

preservation of myocardium by offering additional effect on modulating mPTP opening.

Methods Anesthetized open-chest rabbits underwent 1.5-h regional ischaemia/1.5-h reperfusion and were divided into four groups: control(C), preconditioning (Pre-con), gradual reperfusion (GR), and preconditioning plus gradual reperfusion (Pre-con+ GR). Control hearts underwent no additional intervention. Preconditioning consisted of three cycles of 5 min of ischaemia and 5 min of reperfusion before the 1.5-h ischaemia. Gradual reperfusion hearts underwent 5 stages involving 10-s occlusion/50-s reperfusion, 20-s occlusion/40-s reperfusion, 30-s occlusion/30-s reperfusion, 40-s occlusion/20-s reperfusion, 50-s occlusion/10-s reperfusion starting 10 s after release of the index coronary occlusion. Preconditioning plus gradual reperfusion performed both interventions in preconditioning and gradual reperfusion. 1.5 h reperfusion later, mitochondria were isolated from the risk region myocardium, and mPTP opening was determined by using the mPTP kinetics method.

Results Preconditioning, and gradual reperfusion alone significantly limited infarct size, which averaged $7.21 \pm 4.76\%$, $5.36 \pm 1.90\%$ of left ventricular weigh, respectively, versus $11.94 \pm 3.75\%$ in controls ($p < 0.05$ vs control). Preconditioning plus gradual reperfusion averaged $7.53 \pm 3.45\%$ of left ventricular weigh offering no greater effect than preconditioning or gradual reperfusion alone ($p > 0.05$). The $t_{1/2}$ of mPTP kinetics averaged 5.37 ± 4.76 min, 5.27 ± 4.76 min, in preconditioning and gradual reperfusion, respectively, significantly higher than the value of 5.06 ± 4.76 min in controls ($p < 0.05$). The $t_{1/2}$ of mPTP kinetics averaged 6.62 ± 4.76 min in preconditioning plus gradual reperfusion, however, has no more effect than preconditioning or gradual reperfusion alone ($p > 0.05$).

Conclusions The combination of ischaemic preconditioning and gradual reperfusion has no greater effect on mitochondrial permeability pore but provides more powerful anti-ischaemic protection than either intervention alone.

e0067 ASPIRIN ATTENUATES PULMONARY ARTERIAL HYPERTENSION IN RATS BY REDUCING PLASMA 5-HYDROXYTRYPTAMINE LEVEL

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Pulmonary arterial hypertension (PAH) is characterised by increasing pulmonary pressure, right ventricular failure, and death. The typical pathological changes include medial hypertrophy, intimal fibrosis and in situ thrombosis. 5-HT and other factors contributed to the development of pathologic lesions. Aspirin (ASA), the platelet aggregation inhibitor, inhibits 5-HT release from platelet. The aim of the current study was to determine the efficacy of aspirin in preventing or attenuating pulmonary hypertension. Sprague-Dawley (SD) rats injected with monocrotaline (MCT) at day 0 developed severe PAH at day 31. Rats were randomised to receive either vehicle or different dosages of aspirin (ASA 0.5 mg/kg/d, ASA 1 mg/kg/d, ASA 2 mg/kg/d, ASA 4 mg/kg/d). Aspirin suppressed PAH and increased survival rate compared with the placebo group (84% vs 60%, $p < 0.05$). Aspirin treatment also reduced right ventricular hypertrophy and pulmonary arterioles proliferation. Plasma 5-HT measured by High Performance Liquid Chromatographic (HPLC) was decreased in aspirin treated PAH model. The degree of 5-HT reduction was associated with systolic pulmonary arterial pressure, right ventricular hypertrophy and wall thickness of pulmonary arterioles in rats. These results showed ASA treatment has effectively attenuated MCT-induced pulmonary hypertension, right ventricular hypertrophy and occlusion of pulmonary artery. The effects of ASA may be associated with reduction of 5-HT.

e0068 INVESTIGATION OF VERAPAMIL IN REVERSING ALTERATIONS OF CELLULAR ELECTROPHYSIOLOGY UNDERLYING VENTRICULAR ARRHYTHMIA IN DOGS WITH MULTIPLE ORGAN DYSFUNCTION SYNDROME

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Objective The mechanism of Verapamil in reversing alterations of cellular electrophysiology underlying ventricular arrhythmia in dogs with multiple organ dysfunction syndrome (MODS) was not reported and their relationship to arrhythmogenesis was likely very limited.

Methods 12 dogs, of weight 8.67 ± 0.75 kg, were divided into two groups: control group (n=6) and MODS group (n=6). MODS lasting for 72 h was induced. Ventricular myocytes were enzymatically isolated. Early afterdepolarizations (EAD), action potential durations (APD) and L-type calcium currents were assessed before and after Verapamil perfusion.

Results Sinus arrhythmias in all MODS dogs (100%; 6 of 6, n=6) and premature ventricular beats in 4 MODS dogs (66%; 4 of 6, n=6) were recorded, while no arrhythmias were found in control animals. The prolongation of APD associated with decreased L-type Ca^{2+} currents and frequent provocation of EAD were the typical electrophysiological alterations in myocytes of MODS dogs. The AP prolongation was shortened, L-type calcium currents was decreased, EAD was suppressed by using Verapamil (100 μ mol/l) in ventricular myocytes of MODS dogs (66%; 4 of 6, n=6). EAD could be induced after elusion of Verapamil.

Conclusions The cellular electrophysiology changes within 72 h in the heart of MODS dogs were APD prolongation, markedly decreased L-type Ca^{2+} currents as well as frequently provoked EAD. Verapamil appears to be an effective agent in reversing the alterations of cellular electrophysiology in the early stage of MODS.

e0069 THE INFLUENCE OF NETWORK BETWEEN CERVICAL VAGUS TRUNK AND FAT PADS ON SINUS NODE FUNCTION, ERP OF ATRIAL AND PULMONARY VEINS AND ATRIA FIBRILLATION

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Objective To investigate the mechanism of influence of network between cervical vagus trunk and fat pads on sinus node function, ERP of atria and pulmonary veins and inducibility and maintenance of atria fibrillation.

Methods 7 dogs, of weight 14 to 18 kg, were placed under anaesthesia using sodium pentothal 30 mg/kg, midazolam 0.4 mg/kg IV and 0.05 mg/kg/h. Bipolar electrode catheters were placed into the right atrial, right ventricular and bundle branch for mapping and stimulating. The hearts were exposed via right thoracotomy to expose the SAN-FP (sinus-atrial node fat pad) and AVN-FP (atria ventricular node fat pad). Bipolar electrodes and ten-polar electrodes were fixed on the left atrial appendage and the pulmonary veins. Comparison of sinus rate (SR), effective refractory period (ERP) of atrial and pulmonary vein, and both inducibility and maintenance of atrial fibrillation were performed before and after sequential ablation of SAN-FP and AVN-FP.

Results (1) The heart rate (HR) decreased significantly from 133.0 ± 13.5 ms and 130.0 ± 15.9 ms to 32.6 ± 20.4 ms and 85.6 ± 33.2 ms by stimulating right and left cervical vagus trunk,