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Titel des Beitrags: Abnormal sympathetic innervation of viable myocardium and the substrate of ventricular tachycardia after myocardial infarction.

Abstract: OBJECTIVES: The aim of this study was to characterize the relationship between impaired sympathetic innervation and arrhythmia with noninvasive biologic imaging in an animal model of post-infarct ventricular tachycardia (VT). BACKGROUND: Innervation might be abnormal in the normally perfused borderzone of myocardial infarction, contributing to myocardial catecholamine overexposure and arrhythmogenic risk. METHODS: Myocardial infarction was induced by mid-left anterior descending coronary artery balloon occlusion in 11 pigs. Positron emission tomography (PET) of tissue perfusion and catecholamine uptake and storage was performed with [13N]-ammonia and [11C]-epinephrine 4 to 12 weeks later. Magnetic resonance imaging and invasive electrophysiology (electroanatomic mapping, basket catheter, VT inducibility) were performed within 1 week of PET. RESULTS: When compared with a normal database of 9 healthy animals, reduced perfusion was observed in 37 +/- 7% of the left ventricle (LV). Epinephrine retention was reduced in 44 +/- 7% of LV, resulting in a perfusion/innervation mismatch of 7 +/- 4% LV. Sustained monomorphic VT was inducible in 7 of 11 animals. These animals showed a larger perfusion/innervation mismatch (10 +/-
4% vs. 4 +/- 2% LV for animals without VT; p = 0.02). Regionally, the degree of perfusion/innervation mismatch did not correlate with wall thickness or thickening but showed a significant correlation with reduced myocardial voltage (r = 0.93; p = 0.001) and with the site of earliest VT activation (chi-square 13.1; p< 0.001). CONCLUSIONS: Noninvasive mapping of cardiac sympathetic nerve terminals reveals regionally impaired catecholamine uptake and storage in the normally perfused borderzone after experimental myocardial infarction. These areas might be useful to characterize the individual risk for ventricular arrhythmia.