Cardiac Tissue Preconditioning

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Preconditioning was first described by Murry and his colleagues in 1986 and later was confirmed by numerous other researchers. They have concluded that “Hypoxia as a result of ischemic preconditioning could be a powerful method for limiting ischemia-induces tissue necrosis”.

It has been well documented that brief episodes of ischemic-reperfusion, known as ischemic preconditioning (IPC), through the release of various endogenous cardioprotective substances, such as adenosine, nitric oxide (NO) and reactive oxygen species (ROS) protect the heart in a biphasic profile; both early and late phase of preconditioning. A powerful form of endogenous protection against myocardial infarction, has also been demonstrated in several animal species and recently, in isolated human cardiomyocytes.

Since, IPC have a limit in medicine realm, its induction with the new safe methods and compositions have a special relevance. Therefore, it has been indicated that hyperoxia, as an oxidative stress, instead of IPC protected the isolated hearts as the same as hyperbaric hyperoxia against infarction and arrhythmias in a biphasic mode. Recently in our lab, experiments have been done and shown that normobaric hyperoxia could also protect the rat brain and kidneys.

Clinical application: It has been documented that hyperbaric hyperoxygenation therapy is a safe for human cases if it is administrated according to the standard protocols, oxygen pressure less than 3ATA and at least for 2 hours. Moreover, Diana L. sterling et al reported that administration of hyperbaric hyperoxia during regional myocardial ischemia or reperfusion and/or during both periods reduced the cell death from ischemic – reperfusion injury. They concluded that the beneficial effects of hyperoxic therapy overcome its harmful effect from ROS production. Thus, due to simplicity and safety, normobaric hyperoxic pretreatment may be easily used for protecting of whole animals. Susanna mak et al using human cases reported that the exposure of patients with or without cardiac heart failure to normobaric hyperoxia 100% for 20 min reduced cardiac output, coronary flow and stroke volume and increased the left ventricular end diastolic pressure and ventricular relaxation time constant. They did not observe any difference in lactate transcardial gradient, as a myocardial ischemia. Therefore, they suggested that hyperoxia through the Ca^{2+} homeostasis interruption or via reaction with NO leads to those harmful effects.

Keywords: Heart, Preconditioning, Hyperoxia, Nitric oxide, Reactive oxygen species.