GW26-e0103
The Effect of Coronary Artery Ligation at Different Sites on Sympathetic Remodeling Post Myocardial Infarction in Rats
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OBJECTIVES Sym pathetic remodeling plays an important role in the initiation and development of ventricular arrhythmia and sudden cardiac death (SCD) secondary to myocardial infarction (MI). The rat model of heart failure is a notable method in the study of sympathetic remodeling post MI. This model generally requires ligating the left anterior descending coronary artery (LAD) quite close to its origin, in order to produce a sufficiently large infarct size to induce discernible heart failure. However, this model is associated with high mortality. Furthermore, severe heart failure may develop confounding effects in the study of the relationship between sympathetic remodeling and cardiac arrhythmia per se. We hypothesize that ligation of the LAD at a more distal site from its origin reduces mortality and produces a similar level of sympathetic remodeling. The objective of this study is to investigate the effect of coronary artery ligation at different sites on sympathetic remodeling post MI in rats.

METHODS LAD of Sprague-Dawley rats was ligated 2mm (conventional site group, CS, n=25) or 6mm (distal site group, DS, n=25) distal to the aortic arch. A sham operated group (SO, n=10, passage of a needle beneath the LAD 2mm distal to its origin and without LAD ligation) was included. The mortality, and histological changes, including the density of tyrosine hydroxylase (TH)-positive nerve fibers, were determined at six weeks post MI induction.

RESULTS Compared with the CS group, in the DS group there was a significant reduction in total mortality (48% vs 12%, p<0.01). The reduction in mortality was predominantly due to less incidence of ventricular fibrillation within 24 hours post surgery (36% vs 8%, p<0.05). Histology confirmed MI in both CS and DS groups. The densities of TH-positive (i.e., mature) nerve fibers were more abundant in the infarct marginal zone of the CS or DS group rats compared with the corresponding zone in the SO group of animals (3589±1332 vs. 3173±775 vs. 1108±356 mm²/mmp <0.01) resp. CS vs. DS vs. SO). The DS and CS groups had similar densities of TH-positive nerve fibers (3589±1332 vs. 3173±775 mm²/mm², p=NS).

CONCLUSIONS LAD ligation at different sites resulted in similar levels of sympathetic remodeling in the infarct marginal zone at six weeks post MI. These results suggest that MI induced by LAD ligation at a more distal site with lower mortality is suitable for the study of sympathetic remodeling.

GW26-e0377
The Study of Chronic Intermittent Hypoxia Caused by Obstructive Sleep Apnea to the Rat Myocardial Cell Apoptosis and Myocardial Fibrosis
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OBJECTIVES The study of chronic intermittent hypoxia caused by obstructive sleep apnea to the rat myocardial cell apoptosis and myocardial fibrosis.

METHODS (1) Rats were randomly divided into chronic intermittent hypoxia group (CIH) and control group (NC): chronic intermittent hypoxia rats group (CIH) and control rats group (NC). The rats were placed in the same tank but as normal oxygen concentration environment. (2) Using echocardiography to determine left ventricular end-diastolic diameter (LVDD), left ventricular end systolic diameter (LVDDs), left ventricular short axis shortening rate (LVFS) and left ventricular ejection fraction (LVEF) till the rats were raised at the end of the 35th day. (3) Using heart rate to stain with H&E, TUNEL and with Pericardic SIRius, then we detected the differences in myocardial structure, in myocardial apoptosis, and in the levels of myocardial fibrosis. (4)The myocardial protein was extracted to detect by Western blot comparing the expression level of HIF-1α protein. (5) The data were analyzed by Spss 13.0. A p value less than 0.05 was considered statistically significant.

RESULTS (1) Compared with NC group, the rats of CIH group’s left ventricles were dilated with expanded left ventricular internal diameter in systole (LVDDs) (4.094 ± 1.131 mm/CIH versus 3.060±0.923mm NC, p < 0.01), while left ventricular short axis shortening rate (LVFS) (38.127 ± 11.564% CIH versus 51.170 ± 12.425 % NC, p < 0.01) and left ventricular ejection fraction (LVEF) (74.247 ± 10.345 % CIH versus 87.290 ± 9.436 % NC, p < 0.01) were both significantly reduced in CIH rats. (2) In CIH group, HE staining presented that myocardial cells were injured. Myocardial cell edema and part of cells necrosis appeared. The TUNEL positive apoptotic cells were markedly increased in the cardiac tissues of CIH-treated rats compared to control rats. Sirius staining showed a significant amount of collagen fibers in the heart of CIH rats. (3)The expression level of HIF-1α was markedly increased in the cardiac tissue of CIH rats compared to control rats by using Western blot test (0.62 ± 2.89E-0.5 CIH versus 0.45 ± 0.006 NC, P < 0.05).

CONCLUSIONS (1) CIH caused cardiac dysfunction. (2) CIH induced cardiac injuries and fibrosis. (3) CIH activated hypoxia inducible factor-1α responses, which advanced the cardiac hypoxia and inflammatory effects. Thus, the myocardial cell apoptosis and myocardial fibrosis were reflected on chronic intermittent hypoxia caused by obstructive sleep apnea.

GW26-e0803
Adipose Derived Stem Cells With Basic Fibroblast Growth Factor Improves Myocardial Repair in Rats
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OBJECTIVES To investigate whether adipose derived stem cells (ADSCs) with basic fibroblast growth factor (bFGF) could improve myocardial repair in rats.

METHODS Sixty rats with myocardial infarction (MI) were randomly divided into four groups (n=15/group): group-PBS, group-bFGF, group-ADSCs, and group-bFGF/ADSCs. Cardiac function was evaluated by echocardiogram. Bioluminescence imaging (BLI), histological analysis and immunofluorescence staining were performed to observe differentiation of ADSCs.

RESULTS The BLI signals gradually decreased in group-ADSCs and group-bFGF/ADSCs, but the signal in group-bFGF/ADSCs was constantly stronger than that in group-ADSCs (p<0.05). Injections of bFGF or ADSCs or bFGF/ADSCs significantly increased left ventricular ejection fraction (LVEF) compared with PBS (41.5±2.6%, 44.8±3.1%, 55.8±3.4% vs. 31.5±3.2% respectively, p<0.01), with implantation of bFGF/ADSCs highest. The percentage of cTnT/mRFP and SMA/mRFP+ cells in group-bFGF/ADSCs was much higher than that in group-ADSCs (9.73±1.87% vs. 4.65±2.14%, 8.34±2.38% vs. 4.12±1.87% respectively, p<0.05). Injections of bFGF or ADSCs or bFGF/ADSCs significantly decreased apoptosis compared with PBS (p<0.01, respectively, with implantation of bFGF/ADSCs lower). The infarcted size was significantly reduced by the injection of bFGF/ADSCs compared with the injection of PBS (24.34% vs. 49.68%, p<0.01), or bFGF (36.47%, p<0.01), or ADSCs (33.56%, p<0.01). The microvessel density (MVD) in group-bFGF/ADSCs was the highest (p<0.01, respectively), while there was no difference in MVD between group-ADSCs and group-bFGF.

CONCLUSIONS Administration of ADSCs with bFGF could promote the growth of microvessels and improve left ventricular function and myocardial viability in the early period of MI.

GW26-e1023
Effect of β-Blocker on the Production of Collagen I in Acute Myocardial Infarction Rat
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OBJECTIVES To study the effect of carvedilol on the production of collagen and TGF-β1 in acute myocardial infarction (AMI) rat.

METHODS Coronary ligation of left anterior descending artery 40 patients with acute myocardial infarction model in rats were randomly divided into three groups: myocardial infarction control group (MI-0), in with dose of carvedilol treatment group (MI-LC treated) and non-infarct zone (NIZ).

RESULTS The expressions of collagen I and TGF-β1 were measured by reverse transcription-polymerase chain reaction (RT-PCR). The difference in infarct zone (IZ) and non-infarct zone (NIZ)

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