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Postischemic recovery of mitochondrial adenine nucleotides in the heart.
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BACKGROUND. Adenine nucleotides (AdNs) are lost from the mitochondrial fraction of the heart cell during ischemia. It is unknown whether this pool of AdNs can be replenished after reperfusion. The purpose of this study was to evaluate the postischemic recovery of the mitochondrial AdN pool. METHODS AND RESULTS. The left anterior descending coronary artery (LAD) of the canine heart was occluded for 30 minutes followed by either no reflow, 30-minute reflow, 1-day reflow, or 7-day reflow. Systolic shortening in the LAD-supplied region was absent during occlusion but recovered to approximately 30% of preocclusion values during early reperfusion. Mitochondrial and tissue AdNs (ATP, ADP, and AMP) were determined in the LAD-supplied and left circumflex-supplied (control) regions of the heart. The AdN content (expressed as percent of control values) of mitochondria from the LAD region was 55 +/- 10% (p less than 0.002), 64 +/- 7% (p less than 0.001), 81 +/- 6% (p less than 0.03), and 94 +/- 8% for the no-reflow, 30-minute-reflow, 1-day-reflow, and 7-day-reflow groups, respectively. The AdN content (expressed as percent of control values) of tissue samples from the LAD region was 52 +/- 9% (p less than 0.002), 48 +/- 12% (p less than 0.02), 68 +/- 5% (p less than 0.002), and 70 +/- 9% for the no-reflow, 30-minute-reflow, 1-day-reflow, and 7-day-reflow groups, respectively. There was a good correlation between mitochondrial and tissue AdN ($r = 0.95$). Using initial exchange rates, adenine nucleotide translocase activities of mitochondria from the LAD and control regions were not significantly different. State 3 respiration of LAD mitochondria was depressed (approximately 25%, p less than 0.05) only in the no-reflow group. Acceptor control ratios of the LAD mitochondria were not significantly different from control values in any group. CONCLUSIONS. After 30 minutes of regional ischemia, postischemic restoration of the mitochondrial AdN pool occurs between 1 and 7 days; this restoration is preceded by recovery of respiratory and adenine nucleotide translocase functions. Although the abnormally low levels of AdN persist in the mitochondrial compartment during the early reperfusion period, postischemic contractile dysfunction cannot be explained by depressed mitochondrial respiratory activity.