

this conformation. The results learnt here strength a close association between amyloidosis and atherosclerosis.

**29. (261) COBALT CHLORIDE PROTECTS THE HEART AFTER A GLOBAL ISCHEMIC INSULT.**

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**Introduction:** Ischemia-reperfusion (I/R) is one of the main cardiovascular risk factors and leads to heart contractile and energy dysfunction. I/R-induced damage is reduced by ischemic preconditioning. CoCl<sub>2</sub> has properties to function as a postconditioning agent, since it can trigger transcriptional changes that resemble the response to a hypoxic event under normoxic conditions.

**Objectives:** To evaluate CoCl<sub>2</sub> as a postconditioning therapeutic tool after a myocardial and arterial I/R.

**Materials and Methods:** Isolated adult Wistar rats hearts were arterially perfused at 37°C by Langendorff method, paced at 3 Hz, exposed to 30 min ischemia followed by 45 min reperfusion (R) in the presence or absence of 0.23 mM CoCl<sub>2</sub> which was maintained or removed after 20 min of R.

Aortic contractility was evaluated in an isolated organ bath trough incubation with cumulative noradrenaline (NA) doses. After NA washing, 20 min of simulated arterial ischemia (SI) and R with or without CoCl<sub>2</sub> was performed, and NA response was re-evaluated.

**Results:** During R, the presence of CoCl<sub>2</sub> did not alter the cardiac resting pressure, nor the perfusion pressure, but increased the developed pressure (p<0.05) until 20 min which then descend reaching controls values. This decrease was prevented when CoCl<sub>2</sub> was eliminated at 20 min of R. CoCl<sub>2</sub> in R increased the contractile economy (P/Ht) and decreased the cardiac damaged area (p<0.05) and the incidence of arrhythmias (p<0.001).

Post-SI arterial contractility increased at the lowest NA dose but not at higher ones. CoCl<sub>2</sub> in post -SI R did not affect arterial force, but decreased NA sensitivity (EC 50: control: 10<sup>-7.5</sup>, CoCl<sub>2</sub>: 10<sup>-6.5</sup> M) and the maximum contractile response.

**Conclusion:** The use of CoCl<sub>2</sub> after an ischemic event attenuates the cardiac damage produced at least during the first 25 min of R and reduces the arterial adrenergic contractile response. These results support the use of CoCl<sub>2</sub> as a potential cardioprotective tool of clinical relevance.

**30. (267) H<sub>2</sub>O<sub>2</sub>, NO AND ONOO<sup>-</sup> IN THE CARDIAC MITOCHONDRIAL DYSFUNCTION IN A TYPE 1 DIABETES MODEL**

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**AIM:** To study the changes of mitochondrial production rates and/or steady-state concentrations ([X]<sub>ss</sub>) of O<sub>2</sub><sup>-</sup>, H<sub>2</sub>O<sub>2</sub>, NO and ONOO<sup>-</sup> in the temporal evolution of cardiac mitochondrial dysfunction in a type 1 diabetes model. **METHODS:** Diabetes was induced by a single dose of Streptozotocin (STZ, 60 mg/kg, ip) in male rats. Glycemia (mg/dl) was determined after 72 h (C:130 ± 5; DM:415 ± 23). The animals were sacrificed after 10 or 28 days of STZ-injection (7 or 25 days of hyperglycemia). Mn-SOD activity, and H<sub>2</sub>O<sub>2</sub> and NO production rates were determined in the cardiac mitochondrial fraction. [O<sub>2</sub><sup>-</sup>]<sub>ss</sub>, [NO]<sub>ss</sub> and ONOO<sup>-</sup> generation were estimated from experimental data. **RESULTS:** When animals were sacrificed 10 days after STZ-injection, heart mitochondrial NO (30%) and H<sub>2</sub>O<sub>2</sub> (117%) productions were higher and Mn-SOD activity was lower (15%) than control values. Moreover, mitochondrial [O<sub>2</sub><sup>-</sup>]<sub>ss</sub> was 2.5-fold higher in heart from diabetic rats, along with a 30% increase in [NO]<sub>ss</sub>. Thus, ONOO<sup>-</sup> production rate resulted 3 times higher. When animals were

subjected to 25 days of hyperglycemia, Mn-SOD activity was really reduced (50%). While H<sub>2</sub>O<sub>2</sub> generation was extremely augmented (128%), the increase in NO generation (23%) was similar to the one observed at 7 days. Increases in [O<sub>2</sub><sup>-</sup>]<sub>ss</sub> (350%), [NO]<sub>ss</sub> (25%), and ONOO<sup>-</sup> production (450%) were obtained. Moreover, nitration of tyrosine residues of mitochondrial proteins was observed in diabetic animals sacrificed at day 28. **CONCLUSIONS:** Heart mitochondrial production rates of H<sub>2</sub>O<sub>2</sub>, NO and ONOO<sup>-</sup> were higher in diabetic than in control animals, both after 7 and 25 days of hyperglycemia. No difference in the increase -over the control values- of [NO]<sub>ss</sub> was observed over time, while a much greater rise in [O<sub>2</sub><sup>-</sup>]<sub>ss</sub> was detected after 25 days of sustained hyperglycemia respect to the enhancement obtained at 7 days, intensifying the difference in ONOO<sup>-</sup> generation. Therefore, ONOO<sup>-</sup> generation rate is mainly controlled by [O<sub>2</sub><sup>-</sup>]<sub>ss</sub> rather than by [NO]<sub>ss</sub>.

**31. (334) HYPERBARIC OXYGENATION THERAPY IN THE TREATMENT OF COVID-19 (PRELIMINARY REPORT)**

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**Introduction:** Hyperbaric oxygenation therapy (HBOT) has been shown to reduce the production and release of pro-inflammatory cytokines. Its use in patients with CoViD-19 and hypoxemia in China showed promising results although its use was poorly evaluated. **Methods:** A randomized, controlled study was started, comparing standard care (Control Group which includes non-hyperbaric oxygen supply) versus standard care plus HBOT (Test). The HBOT was carried out with Biobarica chambers of national development and medium pressure (1.45 atm) in sessions of 90 minutes per day for at least 5 days. Baseline daily oxygen saturation (SatO2) breathing ambient air (FiO2 = 0.21%) prior to HBOT was recorded. **Results:** 18 patients were included (ratio 1:1). The comparison between the groups did not show significant differences in terms of clinical status, general compromise, laboratory, age, comorbidities or history. The evolution of the HBOT group showed a rapid increase in SatO2 with a significant difference between groups from day 4 onwards. On day 5, the HBOT group presented SatO2 94.4 ± 2.7 (91.0-98.0)% Vs 89.8±2,3 (87,0-94,0) in control group. The time to normalize oxygen Saturation was significantly shorter in the HBOT group [mean±SD (min-max)]: 3,3±1,4 (1,0-5,0) VS 5,8±1,4 (3,0-7,0) days (P=0,002). The ascending slope for SatO2 in HBOT group was significantly higher than the control group: 2,1±0,6 (1,3-3,2) VS 1,5±0,6 (0,8-2,8) %/day (P 0,04). No adverse reactions were recorded. **Discussion:** HBOT shown to be more effective than oxygen supply at ambient pressure. Although the study continues to recruit individuals, initial clinical results show a clear beneficial effect.

**Reference:** Rui-Yong C et al. Efficacy analysis of hyperbaric oxygen therapy in the treatment of severe coronavirus disease 2019 patients. Acad. J. Second Mil. Med. Univ. ; 6(41): 604-611, 2020.

**32. (429) EFFECTS OF REMOTE ISCHEMIC PRECONDITIONING ON EARLY MYOCARDIAL POST-INFARCTION REMODELING**

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**Introduction:** It is known that remote ischemic preconditioning (rIPC) reduces infarct size in experimental models of myocardial infarction (MI); while its effect is controversial in the clinical setting. Particularly, the effect of rIPC on post-infarction ventricular remodeling is unknown. The aim of this work is to evaluate the effect of rIPC on early ventricular remodeling, considering the myocardial infarction expansion.