Fast rewarming after deep hypothermic circulatory arrest in rats impairs histologic outcome and increases NFκB expression in the brain.

Deep hypothermia is used as a neuroprotectant during cardiac surgery utilizing deep hypothermic circulatory arrest (DHCA), although the ideal rewarming strategy is not known yet. Some of the neuroprotective properties of hypothermia seem to be mediated by Nuclear Factor Kappa B (NFκB) as an important transcription factor. The current study was designed to investigate the effect of the rewarming rate on histologic outcome and cerebral NFκB expression one day following DHCA in rats. With IRB approval, 20 rats were cannulated for cardiopulmonary bypass (CPB), cooled to a rectal temperature of 15-18°C, subjected to 45 min of DHCA and randomly assigned to either a slow (40 min) or a fast (20 min) rewarming protocol. At 24 hours post DHCA, the number of eosinophilic neurons was analyzed with hematoxylin and eosin (HE) staining, and NFκB expression immunohistochemically. The two experimental groups were compared with untreated control rats. HE staining showed more eosinophilic neurons in the motor cortex following fast rewarming (60 [15-388]) compared to slow rewarming (15 [10-21]) (p<0.05). Neuronal expression of NFκB was increased in the fast rewarming group in both brain areas, the motor cortex (fast: 258 [135-393]; slow: 165 [80-212]; control: 73 [44-111]) as well as the hippocampus (fast: 243 [209-314]; slow: 202 [187-239];
control: 86 [68-108]) (p<0.05). Hyperthermic episodes were strictly avoided. Fast rewarming with strict avoidance of hyperthermia after DHCA in rats was accompanied by pronounced histologic damage and accentuated cerebral NFkB expression.