RESULTS Valve hemodynamic performance was assessed by ICE and invasive left heart catheterization. Mean trans-valvular gradients and effective orifice area (EOA) at baseline, 90d and 140d were within the VARC guidelines, and listed in the Table. Histopathological healing response was characterized by a thin layer of largely endothelialized collagen-rich neointimal tissue that covered the luminal surface of the braided nitinol. The polyurethane Adaptive-Seal? conformed well to the local anatomy of the aortic annulus without signs of inflammation. Thrombus was rarely seen on the smooth aortic (serosal) surface. Overall, inflammation was minimal. All leaflets were intact up to 140d without separation, fracture, or fraying of collagen.

CONCLUSIONS The current study demonstrates the feasibility of evaluation of Lotus? in a sheep model using a transfemoral approach. The Lotus valve showed normal hemodynamic performance and healing response without evidence of safety concerns.

GW27-e0772

Effects of Lipopolysaccharide on the Expression of Inflammatory Cytokines in Human Coronary Artery Smooth Muscle Cells Cultured in Vitro

Hongmei Li,[1,2] Xian Wang[1,2]
[1]Institute for Cardiovascular Disease, Dongzhimen Hospital Affiliated to Beijing University of Chinese Medicine; [2]Key Laboratory of Chinese Internal Medicine of Ministry of Education and Beijing, Dongzhimen Hospital Affiliated to Beijing University of Chinese Medicine

OBJECTIVES Based on the safe range of lipopolysaccharide(LPS) density explored in the preliminary study, we investigated the effects of different ratios of LPS at different points on the expression of inflammatory cytokines in human coronary artery smooth muscle cells(HCASMC) cultured in vitro.

METHODS The 3-5 generations of HCASMC was respectively seeded onto 6-well plates, then co-incubated with different concentration of LPS (0,0.01,0.1,0.5,1,and 10μg/ml) at different points (0,6,24,and48h). Then the expression of inflammatory cytokines were determined via enzyme-linked immunosorbent assay(ELISA).

RESULTS The effect of LPS on the expression of inflammatory cytokines in HCASMC were related with the density and action-time. The dose of (0.01,0.1,0.5,1,10)μg/ml LPS with action-time of (24,48h) could significantly induce the expression of IL-1β($P<0.05$). 0.5μg/ml LPS with action-time of (6,24,48h) could induce the expression of IL-6 and TNF-α($P<0.05$), while 1μg/ml LPS with action-time of (24,48h) could induce the expression of IL-6 and TNF-α significantly($P<0.05$).

CONCLUSIONS Under certain conditions, LPS can activate HCASMC pathway and increase secretion of specific inflammatory factors. The dose of (0.5,1)μg/ml LPS with action-time of (24,48h) can significantly induce the expression of IL-1β, IL-6 and TNF-α, which can be used in the experimental study of HCASMC cultured in the inflammation condition.

GW27-e0773

Establishment of Cell Models of Toxin-Heat Engendering Endogenous Collateral Wind Syndrome Based on LPS Induced Inflammatory Activation of Human Coronary Artery Smooth Muscle Cells

Hongmei Li,[1,2] Xian Wang[1,2]
[1]Institute for Cardiovascular Disease, Dongzhimen Hospital Affiliated to Beijing University of Chinese Medicine; [2]Key Laboratory of Chinese Internal Medicine of Ministry of Education and Beijing, Dongzhimen Hospital Affiliated to Beijing University of Chinese Medicine

OBJECTIVES To establish the cell models of toxin-heat engendering endogenous collateral wind syndrome induced by LPS to activate the TLR4-MyD88 inflammatory pathway of HCASMC, seeking a new breakthrough for the scientific research platform of endogenous collateral wind.

METHODS HCASMC were cultured in vitro. And cell viability was determined via methylthiazoletetrazolium (MTT) assay to determine the optimal concentration range of LPS for promoting the growth of HCASMC. Then co-incubated with the safe range of LPS at different points (0,6,24,and48h), the expression of inflammatory cytokines were determined via enzyme-linked immunosorbent assay(ELISA). Besides, semi-quantitative reverse transcription-PCR was performed to detect mRNA expression level of IL-6, TNF-a, IL-1β, TLR4, MyD88 and NF-κB p65. The expressions of TLR4,MyD88 and NF-κB p65 were measured by western blotting to determine the final mold conditions.

RESULTS Beyond certain density of LPS(5μg/ml,100μg/ml), the smaller action-time of LPS did not have cytotoxicity. The dose of (0.5,1)μg/ml LPS with action-time of (24,48h) could induce the expression of IL-6, IL-1βand TNF-α significantly($P<0.05$). The RT-PCR and Western blotting indicated that 1μg/ml LPS with action-time of 48h could maximum activation of TLR4-MyD88 inflammatory pathway.

CONCLUSIONS 1μg/ml LPS with action-time of 48h can ensure that the HCASMC is in a sustained high inflammatory activation state, which can be used as the best cell models of toxin-heat engendering endogenous collateral wind syndrome.

GW27-e0801

Shensong Yangxin Capsule Prevents Atrial Fibrillation by Inhibiting Atrial Fibrosis in Post-myocardial Infarction Induced Heart Failure Rats

Jin Ma,[1,2] Shiyu Ma,[3] Huiliang Qiu,[1,2] Chunhua Ding[1,2]
[1]Cardiac Electrophysiology Lab, Guangdong Provincial Academy of Chinese Medical Sciences; [2]Arrhythmia Center, Guangdong Provincial Hospital of Chinese Medicine; [3]Department of Critical-care medicine, Guangdong Provincial Academy of Chinese Medical Sciences

OBJECTIVES Atrial fibrillation (AF) is the most common arrhythmia in clinic, which is associated with myocardial infarction (MI) and heart failure (HF). Shensong Yangxin Capsule (SSYX) is a traditional Chinese medicine for treating cardiac arrthymia. Clinical studies demonstrated that SSYX can effectively reduced paroxysmal AF. Extracellular matrix fibrosis in left atrium were strongly relative to the occurrence of AF. Previous studies have suggested that SSYX can inhibit ventricular fibrosis. The purpose of the present study was to investigate whether SSYX prevents AF by inhibiting left atrial fibrosis in post MI induced HF rats, and explored the underlying molecular mechanisms.

METHODS MI was produced by ligation of the left descending coronary artery. One week after surgery, echocardiography was taken to determine cardiac function in all surviving animals. We enrolled rats with ejection fraction (EF) < 45% as HF rats in this study. Then 600 mg/Kg/d SSYX was gavaged for 4 weeks. AF inducibility and duration were detected by transesophageal programmed electrical stimulation AF inducing technology. The atrial conduction velocity was detected by multi-electrodes arrays measurements. The expression of type I and III collagen and the changes of transforming growth factor β1 (TGF-β1), matrix metalloproteinase 9 (MMP-9), and tissue inhibitor of metalloproteinase 1 (TIMP-1) in left atrial were measured by western blot.

RESULTS Four weeks after the administration, SSYX-treated rats had lower rates of AF inducibility ($33.3 \pm 5.8\%$ in SSYX vs. $70 \pm 10\%$ in MI) and shorter AF duration (52.5-303.8 s in SSYX vs. 251-1060 s in MI, $P<0.05$). The left ventricular ejection fraction and fractional shortening in SSYX were higher than MI group ($P < 0.05$). There was some decline in left atrial fibrosis areas in SSYX treated rats ($P<0.01$). Type I and III collagen in left atrium were both decreased in SSYX group compared with MI group ($P<0.05$). SSYX-treated rats had faster conduction velocities in their left atrium (21.3 ± 3.6cm/s in SSYX vs. 11.3 ± 2.5 cm/s in MI, $P<0.05$). The protein of TGF-β1, MMP-9 and TIMP-1 in SSYX group were lower than MI group, MMP-9/TIMP-1 ratio decreased.

CONCLUSIONS SSYX reduces the inducibility and duration of AF after MI by inhibiting left atrial fibrosis and improving atrial electrical conduction function. Anti-fibrotic therapy can prevent AF effectively in the early stage of MI.

GW27-e0817

Gene Delivery of CYP2J2 Ameliorates Right Ventricular Remodelling in Monocrotaline-induced Pulmonary Hypertension in Rats

Xiaoyan Yang, Changlong Zheng, Caiqian Liang, Yongbiao Zhang
Department of Emergency, the Third Affiliated Hospital of Sun Yat-sen University, Guangzhou, China

OBJECTIVES We aimed to observe the effects of cytochrome P450 epoxygenase 2J2 (CYP2J2) overexpression on right ventricular remodelling in monocrotaline-induced pulmonary hypertension in rats.

METHODS Forty SD rats were randomly divided into normal control group (NC group), monocrotaline group (MCT group), pCB6-treated group (MCT+pCB6 group) and pCB6-CYP2J2-treated group (MCT+2J2