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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 ${\mbox{\bf 0bjective}}$ 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 evaluate 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.

e0228TRANSFECTION OF RECOMBINANT ADENO-ASSOCIATEDVIRUS SEROTYPE 9 TO MOUSE HEART IN VIVO AND THEEFFECTS 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.

 ${\rm Methods}$ 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).

 ${\bf Conclusion}\,$ rAAV9-eGFP gene can be stably and efficiently expressed in mouse heart, and has no toxic effect on cardiac function.

e0229 INHIBITION OF NF-KB 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- κ B 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). 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- κ B 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