developed pressure (LVDP), the maximum change rate of left ventricular pressure rise and fall (±dp/dtmax) were recorded. The activity of creatine kinase (CK) in coronary outflow, the activity of malonyldialdehyde (MDA) and superoxide dismutase (SOD) in myocardium were dectected. The percentage of necrotic area were observed.

Results In adult rats, the content of CK (89.48±18.72 U/l vs 115.76±16.72U/l, p<0.01) and MDA (9.53±3.44 nmol/mg vs 16.84±2.29 nmol/mg, p<0.01) were significantly less in IPC group than those in I/R group. In IPC group, the activity of SOD (584.7±122.62 U/mg vs 429.46±85.24 U/mg), the recovery rate of the left ventricular function, including CO, LVDP and±dp/dtmax, were much higher than those in I/R group (78.69±9.68% vs 65.10±8.63%, 83.61±8.46% vs 67.23±8.68%, 81.68±8.68% vs 67.89±6.89%, 89.79±7.78% vs 66.79±8.46%, p<0.01). And the percentage of necrotic area were lower in adult IPC group than in I/R group (5.25±4.33 vs 14.75±8.02, p<0.01). But there were no significant changes between IPC group and I/R group in elderly rats (p>0.05). However, there were great significant changes between enhanced IPC group and IR group in elderly rats, the content of CK (88.60±28.32 U/l vs 105.76±9.64 U/l, p<0.01) and MDA (8.38±3.36 nmol/mg vs 16.80±3.06 nmol/mg, p<0.05), the activity of SOD (558.87±78.66 U/mg vs 433.75±86.65 U/mg, p<0.01), the recovery rate of the left ventricular function, such as CO, LVDP and±dp/dtmax, were much higher than those in I/R group (77.99±10.02% vs 66.26±9.78%, 85.59±6.67% vs 73.90±6.66%, 83.87±9.98% vs 68.90±8.68%, 86.01±7.66% vs 70.39±7.98%, p<0.01). The percentage of necrotic area were lower in elderly IPC group than in I/R group (7.95±6.32% vs 15.68±10.36%, p<0.01).

Conclusion The effect of IPC on ischaemic reperfused myocardium of elderly rats was weaken. Prolonged ischaemia was able to resume the protective effect of IPC on elderly rat hearts.

e0016 TONGXINLUO REDUCES MYOCARDIAL ISCHAEMIA-REPERFUSION INJURY AND NO-REFLOW BY STIMULATING THE EXPRESSION AND PHOSPHORYLATION OF ENOS VIA PKA PATHWAY

doi:10.1136/hrt.2010.208967.16

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Objective To investigate whether oral administration of Tongxinluo (TXL), a traditional Chinese medicine, at a single low loading dose 1 h before myocardial ischaemia can attenuate ischaemia-reperfusion injury by regulating endothelial nitric oxide synthase (eNOS) via protein kinase A (PKA) pathway.

Methods and results In 90-min ischaemia and 3-h reperfusion model, Minipigs were randomly assigned to four groups (n=8 in each group): (1) Sham; (2) Control; (3) TXL: 0.05 $g \cdot kg^{-1}$ of TXL was gavaged 1 h prior myocardial ischaemia; (4) TXL+H-89 (1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$, an inhibitor of PKA). TXL significantly decreased creatine kinase (CK) activity, reduced the infarct size from 78.5% to 59.2% and no-reflow area from 48.6% to 9.5% (p<0.05), while H-89 completely abolished the reduction of CK activity and necrosis size, and partially diminished the reduction of no-reflow size. TXL enhanced the PKA activity in ischaemic myocardium, increased the expression of PKA, Thr 198 p-PKA and Ser 635 p-eNOS in no-reflow area, and upregulated the expression of eNOS and Ser 1179 p-eNOS in reflow area. H-89 repressed the enhancement of PKA activity and the upregulation of eNOS and Ser 635 p-eNOS, but without great inhibition on the expression of PKA and Thr 198 p-PKA in no-reflow area, and even stimulated the expression of Ser 635 p-eNOS in reflow area.

Conclusion Pretreatment with single low loading dose of TXL 1 h before myocardial ischaemia reduces myocardial no-reflow and ischaemia-reperfusion injury by upregulating the expression of eNOS and p-eNOS (Ser 1179 and Ser 635), and this effect is partially mediated by PKA pathway.

e0017 EFFECTS OF EXTRACORPOREAL CARDIAC SHOCK WAVE THERAPY ON ANGIOGENESIS AND EXPRESSION OF VEGF IN ACUTE MYOCARDIAL INFRACTION PIGS

doi:10.1136/hrt.2010.208967.17

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Objective To investigate the effect of different methods of extracorporeal cardiac shock wave therapy on angiogenesis and expression of VEGF in acute myocardial infraction pigs, and optimise that of methodology.

Methods 25 miniature swine were randomly divided into three groups: group of cardiac shock wave therapy (n=15), positive control group (n=5), negative control group (n=5). According to the method, the animals of cardiac shock wave therapy were divided three subgroups: standard, prolonged course of treatment and extend area. The number of capillary density, mRNA of VEGF were evaluated and compared between with every group.

Results Compared with control positive group, the number of capillary density (837±54 vs 1856±78, p<0.0001) and expression mRNA of VEGF (20.52±4.94 vs 28.56±6.84) increased in the group of cardiac shock wave therapy, especially, in group of prolonged course of treatment. Whereas, there was no significance in the difference of between standard group and extend area group in capillary density (1633±24 vs 1695±32/mm^2, p>0.05) and mRNA of VEGF (26.31±7.24 vs 27.44±3.59, p>0.05).

Conclusions Successive extracorporeal cardiac shock wave therapy at early stage of acute myocardial infarction could improve myocardial micro-vascular circulation. It will be a new and non-invasive angiogenic therapy.

e0018 VAGUS NERVE-MEDIATED ELECTRICAL REMODELLING OF PULMONARY VEINS IN CHRONIC ATRIAL PACING DOGS WITH OR WITHOUT SUPERIOR VENA CAVA AND AORTIC ROOT FAT PAD

doi:10.1136/hrt.2010.208967.18

Xiong Rixin.

Objectives We aim to elucidate the relationship between vagus nerve and the electrical remodelling of pulmonary veins in vagus nerve-mediated atrial fibrillation dogs.

Methods 24 adult mongrel dogs weighing 15–20 Kg were randomly divided into sham operation group (S group, n=8), SVC-Ao fat pad removal group (R group, n=8) and SVC-Ao fat pad reserved group (Re group, n=8). After exposure of superior vena cava and aortic root fat pad, SVC-Ao fat pad was excised in R group dogs, and sewed epicardial pacing lead into high right atrium and successively paced for 6 weeks; for Re group dogs, suture of epicardial pacing leads and pace for 6 weeks, take the sham operation dogs as control. To detect the Left Superior Pulmonary Vein, Right Superior Pulmonary Vein, Left Inferior Pulmonary Vein, Right Inferior Pulmonary Vein respectively, including Sinus Cycle Length (SCL), Effective Refractory Period (ERP), dispersion of ERP (dERP), and the expression of Cx40 and Cx43 by Western Blot, observed the distribution of gap junctions at pulmonary vein sleeves by immunofluorescence and electron microscope.

Results One dog in R group died for anaesthesia, another dog in Re group had pacemaker problem that required us to remove them from the study. The SCL shortened after pacing (510±24 ms vs 430±18 ms, 380±26 ms, $p<0.05$), ERP shortened (146±18 ms vs 125±18 ms, 115±19 ms, $p<0.01$), dERP increased. The expression of Cx40 increased significantly in both experiment groups (1017.23±314.46 Int×mm^2 vs 1709.43±429.88 Int×mm^2, 2956.05±829.38 Int×mm^2 $p<0.01$), the expression of Cx43 increased significantly in above groups (915.21±338.93 Int×mm^2 vs 1859.94±412.11 Int×mm^2, 3048.83±931.35 Int×mm^2, $p<0.01$). The length and width of GJ in pulmonary vein sleeves shortened (L: 0.381±0.034 μm vs 0.390±0.117 μm, 0.260±0.069 μm; W: 23.29±3.70 nm vs 22.38±2.46 nm, 18.54±2.56 nm, $p<0.05$), and the Termination/Side (T/S) ratio of Cx40 and Cx43 decreased (Cx40: 1.34±0.11 vs 1.16±0.07, 0.98±0.06; Cx43: 1.27±0.09 vs 1.10±0.07, 0.90±0.09, $p<0.05$).

Conclusions The vagus nerve originated from SVC-Ao fat pad plays a role in pulmonary vein reconstitution, and these are part of basal elements for the development and maintenance of experimental atrial fibrillation.

e0019 STUDY ON THE CHANGES OF MATRIX METALLOPROTEINASE-9 AND HIGH SENSITIVE C-REACTIVE PROTEIN IN PATIENTS WITH CAROTID INTIMA-MEDIA THICKNESS

doi:10.1136/hrt.2010.208967.19

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Objective To investigate the relationship between matrix metalloproteinase-9, high sensitive C-reactive protein and carotid intima-media thickness.

Methods 180 patients were selected. High-resolution ultrasound was used to scan carotid, brachial arteries of all patients in order to check and measure carotid intima-media thickness (CIMT), plaques of carotid arteries and diameter of brachial arteries at rest. Patients were divided into four groups: the normal carotid intima group, the difficuse proliferative carotid intima group, the stable plaque group and the unstable plaque group. Serum levels of MMP-9, hsCRP were determined with immunological method. The relationship between the results measured by ultrasound instrument and the concentrations of MMP-9, and hsCRP was analysed.

Results The serum levels of MMP-9 and hsCRP in the proliferative carotid intima group were higher than in the normal carotid intima group ($p<0.01$). The serum levels of MMP-9 and hsCRP in the unstable plaque group and in the stable plaque group were higher than in difficuse proliferative carotid intima group ($p<0.05$). The serum levels MMP-9 and hsCRP in the unstable plaque group were higher than in the stable plaque group ($p<0.05$).

Conclusion The increased concentrations of MMP-9 and hsCRP were closely correlated with the increment of carotid intima-media thickness, the unsteady of plaque.

e0020 COMPOUND HETEROZYGOUS NOVEL SPLICING MUTATION D202SP AND MISSENCE MUTATION G272D ON KCNQ1 CAUSED JERVEL AND LANGE-NIELSEN SYNDROME IN A CHINESE FAMILY

doi:10.1136/hrt.2010.208967.20

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Background Long QT syndrome (LQTS) is an inherited cardiac disorder characterised by QT interval prolongation on ECG, ventricular arrhythmias and sudden death. Two forms have been identified: autosomal dominant Romamo-Ward syndrome (RWS) without deafness and autosomal recessive Jervell and Lange-Nielsen syndrome (JLNS) with deafness.

Methods A Chinese family with JLNS was identified. Family members were diagnosed based on the presence of a QT interval prolongation on 12-lead ECG and a history of syncope, palpatation and deafness. Mutational screening in the KCNQ1 potassium channel gene was performed by direct DNA sequence analysis and Blast with Results: A female 12-y-o proband and her 6-y-o brother were diagnosed with JLNS in May of 2009. The QTc were 0.59 s and 0.60 s for the girl and boy, respectively. Both patients had their first syncope at the age of 2. The QTc for parents was normal. The mutation detection showed two mutations: one is a novel splicing mutation, a A to G change in the position of two bases before exon 3 (A 605-2 A→G), in the acceptor site of intron 2, which is always A followed by G. Another one is a G to A change at position of 815, which is a known missense mutation G272D reported before in a RWS European patient. Both JLNS patients were compound heterozygous for these two mutations D202sp and G272D. The father carries a heterozygous D202sp only, while the mother carries a heterozygous G272D. These two mutation were absent in 100 control alleles. The parents' marriage was not consanguineous. Because of the early age of first syncope and the less effectiveness of b-blocker for JLNS, video-assisted thoracoscopic left cardiac sympathetic denervation (LCSD) was performed successfully in June of 2009. Both patients were syncope free during 1-year follow-up after surgery at the dosage of 2.2 and 1.6 mg/Kg of proparanolol for the girl and boy.

Conclusion Our results suggest that the compound heterozygous mutation D202sp and G272D caused JLNS in the siblings of this Chinese family. To our best knowledge, this is the first report of compound heterozygous splicing and missense mutation to induce JLNS so far. The results expand the spectrum of KCNQ1 mutations causing RWS and JLNS. Further mechanism exploration will deep our understanding of this rare disease.

e0021 A NOVEL APPROACH OF PROTEOMICS TO STUDY THE MECHANISM OF ACTION OF GRAPE SEED PROANTHOCYANIDIN EXTRACTS ON AORTIC ARTERIOSCLEROSIS IN DIABETIC RATS

doi:10.1136/hrt.2010.208967.21

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Objective Diabetic macrovascular complications are the leading cause of mortality in diabetic patients. To prevent the development of this disease and to improve advanced arteriosclerosis, effective therapies directed towards the key molecular target are required. Grape seed proanthocyanidin extracts (GSPE) have been reported to be effective in treating arteriosclerosis, while little is known about the functional protein changes.

Methods We used streptozotocin to induce diabetic rats. GSPE (250 mg/kg body weight/day) were administrated to diabetic rats for 24 weeks. Serum glycated haemoglobin and advanced glycation end products (AGEs) were determined. Electronic microscope was used to observe the changes of aortic ultrastructure. Immunohistochemistry was used to evaluate the receptor of advanced glycation end products (RAGE) protein expression in aortic tissue. Consequently, 2-D difference gel electrophoresis and AutoFlex martix-assisted laser desorption/ionization time-of-flight mass spectrometry with LIFT technology or liquid chromatography electrospray ionisation mass spectrometry/mass spectrometry were used to investigate aortic protein profiles among the control, untreated, and GSPE treated diabetic rats.

Results GSPE significantly decreased aortic PWV, blood pressures, aortic medial thickness ($p<0.05$), and inhibited the migration of