

angiography, PCI, and left ventriculography about 6 to 12 h after onset of AMI symptoms and were divided into two groups: the no-reflow group (0 to 1 grade) and reflow group (2 to 3 grade) identified by MBG. Equilibrium radionuclide angiography was performed 1 week after PCI to gain the parameters of left ventricular regional and global systolic function and systolic synchrony. All patients were reinvestigated at 6 months, and major adverse cardiac events were recorded during the 6 months after PCI.

**Results** At 6 months after AMI-PCI, the values of left ventricular end-systolic volume index, left ventricular end-diastolic volume index, wall motion score, and left ventricular end-diastolic volume pressure in the no-reflow group were significantly increased, whereas left ventricular ejection fraction, peak ejection rate, and peak filling rate of radionuclide angiography parameters were significantly decreased ( $p < 0.05$ ), respectively, compared with those in the reflow group. LrEF2-LrEF8 in the no-reflow group at 6 months after AMI-PCI were reduced ( $p < 0.05$ ) more than those in the reflow group, respectively. There was no improvement on the phase analysis in the no-reflow group at 6 months after AMI-PCI, and the values of PS FWHM and PSD were increased compared with those in the reflow group ( $p < 0.05$ ; respectively). Within the 6-month follow-up period, the incidence of major adverse cardiac events in the no-reflow group was significantly higher than in the reflow group.

**Conclusion** The infarct-related zone myocardium post-AMI still has no reperfusion status and directly causes the reduction of the regional and global left ventricular systolic performance with an increase of systolic asynchrony. It then progressively decreases the efficiency of ventricular blood ejection as well as causes ventricular remodelling with adverse long-term outcome in patients with AMI.

#### e0644 ESTABLISHMENT OF CONTRAST INDUCED NEPHROPATHY MODEL IN RATS

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**Objectives** The purpose of this study was to establish a rat model of CIN and to evaluate its efficacy.

**Methods** Totally 24 SD rats were randomly allocated into experimental group (group A,  $n=12$ ) and control group (group B,  $n=12$ ). After dehydration for 3 days, rats in group A were given intravenous MDDS, while rats in group B were given intravenous normal saline. Then, all rats got normal water-drinking to the end of study. Renal ultrasonic examination was performed to observe the morphologic changes, diameters of renal artery and blood flow in renal artery. Blood samples were taken to measure the level of serum creatinine. The tissue of kidney were incised for microscope and electron microscope study.

**Results** The dimensions of the two groups before and after dehydration were not different. It gradually enlarged after CM injection. These changes were the most obvious at 6 and 12 h, which did not recover at 24 h. The PSV, EDV, S/D and VTI were lowest at 6 h and then recover to normal level at 24 h. RI was increased after CM injection, the lowest occurred at 6 h, and recovered to normal level at 24 h. Serum creatinine was significantly elevated after dehydration, the highest level occurred at 12 h and then began to recover at 24 h. Microscope examination to renal sample at 12 h found patch disappearance of tubular structure, widely congestion at medullar area. No pathological glomerular changes were found under microscope. Electron microscope examination found desquamation, sparseness of microvillous of tubular endothelium, membrane confusion, disappearance, swelling, fragmentation of the MIT, with obstructed tubular lumen and basal membrane swelling.

**Conclusion** Combined with dehydration, intravenous injection of contrast lead to obvious acute kidney injury, with the changes of kidney tissue pathology, haemodynamics and kidney functions which are similar to the characteristics of CIN in human beings.

#### e0645 PROTECTIVE EFFECTS OF SIMVASTATIN COMBINED WITH ANISODAMINE ON MYOCARDIAL PERFUSION IN SWINE NO REFLOW MODEL

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**Objectives** To evaluate the preventive effect of simvastatin combined with anisodamine on myocardial perfusion in no reflow, and to probe the possible mechanism.

**Method** Totally 16 minipig of 30–40 Kg were randomly divided into anisodamine groups (A,  $n=8$ ) and anisodamine plus simvastatin group (A+S,  $n=8$ ). Pigs in Group A+S were pretreated with oral simvastatin for 7 days, while pigs in A groups were given placebo. Seven days later, CAG was performed, and the dopper wire was used to record blood velocity. The pressure of aorta (Pa) was monitored. PMBS was injected to establish no reflow model. Anisodamine was injected into the LAD 2 min before PBMS was injected. The TIMI blood flow, TMPG and CTFC were recorded to evaluate the myocardial perfusion. The sample of myocardium in ischaemic zone and normal zone were measured. Blood sample was taken before and after the experiment to measure the level of CK-MB, cTnI and hs-CRP. The percent of necrotic myocardium was calculated by myocardium stain method.

**Results** The TIMI blood flow and TFCs were better in Group A+S ( $p < 0.05$ ). The Pa was increased in both groups after PMBS injection at the early stage ( $p < 0.01$ ), and then it began to decrease in Group A ( $p < 0.05$ ), while it remained its high level in Group A+S ( $p = 0.042$ ). The bAPV was increased in both groups, which was more obvious in the Group A after PMBS injection. After the injection of PMBS, the hAPV was significantly decreased in both groups ( $p < 0.01$ ), but it was still higher in group A+S ( $p = 0.000$ ). The CFR was continuously decreased after the PMBS injection ( $p < 0.05$ ), but it was higher in Group A+S ( $p = 0.025$ ). The h-MR was further increased ( $p = 0.024$ ), with no difference between two groups after the PMBS injection. The level of serum cholesterol was similar between the two groups ( $p = 0.063$ ). CK-MB, TnI, hs-CRP and MDA were increased after the experiment, with the higher levels in Group A. NO was also increased ( $p = 0.000$ ), with the higher level in Group A+S ( $p = 0.006$ ). SOD was decreased ( $p = 0.000$ ) in both groups, with lower level in Group A ( $p = 0.000$ ). The infarcted size in group A was larger than that in A+S group ( $p < 0.05$ ).

**Conclusion** Simvastatin combined with anisodamine can significantly improve myocardial blood perfusion and protect the myocardium against ischaemic injury during PCI. The possible mechanism involves improving of coronary haemodynamics, anti-inflammation and antioxidation.

#### e0646 PROTECTIVE EFFECT OF INTENSIVE STATIN PRETREATMENT ON RENAL FUNCTION IN PATIENTS WITH ACUTE CORONARY SYNDROME UNDERGOING PERCUTANEOUS INTERVENTION

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**Objectives** To evaluate the protective effects of higher dose statin on renal function and the incidence of CIN.

**Methods** 228 patients with acute coronary syndrome undergoing delayed percutaneous coronary intervention were randomly divided into standard statin group (SSG n=115) and intensive statin group (ISG n=113). Patients in SSG were given simvastatin 20 mg/day and patients in ISG were given simvastatin 80 mg/day for at least 7 days before PCI, Serum creatinine was measured at admission, 24 h and 48 h after PCI, and the Creatinine clearance was calculated. The levels of hs-CRP, ICAM-1 and P-selectin were also measured.

**Results** Serum creatinine underwent significant increase after PCI, the peak value occurred at 24 h, and then began to decrease. At 48 h after PCI, the creatinine level significantly decreased ( $p<0.001$ ) to baseline level in ISG, whereas in SSG the creatinine level failed to decrease significantly. Serum creatinine at admission was not significantly different between the two groups, But at 24th and 48th hour after PCI, it were lower in ISG than SSG ( $p<0.05$  at 24th hour and  $p<0.001$  at 48th hour). The creatinine clearance significantly decreased after PCI, the lowest value occurred at 24 h, and then it began to increase. In SSG, the creatinine clearance increased significantly ( $p=0.03$ ) at 48 h, but still significantly lower than baseline level. In ISG, the creatinine clearance increased significantly ( $p<0.001$ ) at 48 h and recovered to the level at baseline. Creatinine clearance improved much more in ISG at 24 and 48 h than that in SSG ( $p<0.001$  at 24th hour and at 48th hour). Although procedure caused significant increase in hs-CRP, P-selectin and ICAM-1 ( $p<0.001$ ), the increase in ISG was smaller than SSG ( $p<0.001$ ).

**Conclusion** Pretreatment with intensive statin dosage before PCI can further decrease the occurrence of CIN. This benefit may be associated with the lowering of hs-CRP, P-selectin and ICAM levels.

**e0647 THE ANIMAL MODEL ESTABLISHMENT OF ACUTE CORONARY NO-REFLOW PHENOMENON IN YORK SWINE VIA INTRACORONARY INJECTION OF MICROSPHERE WITH BLOOD SUSPENSION**

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**Objective** To explore the fusibility of establishing animal model of no reflow phenomenon in swine by super selecting LAD with 4F catheter, and injecting polyethylene microspheres and blood suspension (PMBS) into LAD.

**Methods** Total of 15 york swines were included in this study. CAG was performed by 4F micro-catheter technique, and PMBS was injected into the superselected LAD. The coronary blood flow was evaluated by TIMI frame counts and the level of myocardial tissues perfusion was evaluated by TMPG. The model of NRP of AMI was considered as success while TIMI blood flow being less than grade 2 or TFC more than 36.2 counts or TMPG less than grade 1. Left ventriculography was performed with 4F Pigtail and left ventricular systolic, diastolic pressure was recorded. MRAP, mRVP, mPAP and PCWP were measured by swan-ganz floating catheter, and CO was measured by thermodilution. ECG and blood pressure were monitored, and platelet aggregate ratio, CK-MB, TnI and blood gas analysis were measured. Ischaemic region, normal region and borderline were scissored respectively and sent to check for pathology, and the necrotic zone of myocardium was weighed and the percentage of which in left ventricle was calculated.

**Results** According to the standards of NRP, 11 animals achieved the NRP model of AMI successfully, achievement ratio of the models was 73.3%, average times of injection of PMBS were  $3.2\pm 0.6$ . While the model of NRP was established, instant ECG showed that ST segment elevated and formed single-direction curve with high T wave, and R wave was gradually depressed. Though  $PO_2$  was decreased after establishment of NRP model, the level of which was in normal range. PAP, PCWP and LVEDP were increased at instantly, 30, 60, 120,

180 min after NRP with AMI, which had statistical significance ( $p<0.05$ ). SBP and DBP were decreased after successful establishment of NRP model. Heart rate was increased ( $p<0.05$ ) and LVP was decreased ( $p<0.05$ ) after successful establishment of NRP model. Decrease of mean perfusion pressure was negative correlation with TFCs. Pathological examination showed that myocardium fibre swelling, sarcolysis, reticular formation and local liquefaction necrosis occurred. The area of myocardiolysis in the left ventricle was 28.6%.

**Conclusion** This model had advantages of direct-viewing, simplicity, repetitiveness, mild trauma, closed chest, high achievement ratio and more similar to NRP after PCI in clinical compared with myocardium dyeing and other methods. It might provide better experimental animal model for microcirculation disturbance after AMI.

**e0648 EFFECTS OF PRETREATMENT WITH SIMVASTATIN ON THE AREA OF MYOCARDIAL INFARCTION IN REPERFUSION INJURY RABBITS AFTER ACUTE MYOCARDIAL INFARCTION**

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**Objective** To investigate the effects and mechanism of the pretreatment with simvastatin on the area of myocardial infarction in reperfusion injury rabbits after acute myocardial infarction (AMI).

**Method** 20 New Zealand white rabbits were randomly divided into four groups: group A, AMI/reperfusion; group B, pretreated with simvastatin (5 mg/kg) for 3 days before AMI; group C, treated with glibenclamide ( $K_{ATP}$  channel blocker) (5 mg/kg) before AMI, group D, treated with glibenclamide and simvastatin before AMI. Models of AMI/reperfusion were established by 180-min of coronary occlusion and 60-min of reperfusion. At the end of reperfusion, the coronary artery was reoccluded, and the risk zone was delineated with Evan' blue. Hearts were sectioned (2 mm) and incubated in 1% TTC in phosphate buffer for 20 min to define white necrotic tissue when fixed in 10% formalin for 24 h, and the level of plasma creatine kinase-MB was assessed and evaluated.

**Result** (1)The content of CK-MB was significantly decreased in group B than that in group A and group C ( $P<0.01$ ), however, it was markedly decreased in group D than those of group A, and significantly increased than that of group B. (2) The risk zone volumes were similar among all the groups. The infarct size was  $(43.6\pm 4.6)\%$  in group A, Simvastatin treatment resulted in a significant limitation of infarct size in group B ( $23.6\pm 2.8\%$  VS group A,  $P<0.01$ ), the infarct size was similar in group C ( $45.1\pm 4.5\%$ ) compared with that in group A ( $P>0.05$ ). However, it was markedly decreased in group D ( $36.8\pm 3.4\%$ ) than that of group A ( $P<0.05$ ), and significantly increased than that of group B ( $P<0.05$ ).

**Conclusion** Simvastatin significantly reduced myocardial infarct size and the level of plasma myocardial enzyme during AMI and reperfusion in rabbits, which has protective effects on ischaemic/reperfused myocardium, and the activation of ATP-sensitive K channels might be involved in this protective mechanism.

**e0649 LONG-TERM EFFECTS OF RA-URIS PACING ON LEFT VENTRICULAR REMODELLING IN PATIENTS WITH CHRONIC HEART DYSFUNCTION**

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**Objective** To assess the effects of RA-URIS dual chamber sequential pacing on left ventricular remodelling and cardiac function in SSS or AVB patients with chronic heart dysfunction.