ABSTRACT

Neonatal hearts exhibit higher resistance to ischemia-reperfusion (I/R) injury and cannot be further protected by ischemic preconditioning (IPC). Nothing is known about ischemic postconditioning (IPoC) in neonatal hearts yet. Rat hearts isolated on postnatal days 1, 4, 7 and 10 were perfused according to Langendorff. Hearts were exposed to 40 or 60 min of ischemia and reperfusion up to the maximum recovery of developed force. IPoC was induced by protocols 3x10s, 3x30s, 3x60s and 5x10s. Tolerance to ischemia did not change from day 1 to day 4 but decreased to days 7 and 10. On day 10, none of the IPoC protocols 3x10s, 3x30s and 3x60s led to significant protection, not even when the ischemia was prolonged to 60 min. The 3x30s protocol (the most effective from the previous) was also applied on days 1, 4 and 7 without any significant effect. However, in the next series of experiments, protocol 5x10s had significant protective effect on day 10. IPC and IPoC in adult hearts act through mitochondrial-K-ATP channels and nitric oxide (NO). Surprisingly mito-K-ATP blocker (5-HD) administered 5 min before ischemia and during first 20 min of reperfusion had no effect on neonatal resistance or on IPoC on day 10. Another group of hearts was used for analysis of 3-nitrotyrosine (3-NT) and serum samples were taken to measure serum nitrates. Significant difference was found in serum nitrates between days 1 and 10 but not in tissue 3-NT amount. We found that neonatal hearts during the period of their high resistance cannot be further protected by IPoC. Difference in NO production was found, however neither mito-K-ATP nor ROS seem to play role in the neonatal resistance to I/R injury.