Source to Sink Dynamics in *Commotio cordis*: Quantifying the Requirements for Arrhythmia Induction

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I. INTRODUCTION

Commotio cordis (CC) is a cardiac arrhythmia of varying type, duration, and severity [1] that occurs when myocardial tissue is rapidly stretched. This can trigger abnormal electrical activation during the vulnerable phases of cardiac repolarisation, leading to rapid depolarisation. Fatal arrhythmia may occur if the depolarising current (source) exceeds the repolarisation capacity of the surrounding tissue (sink), leading to a sustained arrhythmia. This is often seen in baseball players struck over the chest. While a quantified source value remains ill-defined, understanding the requirements may allow us to get closer to defining myocardial strain thresholds for arrhythmia induction, an area discussed in the literature [2-3]. Investigations at the cellular and computational levels have provided us with different estimates. Specifically, optogenetics [3] has suggested that in the left ventricle (LV) around 1300–1800 cardiomyocytes are required to overcome the sink, whereas in the right ventricle (RV) this number is reduced to approximately 511–570. Current CC research primarily investigates the mechanical response of tissue stretch under loading conditions, but a comprehensive electrophysiological whole-heart model has been lacking. This type of model is crucial for understanding how tissue strain translates into arrhythmogenic disturbances. The geometry and orientation of tissue in the heart is important not only structurally but for electrical activation. Prior studies have successfully investigated arrhythmia in computationally modelled rabbit ventricles [4] and, more recently, computational approaches toward understanding mechano-electro coupling are going from cellular to tissue level [5]. Computational modelling is a powerful tool to investigate tissue stretch in the heart during CC [6-8]. However, no prior studies have employed a whole-heart electrophysiological model derived from human CT to simulate mechano-electro coupling. This study is the first to integrate tissue strain data from a real-world CC case into an electrophysiological model [9].

II. METHODS

The PyAnsys-Heart platform was used to build a computational model and simulate electrophysiological (EP) responses of the heart based on a patient-specific human CT scan [10-11]. This model has been built based on published literature, for which implementation has been verified [12]. The SA and AV nodes, Purkinje fibers and bundle branches are all included for accurate impulse propagation. A previous mechanical impact model was used to simulate 90 mph baseball impacts to the chest, replicating a real-world case of CC [9] using a scaled version of the THUMS v7 AM50 model. The maximum principal strain (MPS) patterns from these simulations were spatially mapped to the corresponding myocardial regions of the PyAnsys-Heart model. Regions exceeding predefined strain thresholds were analysed to assess their influence on arrhythmogenesis by serving as regions for ectopic activation. The Multiphysics solver used was LS-DYNA R16 MPP double-precision with MS-MPI. LS-PrePost v4.12.0 was used for preprocessing and post-simulation analysis, and the EP solver used in this study was monodomain.

To simulate arrhythmia induction, ectopic beats were applied to both the LV and RV to evaluate their respective susceptibility to arrhythmogenesis based on regional myocardial strain distribution. This was initiated using a Ten Tusscher stimulus overlapping the trailing edge of the repolarisation wave, a method consistent with previous findings [4][13]. A stimulation amplitude of +50 mV was chosen as it replicates the peak transmembrane potential from an ectopic stimulus. Tissue volume surpassing 15%, 20%, 25%, 30%, 35% strain threshold was used, which created different volumetric patterns. These tissue strain thresholds were chosen as exploratory cutoffs to quantify strain regions based on prior computational studies of myocardial deformation [6-9]. In total, 10 simulations were conducted.

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III. INITIAL FINDINGS

In both the RV and LV, sustained re-entrant arrhythmia was induced by stimulating the trailing edge of the repolarisation wave. In Table I, re-entry was defined by sustained spiral waves following ectopic stimulation during ventricular repolarisation. In the RV, arrhythmia was induced across strain pattern thresholds up to 30%, with re-entry observed with as low as 7.4% of RV tissue volume stimulated from the 30% strain threshold case. Stimulus in the LV only triggered re-entry at the 15% and 20% strain threshold cases when the volume of tissue stimulated was 15.1% and 6.0%, respectively. There was no arrhythmic activity observed in the LV at strain threshold cases of 25% and above.

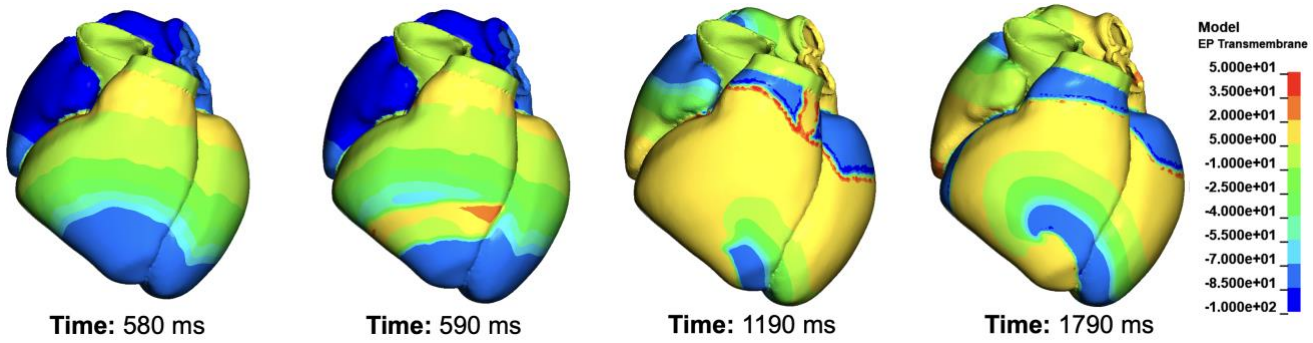

Fig. 1. Frontal view of the cardiac cycle illustrating ventricular arrhythmia. At 590 ms into the cardiac cycle a 50 mV stimulus was applied to 15.8% of RV tissue volume overlapping the trailing edge of the repolarisation wave. Time history progression into spiral wave arrhythmia is shown every 600 ms post-stimulus.

TABLE I

VOLUME OF MYOCARDIAL TISSUE EXCEEDING STRAIN THRESHOLDS AND THEIR ASSOCIATED ARRHYTHMIC OUTCOMES

	Strain Threshold (%)	**15**	**20**	**25**	**30**	**35**
Right Ventricle	Volume of Tissue Stimulated (%)	32.4	17.9	15.8	7.4	3.5
	Arrhythmic Outcome	*Re-entry*	*Re-entry*	*Re-entry*	*Re-entry*	-
Left Ventricle	Volume of Tissue Stimulated (%)	15.1	6.0	2.4	1.3	0.0
	Arrhythmic Outcome	*Re-entry*	*Re-entry*	-	-	-

IV. DISCUSSION

Based on these preliminary data for these specific cases using mechanical impact data aimed at the RV [9], our results suggest that minimum source to sink requirements among the RV is as low as 7.4% of ventricular volume. At 7.4% ventricular volume, sustained re-entry occurred in the RV, while it was unable to overcome the sink at only 3.5% volume stimulus. In contrast, the LV was able to trigger re-entry at ventricular volume sources of 6.0% and 15.1%, while no arrhythmia was observed at the higher strain thresholds (25%, 30%), which consisted of 2.4% and 1.3%, respectively. These data suggest that minimum source requirements in the RV are in the range of 3.6–7.4% of ventricular volume, while requirements in the LV are in the range of 2.5–6.0% of ventricular volume. It remains unclear whether source to sink requirements are absolute or ventricle-dependent, particularly given that the LV in this model is x1.7 times larger than the RV. We have illustrated the progression of translating tissue strain patterns seen in real-world CC impacts to a CT-based whole-heart model that can replicate the electrophysiological effects from mechano-electro coupling.

V. REFERENCES

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