Automated Workflow to Integrate Electroanatomic Maps into Patient-Specific Biatrial Models For Personalized AF Treatment

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Abstract

Personalized computational models can improve atrial fibrillation (AF) treatment but typically require invasive imaging or sinus rhythm electroanatomic mapping (EAM), which limits their practicality for real-time use during procedures. This study aims to create an automated workflow for building patient-specific models from AF EAM data, combining anatomical, structural, and functional details. A pipeline integrating bi-atrial anatomical models, AF recordings, and calibrated simulations was used to study AF dynamics with the Atrial Modelling Toolkit. AF cycle length (CL) measured from bi-atrial basket recordings was mapped onto atrial anatomies using Gaussian Process Manifold Interpolation (GPMI) and used in model calibration. AF was simulated using baseline parameters. AFCL recordings for raw clinical data in the LA ranged from 111-220ms (173.30ms ± 13.73ms) and in the RA from 123-220ms (174.30ms ± 15.10ms) across all cases. AFCL recordings for simulated AF in the LA ranged from 71-290ms (198.13ms ± 8.91ms) and in the RA from 92.5-370ms (225.52ms ± 10.90ms) across all cases. AF simulations with baseline parameters showed chaotic activation patterns, highlighting the effects of AF dynamics. Personalized computer models can be created rapidly using this pipeline to create simulations that may provide insights into AF mechanisms and guide individual therapy.

1. Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia and is associated with an increased risk of stroke, heart failure, and mortality. Current therapies, including anti-arrhythmic drugs and pulmonary vein isolation, remain suboptimal, with recurrence in 20%–40% of patients. Personalised computational models, or cardiac digital twins, provide a promising avenue to improve AF management by tailoring therapy to individual patient data [1].

A key challenge in developing such models is the uncertainty inherent in clinical measurements used for calibration. In particular, electrogram recordings of AF cycle length (AFCL) are often noisy, spatially limited, and variable across regions of the atria [2]. This uncertainty can propagate through the modelling pipeline, potentially misrepresenting AF dynamics and reducing the accuracy of therapy guidance. Addressing these uncertainties is therefore essential for reliable model predictions and clinical translation.

In this study, we present an automated workflow to generate patient-specific biatrial models directly from AF electroanatomic mapping data. AF cycle length (AFCL) measurements from basket recordings were interpolated across biatrial anatomies using Gaussian Process Manifold Interpolation and used to calibrate homogeneous AFCL simulations. Comparing calibrated simulations and baseline simulations to our clinical data enables understanding AF dynamics. By quantifying and incorporating these uncertainties, we aim to improve the robustness of personalised models for guiding AF treatment.

2. Methods

Our approach is to analyse clinical recordings; generate anatomical models; calibrate these models with uncertainty; simulate atrial fibrillation; test the contributions of atrial fibrillation dynamics and their rate response on model predictions; and validate these calibrated models against clinical ranges of behaviours. A workflow of our methodology can be seen in Figure 1.

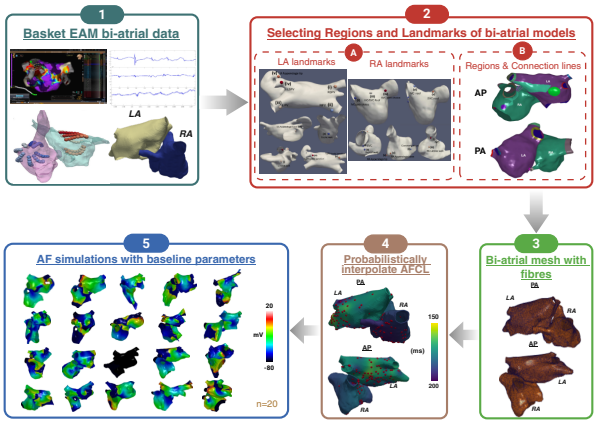

Figure 1. Overview of model construction pipeline.

2.1. Clinical Recordings

Twenty patients undergoing catheter ablation for atrial fibrillation at Stanford University were included in this study. We analysed atrial fibrillation data collected from the left atria using a constellation catheter (Boston Scientific). These recordings were each 60s duration, and there were a total of 96 epochs of data to analyse across the 20 patients, corresponding to different locations of the basket catheter. For each case, we exported the electroanatomical geometry, the roving electrode locations, surface electrode locations, and unipolar signals.

2.2. Calculating AF Cycle Length Uncertainty Quantification

We calculated atrial fibrillation cycle length (AFCL) for each unipolar signal using autocorrelation [3] (Figure 2). This technique identifies the interval shifts corresponding to the highest correlation of the signal. We considered a histogram of these values and calculated the value corresponding to the lowest 5% (to eliminate the effects of noise) which were then excluded.

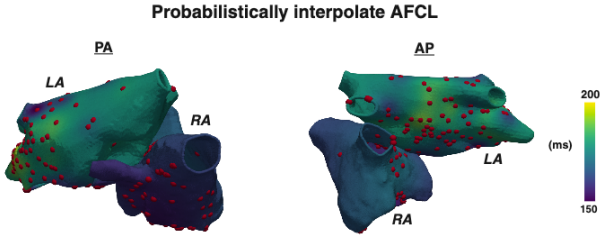

Figure 2. Probabilistic interpolation of AF cycle length (AFCL). Interpolated AFCL maps of the LA and RA in posterior–anterior and anterior–posterior views. Red dots indicate CL points.

2.3. Mapping AFCL to Atrial Geometry

AFCL values from basket recordings were spatially mapped onto the atrial surface using Gaussian Process Manifold Interpolation (GPMI). This approach accounts for sparse and noisy data, enabling interpolation of AFCL across the biatrial geometry while preserving smoothness and continuity.

2.4. Generating Anatomical Models from Electroanatomic Data

Patient-specific atrial anatomies were reconstructed from electroanatomic maps (EAM). Anatomical meshes were pre-processed and rotated in Blender, where they were rotated and aligned to ensure that the left and right atria were in the correct anatomical orientation. Biatrial computational models were built using the AtrialMTK toolkit [4]. Fibre anisotropy was assigned using a rule-based approach and baseline electrophysiology was represented with the Courtemanche et al. human atrial ionic cell model using default ionic conductances [5].

2.5. Calibrating Models to Homogenous AFCL

The simulations calibrated to a homogeneous AFCL used the mean AFCL values extracted directly from the raw clinical data. Ionic conductances in the Courtemanche atrial cell model were systematically adjusted so that the simulated AFCL reproduced the mean clinical AFCL for each atrium (RA and LA). Calibration was performed separately for the right atrium (RA) and left atrium (LA), with the resulting conductance scaling applied homogeneously across each respective atrium. This approach ensured that the simulated atrial cycle length best represented the patient-specific clinical recordings with a consistent AFCL applied throughout each atrium.

2.6. Arrhythmia Simulations

Simulations of sustained AF were performed under two conditions (Baseline and Calibrated homogeneous AFCL). The baseline simulations used the default ionic conductances from the Courtemanche ionic cell model [5] without calibration using openCARP [6]. The simulations calibrated to a homogeneous AFCL systematic adjustment of Courtemanche ionic model conductances to reproduce patient-specific AFCL values. For each patient, AFCL maps from basket recordings were compared with simulated AFCL maps to assess how calibration influenced the accuracy of AF dynamics. Comparisons included both mean AFCL values and regional distributions across the

atria. The calibrated homogeneous models were then compared with baseline models to evaluate the effect of AF dynamics, and with clinical AFCL to see if either or both simulations had a good match.

2.7. Post-Processing Simulation Data

We post-processed each AF simulation to calculate phase singularity locations, numbers and density maps. We also calculated AF cycle length using autocorrelation as per our clinical data, applied to unipolar electrograms simulated at the registered electroanatomic mapping locations. We registered these spatial maps for each simulation to an atlas anatomy using atrial coordinates. We compared simulated AF cycle length to the clinical values.

3. Results and Discussion

AF cycle length (AFCL) was calculated from biatrial basket electrogram recordings. The number of signals ranged from 64–256 (192 ± 78.4) in the LA and 64–128 (115.2 ± 28.6) in the RA. Clinical AFCL values showed variability: mean AFCL ranged from 141–207 ms in the left atrium and from 150–212 ms in the RA. Within patients, AFCL also varied regionally, with localised zones of rapid activity contributing to heterogeneous conduction patterns. This variability highlights the complex and patient-specific nature of AF, underlining the need for models that can incorporate such differences.

Simulations performed with baseline parameter values reproduced sustained AF and generated complex, chaotic activation patterns (Figure 3). These patterns are valuable for understanding AF dynamics. However, the baseline models did not reproduce the AFCL observed clinically.

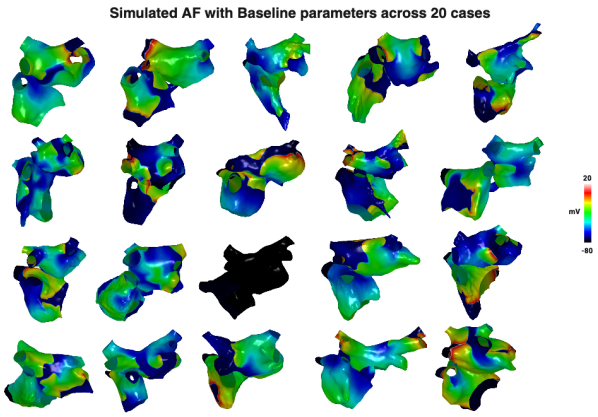

Figure 3. Simulations of AF using baseline parameters across 20 cases. Each biatrial anatomy represents an individual simulation with baseline ionic parameters from Courtemanche ionic cell model.

Simulated AFCLs clustered around 170–180 ms across patients, failing to reflect either the inter-patient variability or the regional heterogeneity seen in the clinical data. This mismatch highlighted the limitations of uncalibrated baseline models for representing patient-specific electrophysiology and motivated calibration to homogeneous AFCL values.

The simulations calibrated to a homogeneous AFCL used the mean AFCL measured in the raw clinical recordings for the RA and LA separately, with ionic conductances adjusted so that simulated AFCLs matched these values. While this approach improved the representation of atrial cycle length compared to baseline models, homogeneous calibration still did not fully capture the variability and heterogeneity of the clinical AFCL maps (Figure 4). The resulting simulations revealed differences in AF organisation compared to baseline which included changes in driver localisation and propagation patterns, but there was still mismatch with clinical AFCL.

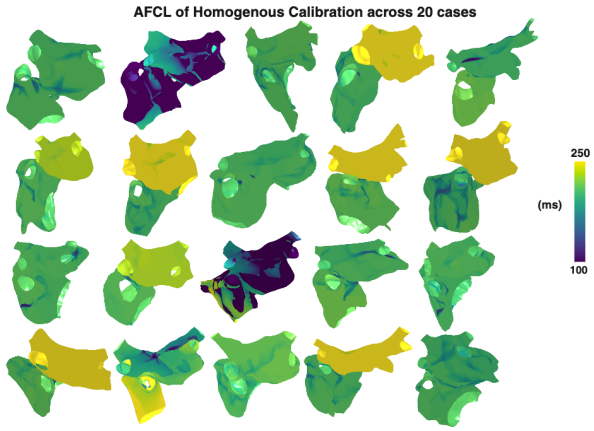

Figure 4. Atrial fibrillation cycle length (AFCL) from homogeneous calibration across 20 cases. Each biatrial anatomy representing AFCL measured in an individual simulation calibrated to their mean clinical AFCL.

A comparison between baseline and calibrated AFCL maps showed that calibration altered AF dynamics, changing the spatial organisation of activity and shifting predicted driver locations (compare Figure 5 & 6). Simulations of AF with baseline parameters had an average of 3.78 number of rotational activities across all cases (SD=2.71), and simulations of AF with homogeneous AFCL had an average of 3.78 number of rotational activities across all cases (SD=1.70). However, while calibration to homogeneous AFCL provided a closer approximation of clinical data than baseline simulations, it still failed to fully match the clinical distributions.

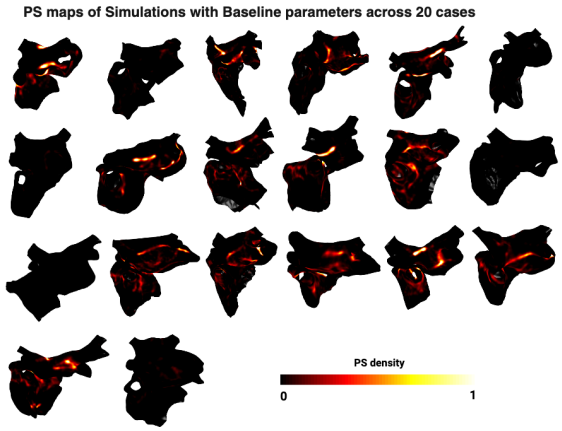

Figure 5. Phase singularity (PS) maps from simulations with baseline parameters across 20 cases. Each biatrial model represents a separate patient-specific simulation performed using baseline Courtemanche ionic model parameters. The colour scale indicates normalised PS density (0–1), highlighting regions of re-entrant activity under baseline conditions.

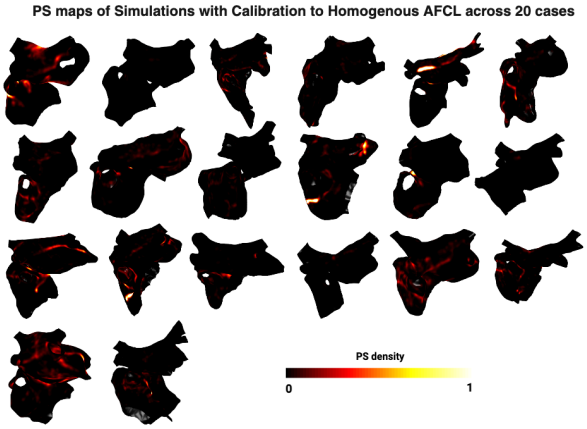

Figure 6. Phase singularity (PS) maps from simulations calibrated to homogeneous AFCL across 20 cases. Each biatrial model represents an individual patient-specific simulation calibrated to homogeneous atrial fibrillation cycle length (AFCL). The colour scale indicates normalised PS density (0–1), highlighting regions of re-entrant activity after calibration to homogeneous AFCL.

4. Conclusion & Future Research Plans

Simulations with baseline parameters reproduced AF but resulted in complex and chaotic activation patterns that did not reflect patient-specific variability. Calibration to patient-derived AFCL maps provides a pathway to investigate how functional properties influence AF dynamics, offering improved alignment with clinical observations. Extending this approach to incorporate heterogeneous regions with differing cycle lengths will be essential to better capture the spatial variability of AF and enhance the predictive power of personalised models.

Acknowledgments

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