Impacts of Cellular Electrophysiological Variability on Conduction Velocity Within Isolated Tissue and Depolarization and Repolarization Across the Completed Atrial Model.

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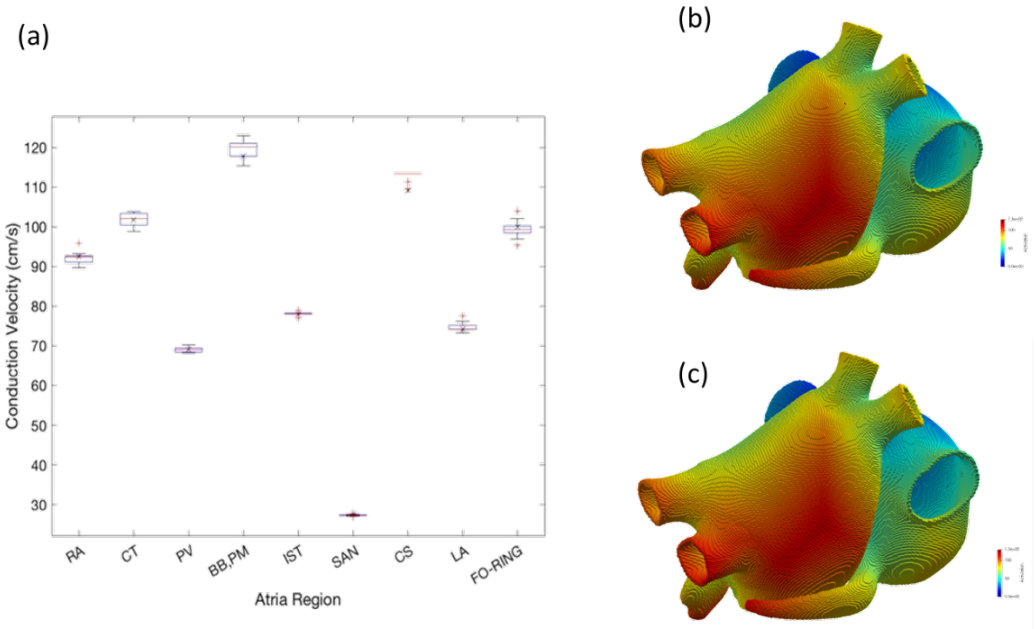
Atrial models are used to further understand the mechanisms behind common atrial arrhythmias. In order to represent the behaviour of the atria, it is important to create models with the variability observed in the human atria. Due to difficulties incorporating cellular variability, models typically assume cellular coupling masks the impact of electrophysiological variability on the cellular level. This study aims to determine the impact of cellular variability on the overall electrophysiological behaviour of the human atria. This study presents the impact of cellular variability on conduction velocity (CV) across isolated tissue samples and on the electrophysiological behaviour in the entire atria.

Variability was introduced using a population of models approach and the Courtemanche cellular model, creating 10 variable patient models. Variable model tissue CV was calibrated to within 3% of homogenous tissue. Isolated variable tissue results using the same Gi values showed the standard deviation in CV ranged from 0.19cm/s to 2.4cm/s depending on atrial region. Whole atrial simulations showed cellular variability resulted in a similar average total activation times (TAT) compared with the regionally homogenous model (117ms), varying between 117ms and 118ms. Depolarization patterns between the regionally homogenous model and the variable models remained consistent. Repolarization in the variable atrial models occurred quicker than in the homogenous model.

Cellular variability across isolated tissue can result in CV variation of up to 4cm/s. Variability typically results in increased CV compared with homogenous tissue for comparable TAT, resulting in faster depolarization across the atria, whilst maintaining the activation sequence. Repolarization across the atria is faster due to variability. Most of the observed variability in activation times is caused by anatomical variability. However, electrophysiological variability has a significant impact on the depolarisation phase. Accounting for cellular variability could result in models better representing healthy atrial behaviour and that of different arrhythmias.



Figures 1. A) variation in conduction velocity among the 10 variable patient models. B) and C) Activation maps of the homogenous (B) and a representative variable (C) atrial models respectively.