Non-hypoxic preconditioning of myocardium against postoperative atrial fibrillation: Mechanism based on enhancement of the antioxidant defense system

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Oxidative stress underlies postoperative atrial fibrillation and electrophysiological remodelling associated with rapid atrial pacing. An increasing body of evidence indicates that the formation of reactive oxygen species (ROS) released following extracorporeal circulation are involved in the structural and functional myocardial impairment derived from the ischemia-reperfusion cycle. ROS behave as intracellular messengers mediating pathological processes, such as inflammation, apoptosis and necrosis, thereby participating in the pathophysiology of atrial fibrillation. Thus, increased superoxide (O2· -) production has been found in isolated atrial cardiomyocytes from patients with atrial fibrillation. Therefore, it seems reasonable to assume that the reinforcement of the antioxidant defense system should protect the heart against functional alterations in the cardiac rhythm. On this line, antioxidant enzyme induction through in vivo exposure to moderate concentration of ROS is associate