induce in vitro cardiomyocytes ischemia-reperfusion injury. Propofol (5-20 μM) was added to the cell culture before and during the OGD/R phases to investigate the underlying mechanism.

RESULTS

Our data showed that OGD/R decreased cell viability, increased lactate dehydrogenase leakage, reactive oxygen species and malondialdehyde production in H9c2 cells, all of which were significantly reversed by propofol. Moreover, we found that propofol increased both the activities and protein expressions of superoxide dismutase and catalase. In addition, propofol increased FoxO1 expression in a dose-dependent manner and inhibited p-AMPK formation significantly.

CONCLUSIONS

These results indicate that the propofol might exert its antioxidative effect through FoxO1 in H9c2 cells, and it has a potential therapeutic effect on cardiac disorders involved in oxidative stress.

GW26-e2343

Echocardiography-guided percutaneous laser ablation of canine ventricular septum

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OBJECTIVES

Currently surgical myectomy and ethanol ablation are two established interventions for relieving the left ventricular outflow obstruction in hypertrophic cardiomyopathy (HCM) patients. The limitations in safety and efficacy in these interventions call for minimally invasive, potentially safer and more efficacious approach. The aim of this study is to validate the feasibility of echocardiography-guided percutaneous per-ventricular laser ablation of the canine ventricular septum.

METHODS

Six domestic dogs were chosen for the study. Laser (Nd: YAG, 800-1064 nm-wavelength, 300μm-diameter fiber, Echo Laser X4, Eletsa S.R.L., Italy) was used. The laser passed through a needle (2G, PTC, ECOCHIBA, Italy) that inserted from the right ventricle into the targeted septum under the guidance of the echocardiography via a percutaneous route. Laser ablation was performed as follows: 1 W laser for 3 and 5 min at 180 and 300 J, respectively. Echocardiography, serology examination and pathology were performed to assess the results of laser ablation.

RESULTS

There was no death or major complication, i.e. tamponade, pericardial effusion or ventricular fibrillation. The real-time echocardiography monitor of M-mode, 2D (LVEF), PW Doppler and TDI presented no significant variation before and after the laser ablation. Contrast echocardiography confirmed the perfusion defects in the ablated septal regions. The laser ablated areas were well demarcated on pathology examination and the diameters of the ablated region (mm) 4 ± 0.57 and 5.28 ± 0.83 for 3- and 5-minute ablation, respectively. Pre- and post-ablation cardiac enzymes (IU/L) were: AST: 39.17 ± 11.23 vs 153 ± 101.07 (p < 0.02), LDH: 71 ± 33.89 vs 253.33 ± 179.92 (p < 0.05), CK: 488.17 ± 192.42 vs 2775.17 ± 1399.35 (p < 0.001), and CK-MB: 174.33 ± 113.34 vs 897.17 ± 486.84 (p < 0.05). Microscopically, the ablated myocardium showed contracted coagulative changes. Nuclei disappeared and a zone of vacuoles was formed with red blood cells infiltrating into the widened intercellular space.

CONCLUSIONS

Our research showed that percutaneous laser ablation of the septum is feasible, potentially safe and efficacious and may become a viable alternative solution to septum ablation.

GW26-e2407

Protective Effects of Dihydromyricetin and Myricetin against Myocardial Ischemia/Reperfusion Injury in Vivo and In Vitro

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OBJECTIVES

The aims of this study were to determine whether Myr or DMY exert any cardioprotective effect against I/R injury and investigate the responsible underlying mechanisms.

METHODS

The study utilized an in vitro rat cardiomyocyte H9C2 model of hypoxia/reoxygenation (H/R) injury and an in vivo rat model of MI/R injury. H/R injury was determined by Cell Counting Kit-8 (CCK-8) assay and lactate dehydrogenase (LDH) leakage assay. In the in vivo experiment, histopathology staining was examined. Additionally, cardiac injury markers malondialdehyde (MDA), superoxide dismutase (SOD), glutathione reductase (GSH), myeloperoxidase (MPO), nitric-oxide synthase (NOS), Ca2+/Mg2+/ATPase and Na+/K+-ATPase were detected. TUNEL assay and caspases activation assays were used to investigate apoptosis. To assess levels of apoptotic regulators, immunocytochemical staining and real-time PCR were employed.

RESULTS

Both Myr and DMY have no cytotoxic effect at the concentrations of 0.5-80 μM Myr or 10-200 μM DMY for 24 hours in H9C2 cells. After being subject to H/R, cellular viability was significantly reduced in the H/R group (P < 0.01 compared to control), and LDH leakage was highly increased (P < 0.01 compared to control (P < 0.01). DMY or Myr post-conditioning significantly increased cell viability compared with the H/R group. DMY (50, 100 μM) or Myr (20 and 50 μM) markedly reduced H/R-induced cell death and decreased LDH leakage (P < 0.05 or P < 0.01). These results indicate that DMY and Myr significantly preserved cellular viability post-H/R injury in a dose-dependent manner. Both DMY and Myr protected myocardium against I/R (or H/R) injury by increasing NOS, SOD, GSH and ATPases activity, decreasing MDA content and MPO activity at different degrees and attenuating histopathology injury. Meanwhile, DMY and Myr inhibited the cardiomyocyte apoptosis. The level of Bcl-2 protein and mRNA were restored to the normal level by DMY or Myr pharmacological postconditioning. In contrast, the Bax protein level and mRNA level were markedly reduced by DMY and Myr pharmacological postconditioning.

CONCLUSIONS

DMY and Myr pharmacological postconditioning could protect against myocardial I/R injury in both in vivo and in vitro models, which are related to apoptosis pathway and antioxidant activity.

GW26-e2435

Role of calreticulin-induced mitochondrial damage in high glucose induced apoptosis in myocardial cells

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OBJECTIVES

To observe the effect of high glucose on the protein expression of calreticulin (CRT) and its association with cell apoptosis and mitochondrial dysfunction in the cardiomyocytes.

METHODS

AC-16 cardiomyocytes were randomly divided into normal glucose group, high glucose group, high glucose + CRT siRNA and isotonic control group. The cell apoptotic rate, reactive oxygen species (ROS), mitochondrial membrane potential level, respiratory enzyme activities, and protein expression of CRT were observed.

RESULTS

Compared with the cardiomyocytes in normal glucose group, the apoptotic rate of cardiomyocytes and ROS production increased in high glucose groups, accompanying with the decreases in the mitochondrial membrane potential level and enzyme activities of the respiratory chain. The protein expression of CRT was significantly increased in high glucose group. However, compared with high glucose group, high glucose + CRT siRNA decreased the expression of CRT and attenuated the damage of mitochondrion, but CRT siRNA did not reduce the ROS level in cardiomyocytes.

CONCLUSIONS

High glucose brought about CTR over-expression to induce myocardial injury, which may be a reason of increasing myocardial apoptosis.

GW26-e4818

The L-carnitine Ameliorates Pulmonary Arterial Hypertension by Improving Energy Metabolism Dysfunction of Right Ventricular Failure

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OBJECTIVES

L-carnitine is indispensable for energy metabolism and mitochondrial function in the myocardium. Although carnitine