Stretch of the Papillary Muscle Insertion Region Triggers Reentrant Arrhythmia

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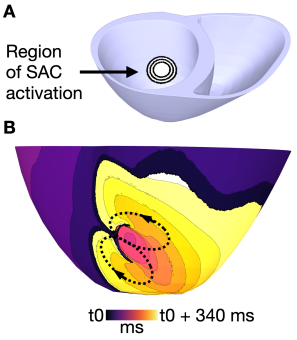
Background: The electrophysiological mechanism linking mitral valve disease, premature ventricular contractions and sudden cardiac death is unknown. Frequently hypothesized is the involvement of stretch-activated-channels (SACs), which can trigger depolarizations or cause early repolarization due to myocardial stretch. Through these mechanisms, pathological traction of the papillary muscle (PM), which has been observed in patients with mitral valve disease, may lead to abnormal electrical activity and subsequent arrhythmia.

Methods: We modeled the effect of abnormal PM traction in an electrophysiological ventricular model by activating SACs in the PM insertion region. Vulnerability windows for reentrant arrhythmia were identified by varying the timing of SAC activation in 1 ms steps from 1 to 350 ms after simulated sinus activation. We investigated conditions for reentry by varying the size of the activated SAC region (radius of 5-10 mm, Fig. A), the SAC reversal potential (E_{SAC} of -10 to -70 mV) and tissue conductivity (0-60% reduction in conduction velocity).

Results: Reentry required a region of SAC activation of at least 8 mm radius. For E_{SAC} of -10 to -30 mV, SAC activation during the T wave could trigger local depolarizations which resulted in reentry (Fig. B). For E_{SAC} of -40 to -70 mV, SAC activation during the QRS complex could cause local early repolarization which lead to reentry. Reduction in tissue conductivity influenced the reentrant vulnerability window and sustainability, but did not cause a consistent increase in inducibility.



Conclusion: Simulated stretch of the PM insertion region following sinus activation can induce reentry in a ventricular electrophysiological model.