and the expression of TNF-a and VCAM-1 in ileum were observed by H.E staining and immune chemical methods.

Results 12 animals in each group, 9 in group NT, 10 in group SC and 9 in group PC were successfully resuscitated; all animals were on mechanical ventilation for 2 to 4 h 5, 6 and 8 animals in each group respectively survived to the end of the experiment. The temperatures of tympanic and peritoneal cavity of animals in group NT were maintained in normal range. The tympanic temperature of animals in group SC and PC was arrived target temperatures at 29±6.55 mins and 62±8.27 mins. During the stage of maintenance of hypothermia, the tympanic and peritoneal temperatures of animals in group SC were in range 33 to 35°C, while the peritoneal temperatures of animals in group PC were in range 31 to 34°C, 1 to 2°C lower than the tympanic temperature. The scores of histological injured of ileum were 1.43±0.53 in group PC, 3.4±0.55 in group NT and 3.17±0.41 in group SC. The differences among them were significantly, PC vs SC, $p<0.000$; PC vs NT, $p<0.000$; while SC vs NT, $p=0.30$. The expression of TNF-a in ileum was 9.98±1.79% in group NT, 5.87±1.43% in group SC and 3.78±0.53% in group PC, the differences among them were significantly. The phenomenon of the expression of VCAM-1 was little like the TNF-a, 3.78±0.53% in group PC was significantly from the 8.53±1.53% in group NT and 5.92±1.06% in group SC.

Conclusion The neotype peritoneal cooling can improve the injured of ileum mucous beside quickly induce hypothermia after ROSC in rabbits.

e0224 MODEL OF CARDIAC ARREST IN RATS BY TRANSCUTANEOUS ELECTRICAL EPICARDIUM STIMULATION

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Objective To establish a new model of Cardiac Arrest (CA) in rats by transcutaneous electrical epicardium stimulation.

Methods Two acupuncture needles connected to the anode and cathode of a stimulator were transcutaneously inserted into the epicardium as electrodes. The stimulating current was steered to the epicardium and the stimulation was maintained for 3 min to induce CA. Cardiopulmonary resuscitation (CPR) was performed at 6 min after a period of nonintervention.

Results The success rate of induction was 12/20 at the current intensity of 1 mA; and reached 20/20 when the current intensity was increased to 2 mA. The average time from the electrical stimulation to CA induction was 5.10 (±2.81) s. When the electrical stimulation stopped, 18/20 rats had ventricular fibrillation and 2/20 rats had pulseless electrical activity. CPR was performed for averagely 207.4 (±148.8) s. The restoration of spontaneous circulation was 20/20. The death rate within 4 h after CA was 5/20, and the 72-h survival rate was 10/20. There were only two cases of complications, a minor muscle contraction and a minor lung lobe injury.

Conclusion The model of CA in rats induced by transcutaneous electrical epicardium stimulation is a stable model that requires low-intensity current and has fewer complications.

e0225 HYDROGEN SULFIDE INHABITS NEURONS APOPTOSIS IN RATS AFTER CARDIOPULMONARY RESUSCITATION

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Objective To investigate the effects of hydrogen sulfide (H_2S) on brain injury after cardiopulmonary resuscitation (CPR) in rats by examining neurons apoptosis.

Methods The 40 male SD rats were randomly divided into experimental and control groups equally. In control group, CPR was performed with Utstein mode at 6 min after CA. On this basis, sodium hydrosulfide was administrated to the rats after restoration of spontaneous circulation in experimental group. On seventh day after CPR, neurons apoptosis was examined using terminal deoxynucleotidyl transferase mediated dUTP biotin nick end labelling (TUNEL) staining and the expression of caspase-3 was detected by the immunohistochemical strepto avidin biotinperoxidase complex (SABC) method in cortex, hippocampus CA1 region and cerebellum of the rats.

Results 1. There were 12 and 10 rats completed the experiment in the experimental and control group respectively. Their fate between the two groups was no significant difference ($\chi^2=0.404$, $p=0.376$). 2. On seventh day after CPR, The serum concentrations of H_2S was 9.12±3.17 μmol/l in the experimental group and the contrast was 3.72±1.05 μmol/l, the difference between the two groups had statistic significance ($t=5.136$, $p=0.000$). 3. Compared with the control group, the experimental group's neurons apoptosis index and the sum of integrated optical density (IOD) of caspase-3 in cortex, hippocampus CA1 region and cerebellum were obviously reduced ($p<0.05$).

Conclusion After CPR, H_2S can inhabit neurons apoptosis and its mechanism may be through caspase-3 pathway. It may play a role in the treatment of the brain injury after CA.

e0226 EFFECTS OF BONE MARROW MESENCHYMAL STEM CELLS ON ELECTROPHYSIOLOGICAL FUNCTION IN RATS WITH MYOCARDIAL INFARCTION

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Objective Concerns that intramyocardial delivery of immature cells could cause potentially life-threatening ventricular arrhythmias have been repeatedly raised. The aim of this study is to investigate the electrophysiological and arrhythmogenic effects for MSCs therapy in AMI.

Methods GFP tagged MSCs were injected into a murine heart with left anterior descending (LAD) ligation. Two weeks after transplantation, effective refractory period (ERP), ventricular arrhythmias (VAs) inducibility and ventricular fibrillation threshold (VFT) were assessed by programmed electrical stimulation (PES), respectively. Epicardial monophasic action potential (MAP) recordings were obtained from infarcted border zone (IBZ) and none infarcted zone (NIZ) of left ventricular epicardium for calculation action potential duration (APD) and activation time (AT). Immunofluorescence and immunoblots were used to determine the expression and distribution of Cx43, collagen I and Kv4.2.

Results PES showed a significant reduced VTs, raised VFT and VERP in MSCs treated rats compared to PBS treated animals. MSCs implantation led to markedly longer APD and shorter AT in IBZ than PBS treated hearts. Histological study revealed that fibrotic area and collagen deposition in infarcted region were significantly lower in MI-MSCs group than in MI-PBS group. Abnormal alterations of Cx43 including reduction and lateralisation were significantly attenuated by MSCs treatment. Inhibition of Kv4.2 expression was partly ameliorated by MSCs therapy.

Conclusions This study provide strong evidence that MSCs implantation ameliorates interstitial fibrosis and the remodelling of gap junction and Kv4.2 expression, attenuates focal heterogeneity of reporlarisation and conduction and reduces vulnerability to VTs. These results suggest that MSC transplantation might be emerge as a new preventive strategy against VAs besides improving cardiac performance in ischaemic heart disease.

e0227 INTRACORONARY INFUSION OF MESENCHYMAL STEM CELLS REDUCES PROARRHYTHMOGENIC RISKS IN SWINE WITH MYOCARDIAL INFARCTION

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Objective To evaluate the risk of ventricular arrhythmias (VAs) after MSC transplantation in swine model with acute myocardial infarction.
Methods Swine models with myocardial infarction were created by intracoronary balloon occlusion and then received MSC solution or 0.9% sodium chloride solution via balloon catheter. 6 weeks after artery occlusion, heart rate turbulence (HRT), dispersion of APD and RT (APDd and RTd), slope of APD reconstitution curve, Threshold cycle length of APD alternan and cardiac electrophysiologic study (EPS) were used to evaluated the VAs risks. Haemodynamic study was assessed to evaluate the cardiac performances. The concentrations of collagen in non-infarcted myocardium was assayed to elucidate the degree of myocardial remodelling.
Results There were significantly abnormality of turbulence onset (TO) and turbulence slope (TS) in MI group relative to control group ($p<0.01$). MSC transplantation could ameliorate the abnormal HRT (MSC group vs MI group, $p<0.01$). The values of APD90, APDd, RT and RTd in the MI and MSC group markedly increased compared with the control group ($p<0.01$). These parameters in the MSC group were significantly lower than MI group ($p<0.05$). The slope of reconstitution curve in the MSC group was higher than control group but lower than MI group. The threshold cycle length of APD alternan in the MSC group was remarkably higher than that in the control group ($p<0.01$) and lower than that in the MI group ($p<0.05$). Inducible malignant VAs in the MSC group were remarkable lower than that in the MI group (30.8% vs 70.0%). MSCs therapy markedly improve impaired cardiac performances and reduce fibrosis deposition after MI.
Conclusions MSC intracoronary infusion does not cause proarrythmogrenic risk but tent to reduce the risk of malignant VAs. MSC therapy might be emerge as a new, safe and effective preventive strategy against VAs besides improving cardiac performance in ischaemic heart disease.

e0228 TRANSFECTION OF RECOMBINANT ADENO-ASSOCIATED VIRUS SEROTYPE 9 TO MOUSE HEART IN VIVO AND THE EFFECTS ON CARDIAC FUNCTION

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Objective To evaluate the transfection efficiency of recombinant adeno- associated virus serotype 9 carrying enhanced green fluorescent protein (rAAV9- eGFP) to mouse heart in vivo and the effects on cardiac function.
Methods 1. 16 C57BL/6 mice were transfected rAAV9-eGFP by tail injection. EGFP expression in the heart, liver, lung, kidney and brain cryosections was observed under inverted fluorescence microscope 7, 14, 21, 28 days after the injection of rAAV9-eGFP and eGFP was quantitated by Western Blot. 2. 20 C57BL/6 mice were divided into control group and rAAV9-eGFP group randomly, and were received with saline or rAAV9-eGFP. The echocardiography and haemodynamics were performed 28 days after the injection of saline or rAAV9-EGFP.
Results 1. EGFP expression in the heart reached the maximum at day 21, at the point of which the transduction efficiency of rAAV9-eGFP in myocardium was 32%. The other tissues had a little or no eGFP expression. 2. The cardiac function did not reveal significant difference between rAAV9-eGFP group and the control group after transfection ($p>0.05$).
Conclusion rAAV9-eGFP gene can be stably and efficiently expressed in mouse heart, and has no toxic effect on cardiac function.

e0229 INHIBITION OF NF-κB ATTENUATES POST-INFARCT LEFT VENTRICULAR RUPTURE AND REMODELLING IN AGED MICE BY RIBOZYME GENE TRANSFER WITH ADENO-ASSOCIATED VIRUS SEROTYPES 9

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Objective Using intravenous injection of adeno-associated virus serotypes 9 carring ribozyme gene (AAV9-R65), we examined whether inhibition NF-κB would prevent post-infarct left ventricular rupture and remodelling in aged mice.
Methods and results Old (18-month-old) C57BL/6 male mice were given AAV9-R65 by tail vein injection 16 days before operation (MI+R65). Myocardial infarction was induced by ligation of the left coronary artery in MI+R65 group and myocardial infarction (MI) group mice. NF-κB activity was inhibited in MI+R65 mice. Inhibition of NF-κB reduced cardiac rupture in MI+R65 group (15.2% vs 32.8%, $p=0.018$). Echocardiographic measurements revealed that diameter of LV was significantly decreased, and ventricular wall thickness, fraction shortening were significantly increased in MI+R65 mice compared with MI mice ($p<0.05$). MMP-9 and TNF-α were decreased in MI+R65 group ($p<0.05$). And collagen was also decreased in MI+R65 group ($p<0.05$). But there were no changes of IL-1β in MI+R65 group.
Conclusions Cardiac rupture and remodelling were attenuated in aged mice by ribozyme gene transfer with adeno-associated virus serotypes 9. It maybe caused by decreased collagen as the result of decreased MMP-9, TNF-α which proved that NF-κB signal pathway may be associated with cardiac rupture and remodelling in aged mice.

e0230 THE EFFECT OF CO-CULTURING WITH NATIVE CARDIOMYOCYTES ON ASCORBIC ACID-INDUCED CARDIOMYOGENIC DIFFERENTIATION IN EMBRYONIC STEM CELLS

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Objective Ascorbic acid has been reported to promote the differentiation of embryonic stem cells (ESCs) into cardiomyocytes (CMs). However, appropriate culture protocols are needed to improve the differentiation efficiency and produce adequate