Personalisation of a 3D Macroscopic Cardiac Electrophysiology Model for Simulation of Induced Ischemic Ventricular Tachycardia

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Despite recent efforts in cardiac electrophysiology modelling [1], there is still a strong need to make macroscopic models usable in planning and assistance of the clinical procedures. This requires model personalisation i.e. estimation of patient-specific model parameters and computations compatible with clinical constraints. Fast macroscopic models allow a quick estimation of the tissue conductivity, but are often unreliable in prediction of arrhythmias [2]. On the other side, complex biophysical models are quite expensive for the tissue conductivity estimation, but are well suited for arrhythmia predictions.

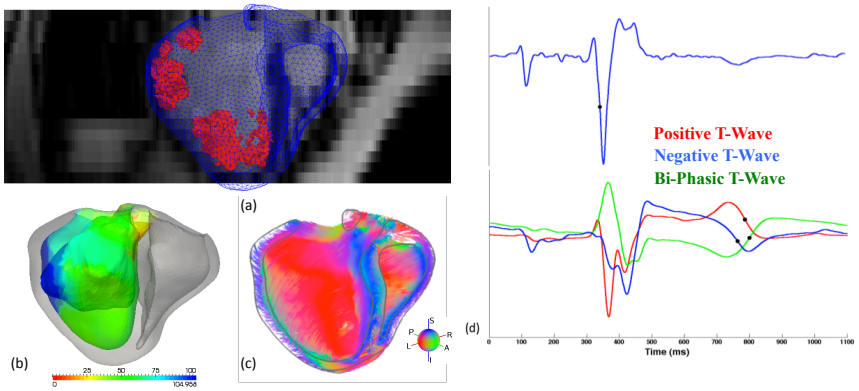

Fig. 1. (a) shows the MR data, segmented mesh with scars (in red), (b) shows XMR registration of Ensite LV surface with MR data mesh, with values projected from Ensite to MR LV surface,(c) shows the fibre orientation used, (d) shows the unipolar electrograms for detection (black dots) of depolarisation time (upper) and repolarisation time (lower) from positive(red), negative(blue) and biphasic(green) T waves.

Here we present a coupled personalisation framework, which combines the benefits of the two models. A fast Eikonal model is used to estimate the conductivity parameters, which are then used to set the parameters of a biophysical model, the Mitchell-Schaeffer (MS) model. Additional parameters related to Action Potential Duration (APD) and APD restitution curves for the tissue are estimated for the MS model [3].

This framework is applied to a clinical dataset provided with an hybrid X-Ray/MR imaging on an ischemic patient shown in figure 1. The estimated parameters using the described framework are shown in figure 2. A qualitative comparison of measured data with

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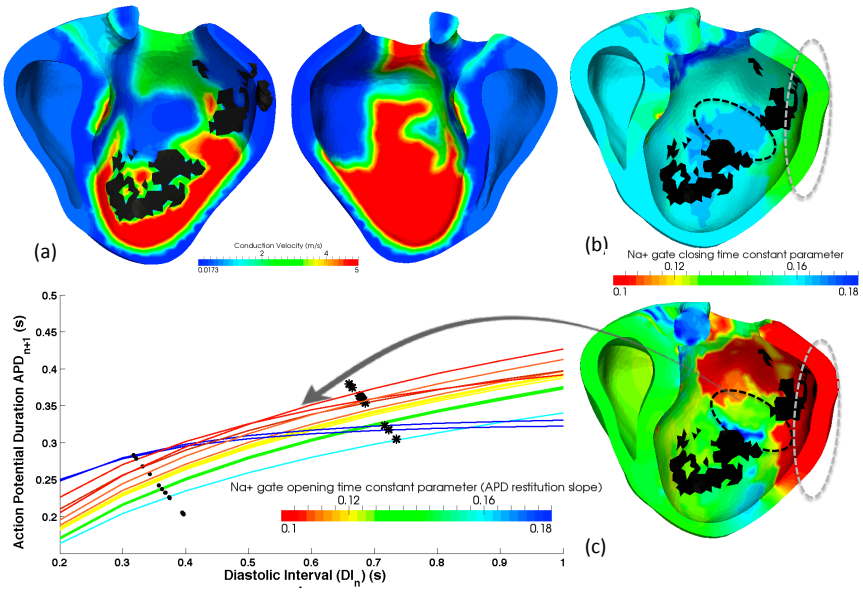

Fig. 2. (a) shows the conduction velocity estimated from AC maps, (b) shows the parameter τ_{close} estimated for APD, lower τ_{close} values has low measured APD (white circle) and vice versa, (c) shows the parameter τ_{open} estimated for APD restitution and the graph shows heterogeneity of the restitution curves for the isthmus (black circle), Lower τ_{open} values (red) have steeper slopes & higher (blue) have flat slopes for restitution curves

model simulations is shown in figure 3. This personalised MS Model is then used for *in silico* simulation of clinical Ventricular Tachycardia (VT) stimulation protocol [4] to predict the induction of VT, as shown in figure 4.

This proof of concept opens up possibilities of using VT induction modelling directly in the intervention room, in order to plan the radio-frequency ablation lines.

References

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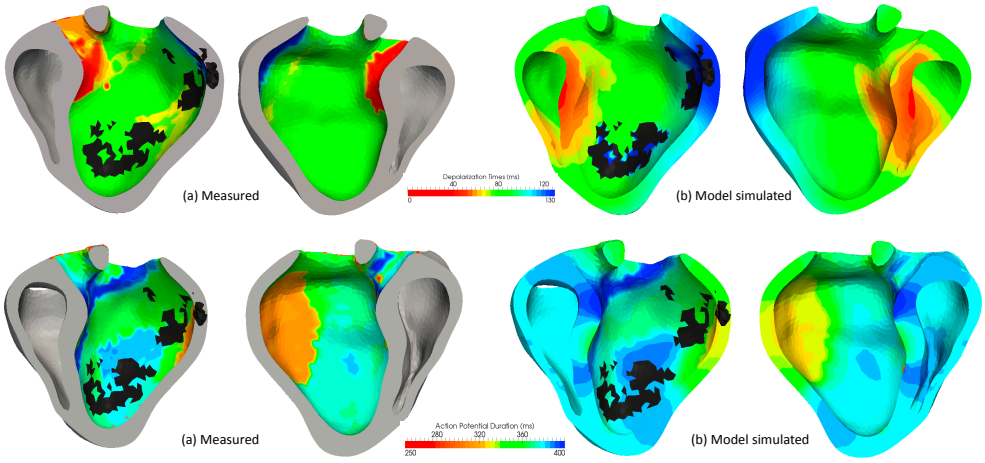

Fig. 3. Upper row shows the comparison of the measured Depolarisation Time (DT) isochrones on the LV surface only with model simulated DT isochrones on the whole heart, lower row shows the same for measured (LV surface only) and model simulated (whole heart) APD maps

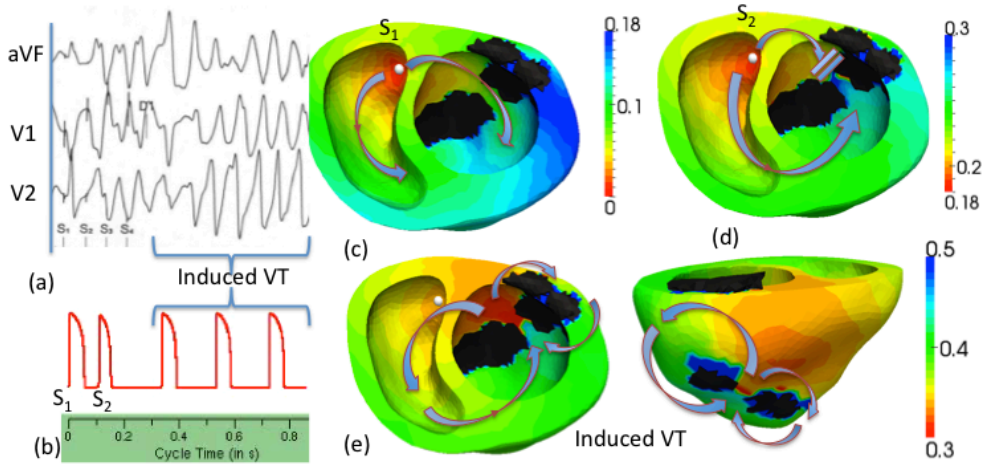

Fig. 4. (a) shows unipolar electrograms recorded for a clinical VT-Stim protocol, (b) shows the simulated protocol for two extrastimuli, with coupling interval of 100 ms. (c) show DT isochrones(in s) for S_1 stimulus and (d) shows for S_2, we have a unidirectional block created in the isthmus. (e) shows DT isochrones for induced monomorphic VT

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