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.

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e0005 | **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.

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e0006 | **EFFECTS OF NEOTYPE PERITONEAL COOLING ON THE INJURED OF INTESTINAL MUCOUS AFTER CARDIOPULMONARY RESUSCITATION IN RABBITS**

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**Objective** To explore whether the peritoneal cooling after cardiopulmonary resuscitation could improve the injured of intestinal mucous in rabbits.

**Methods** 36 adult New Zealand rabbits were induced ventricular fibrillation by AC current. After the restore of spontaneous

circulation (ROSC), rabbits were randomly divided into three groups according to the way of body temperature controlling, that is, nomothermia group (NT), surface cooling group (SC) and peritoneal cooling group (PC). The changing of tympanic temperature and peritoneal temperature were observed after ROSC. The animals were sacrificed by over anaesthesia after ROSC for 12 h, the end ileum was removed and fixed in formalin, the histological injured 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 min and 62±8.27 min. During the stage of maintenance of hypothermia, the tympanic and peritoneal temperatures of animals in group SC were in range 33 to 35℃, while the peritoneal temperatures of animals in group PC were in range 31 to 34℃, 1 to 2℃ 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 versus SC, p<0.000; PC versus NT, p<0.000; while SC versus 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.

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e0007 | **THE EFFECT OF OMEPRAZOLE ON THE OXIDATIVE STRESS AND ACUTE ATRIAL ELECTRICAL REMODELLING IN RABBITS**

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**Objective** To investigate the effect of omeprazole on the acute atrial electrical remodelling and oxidative stress status in rabbit atrial fibrillation (AF) model.

**Methods** 18 rabbits were randomly divided into atrial tachypacing (ATP) group, sham operating (SM) group, and atrial tachypacing with omeprazole therapy (A+O) group. In the ATP group and A+O group the right atrium was tachypaced at 500—600 bpm to induce and maintain AF for 3 h. The A+O group were given intravenous administration of omeprazole treatment (4 mg/kg) 15 min before tachypacing. The ATP group were given intravenous administration of physiological saline 10 ml 15 min before pacing. The SM group were not paced. The atrial electrophysiological indexes (AERP, Rate adaptive of AERP) were measured at different time point (baseline, 0.5 h, 1 h, 1.5 h, 2 h, 2.5 h and 3 h after pacing). Oxidative stress markers (SOD, MDA, T-NOS) in serum were measured at different time point (baseline and 3 h after pacing).

**Results** 1. Compare to SM group, the atrial effective refractory period (ERP) at a cycle length of 200 ms was decreased from 93.89±5.88 to 72.78±5.37 ms (p<0.01) after pacing in ATP group, and the Rate adaptive of ERP appeared non-performing significantly after tachypacing in ATP group (from 0.10±0.02 to 0.04±0.01, p<0.01); but no change in A+O group, with ERP and Rate adaptive of ERP averaging 100.17±8.93 ms and 0.09±0.02. The level of lipid

peroxidation index –MDA increased significantly after tachypacing in ATP group (from $1.99\pm0.51$ to $2.94\pm0.78$ nmol/ml, $p<0.05$), but no change in A+O group. 2. Compare to the result at the same time in R+O group, the ERP shortened dramatically ($p<0.05$) after tachypacing in ATP group; The Rate adaptive of ERP appeared non-performing significantly after pacing in ATP group; The level of MDA increased ($p<0.05$) after tachypacing in ATP group.

**Conclusion** Omeprazole could effectively suppressed tachypacing-induced electrical remodelling in rabbit AF model and greatly attenuated the oxidative stress by downregulating lipid peroxidation.

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e0008 | **EFFECT OF HIF-1A ON MSC TRANSPLANTATION THERAPY OF RAT ACUTE MYOCARDIAL INFARCTION**

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**Aims** To investigate the effect and mechanism of HIF-1a on rat AMI therapy by MSC transplantation. Materials and methods Rat acute myocardial infarction model is made through coronary anterior descending artery ligation. Rats are randomly divided into four groups which are sham operation group, pure infarction group, infarction & MSC transplantation group and infarction & HIF-1a transfected MSC transplantation group. Eight rats from every group are observed. The cell transplantation is carried out immediately after the acute myocardial infarction model is successfully made. The rats are put to death 4 weeks after the operation and the heart is isolated for weight measuring, heart chamber and myocardium thickness testing. We also observe the myocardial angiogenesis in and around the infarct myocardium through HE staining, and the distribution of transplanted cells in the myocardium tissue under immunofluorescence staining. Western blot and RT-PCR is used to test the expression of HIF-1a and VEGF in the myocardium.

**Results** About 37% of the operations on AMI model making are successful. More MSCs transfected with HIF-1 are alive after transplantation than other groups ($p<0.05$). Heart weight and left ventricular chamber of the rats transplanted with MSCs transfected with HIF-1a are lower and smaller than the other three groups ($p<0.05$), the thickness of the left ventricular well is much thicker than the others ($p<0.05$). Capillary regeneration in and around the infarction area is greater than the others ($p<0.05$). Higher expression of HIF-1a ($p<0.05$) and VEGF ($p<0.05$) can be detected in the myocardium of the arts transplanted with MSCs transfected with HIF-1a.

**Conclusions** HIF-1a could raise the survival rate of MSC in the infarct myocardium area. MSC transfected with HIF-1a could restrain myocardium remodelling after infarction, and raise the density of capillary around infarction, which might be the mechanism of the former.

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e0009 | **MULTIMODALITY MOLECULAR IMAGING OF ADIPOSE-DERIVED MESENCHYMAL STEM CELLS WITH VEGF IN HINDLIMB ISCHAEMIA MICE**

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**Background** Peripheral arterial disease (PAD) is highly prevalent and particularly in elders, smokers, diabetics or patients with systemic atherosclerosis. Apart from the surgery and medication, stem cell transplantation offers promising approaches for therapeutic angiogenesis and tissue repair. In this study we try to use in vivo multimodality molecular imaging strategies to investigate adipose tissue-derived mesenchymal stem cells (MSCs) survival, function and relative mechanism.

**Method** MSCs were cultured from murine adipose tissue from transgenic mice, which carried double reporter genes: firefly luciferase (Fluc) and enhanced green fluorescent protein (eGFP), by collagenase digestion method. Hindlimb ischaemia animal model was created in male nude mice by ligating the proximal and distal femoral artery. MSCs ($1\times10^5$) along with/without VEGF (0.4 ng) were transplanted into ischaemic hindlimb. The animals were subjected to be imaged by bioluminescence imaging and CT scan. Laser Doppler perfusion imaging (LDPI) were used to show the spatiotemporal images of peripheral tissue blood perfusion. Micro-CT, histological and molecular analysis were tested to confirm the cells' location and angiogenesis anatomically and mechanically. Result The colour-coded index of LDPI was significantly higher in the MSCs-transplanted group than that in the control group from day 3 to 28 post cell transplantation. On day 3 after transplantation, the bioluminescence signals in MSCs with VEGF group were $4.6\times10^6\pm2.5\times10^5$ photons/s/cm²/sr, while in MSCs group were $2.8\times10^6\pm3.1\times10^5$ photons/s/cm²/sr, respectively ($p<0.01$ vs control). The signals of bioluminescence increased gradually from POD 3 to day 21, which proved survival and proliferation of the MSCs in the host. The group treated with MSCs and VEGF showed higher signals than that injected by MSCs only, which indicated the reinforcement of VEGF. Micro-CT angiography demonstrated more angiogenesis in the hindlimbs of the treated mice on day 21, which were also confirmed by molecular analysis. Histological analysis showed that MSCs therapy recovered vessel density compared with the control group.

**Conclusion** Bioluminescence fusion with CT scan provides higher detailed 3D imaging for monitoring MSCs in vivo. Angiogenesis activator VEGF might promote MSCs' beneficial function for hindlimb ischaemia therapy.

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e0010 | **ATORVASTATIN INHIBITS OXIDISED LOW DENSITY LIPOPROTEIN INDUCED DIFFERENTIATION OF RAW2647 MURINE MACROPAHGES INTO DENDRITIC LIKE CELLS**

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Dendritic cells (DCs) are professional antigen-presenting cells and have an important role in the pathogenesis of atherosclerosis. It has been confirmed that the optimal oxLDL dose (10 ug/ml) can induce approximatelly 74% RAW264.7 cells differentiate into dendritic-like cells in our previous work. In this study, we examined whether atorvastatin could inhibit the differentiation of mature macrophages into DCs induced by oxLDL, since statins are lipid-lowering drugs. After 24 h treatment with atorvastatin (20 umol/ml), almost all the RAW264.7 cells induced by oxLDL simultaneously remained in cell size and macrophages morphology compared with those induced by oxLDL alone. Flow cytometric analysis detected reduced dendritic cell surface markers (CD40, CD86, MHC Class II and CD1d, table 1). Moreover, atorvastatin-treated RAW264.7 cells induced by oxLDL shown functional changes including increased phagocytic ability (table 2) in a time-dependent manner and reduced TNF-a as well as IL-12 p70 (table 3) productin. On the whole, these data suggest dendritic-like cells origined from macrophages induced by oxLDL treatment can be inhibited by atorvastatin and this may contribute to the effect of statins on preventing the formation of atherosclerotic plaques.

Table 1. dendritic cell surface markers expression (%) in 24 h RAW OxLDL OxLDL+atorvastatin CD40 $6.09\%\pm0.48\%$ $51.68\%\pm1.33\%$ * $40.09\%\pm1.59\%$ * CD86 $5.00\%\pm0.62\%$ $55.04\%\pm1.27\%$ * $24.29\%\pm1.30\%$ * CD1d MHC Class II $3.64\%\pm0.41\%$ $5.09\%\pm0.13\%$ $33.79\%\pm2.47\%$ * $17.10\%\pm1.42\%$ * $33.40\%\pm11.54\%$ * $22.87\%\pm6.665$ *