The binding affinity of CaM decreased in TAC cells (Kd: 360nM) compared with Sham(Kd: 180nM). The Ca2+ spark frequency(SpF: s-1100μm-1; by fluo3) was much higher in TAC than in Sham cardiomyocytes{TAC(n=10): 7.6±4.7; Sham(n=5): 5.7±1.9; $p<0.01$}. Addition of saturating concentration of CaM to saponin-permeabilised TAC cells normalize Ca2+ spark frequency{(n=5): 5.6±1.4}. **Conclusions:** In pressure-overloaded heart failure, CaM binding affinity to RyR2 is decreased. Our data suggest that the dissociation of CaM from RyR2 is thought to be the cause of the diastolic Ca2+ leak. CaM binding affinity to RyR2 might be the novel therapeutic target of the pressure-overloaded heart failure.

P-052

Addition of Home Blood Pressure Measurement and Biomarkers for Detection of Concentric Hypertrophy

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Background: Concentric hypertrophy is a phenotype of hypertensive cardiac organ damage and an independent predictor for cardiovascular disease. The aim of this study is to investigate whether the measurement of home blood pressure (BP) and biomarkers provides additional information for discrimination of concentric hypertrophy. **Methods:** In 1370 participants (mean age 65.5±11.2 years) with one or more cardiovascular risk factors from the J-HOP study, we evaluated a screening strategy for concentric hypertrophy determined by echocardiography with home BP level and 2 biomarkers, N-terminal pro-brain-type natriuretic peptide (NT-proBNP) and high-sensitivity cardiac troponin T (Hs-cTnT). **Result:** The patients with concentric hypertrophy were 22.3% (n=305). Area under the receiver operator characteristic curve improved from 0.675 (95% CI, 0.641−0.708) for the baseline model that included the conventional risk factors and clinic BP level to 0.694 (95% CI, 0.661−0.727) for the inclusion of home BP level and to 0.714 (95%CI, 0.682-0.747) for the inclusion of NT-proBNP and Hs-cTnT for the discrimination of concentric hypertrophy. **Conclusion:** The measurement of both home BP level and two biomarkers might provide additional information for detecting of concentric hypertrophy.

P-053

High Sensitivity Cardiac Troponin T Reflects Presence of Left Ventricular Hypertrophy and Diastolic Dysfunction and Predicts Incidence of Cardiac Events

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High Sensitivity Cardiac Troponin T (Hs-TNT) is a reliable biomarker in diagnosis of acute coronary syndrome (ACS). Recent several research reported that high Hs-TNT value predicts incidents of cardiovascular events even in general population. In this study, we researched association of Hs-TNT with clinical characteristics and clinical outcome in patients with cardiovascular disease. 228 consecutive non-ACS patients were examined in this study. We measured Hs-TNT, NT-proBNP, coronary risk factors, and creatinine, eGFR, and examined cardiac function by echocardiography. We divided patients into two groups: high Hs-TNT group (^{3}0.014ng/ml) and low Hs-TNT group ($<$0.014ng/ml). We researched clinical characteristics and investigated occurrence of cardiac events: death: admission for heart failure, coronary artery disease, and stroke. Compared with low Hs-TNT group, high Hs-TNT group had higher age, lower estimated GFR and higher NT-proBNP. In echocardiographic data, left ventricular ejection fraction was comparable between the two group, whereas left ventricular mass index and E/e' were higher in high Hs-TNT group. During the follow up period, incidence of cardiac events was significantly higher in high Hs-TNT group ($p=0.0091$) compared with low Hs-TNT group, whereas there was no difference in cardiac mortality. In conclusion, our results indicated that HS-TNT is an useful biomarker not only in evaluating the presence of left ventricular hypertrophy and diastolic dysfunction, but also in predicting the incidence of cardiac events.

P-054

Serum NT-proBNP Level Correlated with Left Atrial Dimension, not with Left Ventricular Dimension in Patients with Chronic Atrial Fibrillation

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Background: Several studies have reported that plasma brain natriuretic peptide (BNP) levels are increased in patients with chronic atrial fibrillation (AF). This study investigates serum N-terminal pro-brain natriuretic peptide (NT-proBNP) levels and left atrial (LA) diamensions in patients with AF and preserved left ventricular systolic function. **Methods:** Transthoracic echocardiography was performed in 24 patients with chronic AF. At the same time, serum NT-proBNP levels were measured by enzyme-linked immunosorbent assay (ELISA) and Holter recorder was performed in all patients. Subjects were excluded if they had a history of myocardial infarction, cardiomyopathy, or hyperthyroidism that preceded the onset of AF. **Results:** Echocardiography confirmed that the patients had normal LV systolic function with a mean LVEF of 60 ± 9%. Serum NT-proBNP levels were 2042 ± 1920 Pg/ml. LA dimension were 4.6 ± 0.9mm. Significant correlations were observed between the Serum NT-proBNP levels and LA dimensions ($r= 0.62$, $p<0.05$).
In contrast , neither left ventricular (LV) end-diastolic or end-systolic diameters were correlated to serum NT-proBNP levels. In addition, mean heart rate during Holter recording were (78 ± 21/min.) not correlated to serum NT-proBNP levels. **Conclusions:** Serum NT-proBNP level correlated with LA dimension, not with LV dimension in patients with chronic AF. These results showed that Serum NT-proBNP level could indicate LA remodeling in patients with chronic AF.

P-055

Cardiac ß -catenin Alterations Play an Important Role in Gap Junction Remodeling of Cardiomyocytes Exposed to Rapid Electrical Stimulation

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The intercalated disc (ID) contains two complexes, adhesion junction (AJ) and connexin (Cx) gap junction (GJ). GJs provide the pathway for intercellular current flow, and AJ mediates normal mechanical coupling and plays an important role in the stability of GJs. **Objective:** The purpose of this study was to study ID remodeling, especially ß-catenin and Cx43 alterations caused by rapid electrical stimulation (RES) in cardiomyocytes and to investigate the effects of Angiotensin II receptor blockade on ID remodeling. **Methods and Results:** Cultured rat cardiomyocytes were subjected to RES. We used real-time PCR, western blotting, and immunohistochemical methods. Conduction properties were examined by extracellular potential mapping system. Cx43 protein expression in cardiomyocytes was significantly increased after 60 min. Beta-catenin expression was significantly increased after 30 min. The expression level of ß-catenin in nucleus, which functions as TCF/LEF transcriptional activator of Cx43 and its degradation is regulated by glycogen synthase kinase (GSK3ß), was increased after 10 min. Conduction velocity was increased by RES for 60min. Olmesartan prevented most these effects of RES. We showed an increase of phosphorylated GSK3ß, which is phosphorylated by activated mitogen-activated protein kinases and inhibits ß-catenin degradations, was attenuated by olmesartan. **Conclusions:** The changes in ß-catenin precede GJ remodeling. Olmesartan might be a new upstream arrhythmia therapy by modulating ID remodeling through ß-catenin signaling pathway.

P-056

Validation of Lactic Acid Level as a Predictor of Mortality in Acute Heart Failure Patients Who Entered Intensive Care Unit

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Background: The significance of routine measurement of lactic acid level (LAC) is unclear in patients with acute decompensated heart failure (ADHF) patients who entered intensive care unit (ICU) from division of cardiology. **Methods:** This is a retrospective registry in a single center. From January, 2007 to March, 2012, 754 consecutive patients with ADHF patients who entered ICU were enrolled in this study. We included the patients who entered ICU for ADHF or acute myocardial infarction (AMI) with heart failure (Killip≥II). LAC was routinely measured at hospital presentation. **Results:** The mortality rates in the ICU and in-hospital were 5.8% (44 patients) and 11.7% (88 patients), respectively. The LAC had the great power to predict ICU and in-hospital mortality, as suggested by the c-statistics of 0.79 and 0.71, respectively. When the cutoff point, calculated by receiver-operator curves of 3.3mmol/L in LAC was used, the specificities for ICU and in-hospital death were 0.74 and 0.76, respectively. In the multiple logistic regression analysis, the odds ratio of high LAC for in-hospital mortality was 4.3 (2.0-9.9, $p=0.0003$). **Conclusions:** In the measurement of LAC in patients who entered ICU for ADHF, additive measurement of LAC to the conventional acid-base parameters is possibly useful to stratify the risk of mortality.