Reconstructing Conduction Velocity Maps from Sparse Measurements in Atrial Fibrillation

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Aims: High-resolution mapping of conduction velocity (CV) during atrial fibrillation (AF) provides important insight into arrhythmogenic substrate. However, it is rarely available in routine clinical practice, where measurements are sparse and spatially incomplete.

Methods: We propose a method to reconstruct dense CV maps from limited observations using a principal component analysis-based statistical appearance model (95% variance) with posterior inference. The model was trained on charge density mapping data from 49 patients with persistent AF. Sparse sampling was simulated by randomly subsampling spatial locations, and full CV maps were reconstructed by conditioning the model on the observed values. Performance was evaluated using leave-one-out cross-validation, with reconstruction accuracy quantified using root-mean-square error (RMSE) over unobserved points, and compared to a baseline where the predicted CV map was given by the training set mean. Model uncertainty was estimated from the posterior covariance.

Results: Reconstruction accuracy increased with sampling density, with most gains achieved by ~20-30% surface coverage (Figure). At this level, reconstruction error was approximately 0.3 m/s. This error compares favourably to reported beat-to-beat variability in CV during AF, indicating physiologically meaningful accuracy. Spatial patterns of error were non-uniform, with higher errors observed in regions near the pulmonary veins, left atrial appendage, and mitral valve annulus. Model-derived uncertainty closely matched reconstruction error, with strong spatial correlation ($r = 0.89$, $p < 0.001$).

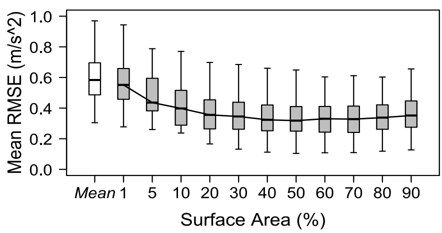

Figure. Reconstruction accuracy as a function of sampling density. Mean root-mean-square error (RMSE) decreases with increasing surface coverage, with most improvement achieved by ~20-30%, beyond which performance plateaus. The mean-only baseline is shown for comparison.

Conclusion: Sparse measurements can capture sufficient information to infer whole-chamber CV distributions. This provides a practical route to reconstruct these maps from limited clinical data, enabling mechanistic analysis of arrhythmogenic substrate and *in silico* modelling of AF, with potential to inform ablation strategies.