechanical dispersion and arrhythmic risk.
3) Holzapfel & Ogden (2009) — Myocardial constitutive modeling; monodomain/forward ECG basics.

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