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Titel des Beitrags: Sympathetic nerve damage and restoration after ischemia-reperfusion injury as assessed by (11)C-hydroxyephedrine.

Abstract: An altered state of the cardiac sympathetic nerves is an important prognostic factor in patients with coronary artery disease. The aim of this study was to investigate regional sympathetic nerve damage and restoration utilizing a rat model of myocardial transient ischemia and a catecholamine analog PET tracer, (11)C-hydroxyephedrine ((11)C-HED). Transient myocardial ischemia was induced by coronary occlusion for 20 min and reperfusion in male Wistar rats. Dual-tracer autoradiography was performed subacutely (7 days) and chronically (2 months) after ischemia, and in control rats without ischemia using (11)C-HED as a marker of sympathetic innervation and (201)TI for perfusion. Additional serial in vivo cardiac (11)C-HED and (18)F-FDG PET scans were performed in the subacute and chronic phases after ischemia. After transient ischemia, the (11)C-HED uptake defect areas in both the subacute and chronic phases were clearly larger than the perfusion defect areas in the midventricular wall. The subacute (11)C-HED uptake defect showed a transmural pattern, whereas uptake recovered in the subepicardial portion in the chronic phase. Tyrosine hydroxylase antibody nerve staining confirmed regional denervation corresponding to areas of decreased (11)C-HED uptake. Serial
in vivo PET imaging visualized reductions in the area of the (11)C-HED uptake defects in the chronic phase consistent with autoradiography and histology. Higher susceptibility of sympathetic neurons compared to myocytes was confirmed by a larger (11)C-HED defect with a corresponding histologically identified region of denervation. Furthermore, partial reinnervation was observed in the chronic phase as shown by recovery of subepicardial (11)C-HED uptake.