Quantifying the Spatiotemporal Influence of Acute Myocardial Ischemia on Volumetric Conduction Speed

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Introduction: Myocardial ischemia occurs when coronary perfusion to the heart is inadequate. This perfusion shortfall initiates an electrochemical cascade known to influence the ST and repolarization phases of the ECG, but it also has a marked effect on propagation (QRS). However, because of sampling limitations, volumetric changes in propagation have not been measured. We used a large-animal experimental model and high-resolution volumetric mapping to study the effects of ischemia on conduction speeds (CS) throughout the myocardium.

Methods: We estimated CS and electrocardiographic changes (ST segments) and evaluated the spatial and temporal correlations between them across 11 controlled episodes. To estimate volumetric conduction velocity, we (1) reconstructed the activation wavefront, (2) calculated the element-wise gradient to approximate propagation direction, and (3) estimated conduction speed with an inverse-gradient technique. All steps were based on activation times estimated from electrograms recorded with intramural plunge needle arrays.

Results: We found that ischemia induces significant conduction slowing, reducing the global median speed by 25 cm/s. Furthermore, there was a high **temporal correlation** between the development of ischemic severity and CS (corr. = 0.93) through each episode. The **spatial correlations** between ST-segment changes and CS slowing were more spatially complex than expected, with substantial slowing at the periphery of the zones with ST-segment changes.

Discussion: This study is the first to report from experiments volumetric conduction speed changes during episodes of acute ischemia. We showed that conduction speed changes are temporally correlated to ischemic severity and illustrated the biphasic response long proposed from cellular studies. Furthermore, our results suggest extreme conduction slowing in regions of severe ischemia, but that the overall relationship between ischemia and CS is more complex than previously anticipated.