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ADVERSE PARASYMPATHETIC REMODELING POST MYOCARDIAL INFARCTION AND ELECTRICAL STABILIZATION BY VAGAL NERVES STIMULATION

Poster Contributions
Poster Hall B1
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Session Title: Arrhythmias and Clinical EP: VT
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Background: Reduced parasympathetic tone increases the risk of sudden cardiac death. The aim of this study was to assess regional electrophysiological effects of vagal nerve stimulation (VNS), bilateral stellate ganglia stimulation (SGS), and neurotransmitter content in the setting of myocardial infarction.

Methods: In 15 infarcted and 7 normal porcine hearts, VNS and SGS were performed and VT inducibility assessed. A 56 electrode sock was used to obtain activation recovery interval (ARI) recordings from scar (voltage < 0.5 mV), BZ (0.5-1.5 mV), and viable (>1.5mV) areas. Acetylcholine (Ach) and norepinephrine content were analyzed.

Results: VNS prolonged global ARI of infarcted more than normal hearts (13±4 vs. 8±2% P<0.05). Ach content was greater in BZ and viable regions of infarct vs. normal hearts, 1.8±0.2 vs. 1.3±0.1 ng/mg, P=0.01. VNS prolonged BZ ARI (66±14 ms) more than scar (39±11 ms P=0.02) or viable areas (48±10 ms P=0.04) and reduced BZ dispersion of repolarization significantly compared to other regions. SGS shortened scar ARI by 63±13, BZ by 62±12, and viable myocardium by 52±7 ms and increased dispersion similarly in all regions. Norepinephrine content of viable and BZ areas of infarcts was similar to sham hearts. VNS decreased VT inducibility from 60 to 33%.

Conclusion: The greater response to VNS and Ach content of viable and BZ myocardium in infarcted hearts suggests a decrease in parasympathetic drive. Electrical stabilization of BZ is a novel mechanism for arrhythmia reduction with VNS.