Dispersion in Repolarization and Arrhythmogenesis

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In Langendorff perfused pig hearts, the anterior descending artery (LAD) was cannulated and perfused. Pinacidil was administered to the LAD, sotalol to the rest of the heart, creating a repolarization gradient of about 120 ms. Premature stimuli were applied to the LAD area to induce arrhythmias. High density epicardial mapping was performed. The critical parameter to distinguish between the occurrence of reentry and ventricular fibrillation (VF) and the mere occurrence of a line of block without reentry was not the magnitude of the repolarization gradient, but the timing of arrival of the premature wavefront distal to the block relative to the repolarization time of the premature beat proximal to the block. In hearts where no VF occurred, administration of flecainide to the area distal of the block resulted in reentry and VF. In hearts where VF occurred, this could no longer be induced when flecainide was infused into the area proximal to the block. Conclusions: Not the repolarization gradient but restitution characteristics of the tissue with the shorter action potential, in combination with the time of arrival of the premature wavefront at the area with the longer action potential, determine whether reentry occurs. Conduction slowing by flecainide in the area distal to the line of block is proarrhythmic, conduction slowing proximal to the block is antiarrhythmic. Keywords: repolarization, ventricular fibrillation, conduction