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±2.7 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 in range 33 to 35 and 62 in group SC. The differences among them were significantly. PC vs SC, p<0.0000; PC vs NT, p<0.0000; while SC vs NT, p=0.30. The expression of TNF-a in ileum was 5.87±1.45% in group NT and 3.17±0.41% in group SC. The differences among them were significantly. PC vs SC, p<0.0000; PC vs NT, p<0.0000; while SC vs NT, p=0.30. The phenomenon of the expression of VCAM-1 in ileum was 9.98±1.79% in group NT, 5.87±1.45% in group SC and 3.78±0.55% 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±0.55% in group PC was significantly from the 5.87±1.55% in group NT and 5.92±1.06% in group SC.

**Conclusion**

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

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**e0224**

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

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Lin Ji-yan, Li Xin, Li Hui, Wei Hong-yang, Liu Rong, Hu Chun-in, Dai Gang, Xiao Xiao-xing.

**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 cathode of a stimulator were transcutaneously inserted into the epicardium of a murine heart with 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 non-infarcted zone (NIZ) of left ventricular epicardium for calculation action potential duration (APD) and activation time (AT). Immuno-fluorescence and immunoblot were used to determine the expression and distribution of Cx43, collagen I and Kv4.2.

**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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**e0225**

**HYDROGEN SULFIDE INHIBITS NEURONS APOPTOSIS IN RATS AFTER CARDIOPULMONARY RESUSCITATION**

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Xiao-xing Liao, Jie-yan Lin, Hong-yan Wei, Hui Li, Xin Li, Rong Liu, Chun-in Hu, Guo-qing Huang, Gang Dai.

**Objective** To investigate the effects of hydrogen sulfide (H2S) 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 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 (p=0.404, p=0.376). 2. On seventh day after CPR, The serum concentrations of H2S 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, H2S can inhibit 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.