RESULTS NVAF patients with thrombosis showed significantly higher sP-selectin (p < 0.001) than did NVAF patients without thrombosis. Allele frequencies of -2123C/G, -1817T/C were significantly higher in NVAF patients with thrombosis than in NVAF patients without thrombosis, but Thr715Pro did not show any polymorphism.

CONCLUSIONS PS gene polymorphism may contribute to thrombotic risk in AF. sP-selectin was a potential risk factor for thrombosis in AF.

GW26-e4504
Sympathetic Nerve Remodeling in Hypertensive Rabbits with Left Ventricular Hypertrophy and Heart Failure
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OBJECTIVES Sympathetic nerve (SN) remodeling plays an important role in the development of sudden cardiac death in patients with myocardial infarction. However, little has been known about the pattern of SN remodeling in patients with hypertensive left ventricular hypertrophy (LHV) or heart failure (HF). We aimed to evaluate the role and pattern of SN remodeling in hypertensive rabbit models.

METHODS Hypertensive LHV (70% diameter stenosis, n = 16) and HF (90% diameter stenosis, n = 12) rabbit models were prepared through abdominal aorta constriction, and confirmed with echocardiographic and hemodynamic measurements. The rabbits in Sham group (n = 14) received the same procedure without abdominal aorta constriction. Eight weeks after the index procedure, ex vivo electrophysiological parameters were determined. The distribution patterns and densities of myocardial SN were determined with immunohistochemistry. The myocardial levels of CAMP and norepinephrine were measured using radioimmunoassay and high-performance liquid chromatography, respectively. Finally, the protein expression level of beta-ARK1 was determined with western-blot.

RESULTS Compared to Sham group, LHV group had a significantly shorter left ventricular effective refractory period (122.75 ± 10.31 ms vs 95.71 ± 16.31 ms, P < 0.001). Six isolated hearts (6/8) in LHV group occurred inducible ventricular tachycardia or ventricular fibrillation under program stimulation, while none (0/8) in Sham group occurred inducible ventricular tachycardia or ventricular fibrillation. Meanwhile, the LVH had significantly lower levels of beta-ARK1 expression compared to Sham group.

CONCLUSIONS The present study suggests that both hypertensive LHV and HF would increase the heterogeneity of myocardial SN distributions with decreased overall SN activities, which could be translated into more vulnerable electrophysiological characteristics.

GW26-e4558
Microarray expression profile of long non-coding RNAs in human endothelial cells exposed to atheroprotective shear stress
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OBJECTIVES To identify differentially expressed long non-coding RNAs (lncRNAs) involved in flow-dependent regulation of vascular function.

METHODS Confluent human umbilical vein endothelial cells (HUVECs) were cultured under static conditions or high shear stress (HSS, 15 dyn/cm²) for 24 hours. Total RNA was isolated from 4 pairs of static control and shear-stressed HUVECs, and hybridized to array human lncRNA 8 x 60 k v3.0 chips containing probes representing 30,586 lncRNA genes and 26,109 protein coding genes. Quantitative real-time polymerase chain reaction (RT-qPCR) was used to validate the microarray results. Bioinformatics analyses including lncRNA classification and subgroup analysis, gene ontology (GO) analysis and pathway analysis were performed.

RESULTS Our data revealed that 8450 out of 30,586 lncRNAs and 19,109 out of 26,109 mRNAs examined were significantly altered in expression levels at a significance level of P < 0.05. Using the cutoff criteria of P-values < 0.05 and fold changes > 2, we identified 804 differentially expressed lncRNAs and 750 differentially expressed mRNAs. We confirmed the induction of one the most prominent lncRNAs CYP1B1-AS1 by HSS by RT-qPCR. 14 lncRNAs were identified as potential enhancers. GO analysis showed that the highest enriched GOs targeted by up-regulated transcripts were cell migration and the highest enriched GOs targeted by the down-regulated transcripts were mitotic nuclear division. Comparison of differentially expressed transcripts between the groups identified 24 pathways that corresponded to down-regulated transcripts and 20 pathways that corresponded to up-regulated transcripts (p value cut-off 0.05).

CONCLUSIONS Our results show significantly altered expression profile of lncRNAs between the static control and shear-stressed HUVECs, indicating that lncRNAs may play important roles in regulating the functions of vascular endothelial cells. Further studies should be conducted to determine the roles of these lncRNAs in flow-dependent regulation of vascular function.

GW26-e4592
Implication of C1q/TNF-related protein-12(CTRP-12) in patients with coronary artery disease
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OBJECTIVES C1q/TNF-related protein-12 (CTRP-12), an adiponectin paralog, was recently identified as novel adipokines with metabolic regulatory properties. We analyzed the relationship between CTRP-12 and coronary artery disease (CAD).

METHODS Subjects (n = 188, 67 ± 10 years, 79% male) suspected of having CAD were enrolled in the study and were divided into two groups, CAD and non-CAD subjects, according to the results of their coronary angiographies. Serum CTRP-12 levels of the subjects were measured by an enzyme-linked immunosorbent assay.

RESULTS Subjects with CAD had significantly lower circulating CTRP-12 concentrations compared to the non-CAD subjects (median [inter-quartile range]: 46.6 [26.6] vs. 53.5 [29.6] ng/mL, respectively, p < 0.001). The serum CTRP-12 levels significantly correlated with body mass index, the homeostasis model of assessment of insulin resistance, adiponectin levels, and CAD severity. Multivariate logistic regression analysis revealed that a decreased serum CTRP-12 level (log transformed) was independently associated with CAD for all subjects.

CONCLUSIONS Serum CTRP-12 levels are significantly associated with CAD in humans, suggesting that low CTRP-12 levels may contribute to CAD.

GW26-e4726
Sestrin isoform expression in the normal and failing hearts
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OBJECTIVES Oxidative stress and AMP-dependent protein kinase (AMPK)/mammalian target of rapamycin (mTOR) signaling pathway were closely involved in the development of cardiac hypertrophy and heart failure (HF). Previous study indicated that the Sestrin family suppressed oxidative stress and regulated AMPK/mTOR signaling. However, the expression and functions of Sestrin in hypertrophic and failing hearts are still unclear.

METHODS We performed quantitative RT-PCR to detect the mRNA expression of Sestrin family in the normal, hypertrophic (2 week after surgery: n = 4), 4 week after surgery: n = 4) and failing (more than 8 week after surgery: n = 4) mice hearts induced by aortic banding