teristics of the pulse wave using Arteriograph Tensioclinic is a simple, reliable, non-invasive method to detect early changes in arterial stiffness.

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Chromogranin A Expression in Right Atrial Tissue in Patients with Severe Aortic Valve Stenosis

Edite Kulmane, Mara Pilmane, Romans Lacis

Pauls Stradins Clinical University Hospital, Latvia

Objective: Chromogranin A is a neuroendocrine system marker and might represent overall sympathetic activity. Elevated serum chromogranin A levels are associated with risk of clinical deterioration and death with acute coronary syndromes or chronic heart failure.

In this study we examined whether chromogranin A is produced in right atrial tissue in patients with severe aortic valve stenosis and angiographically confirmed absence of coronary heart disease.

Methods: In our hospital, right atrial tissue fragments were taken from 5 patients with severe aortic valve stenosis during elective aortic valve replacement surgery. The mean age was (mean ± SD) 73.8 ± 5.0 years (range 69–83 years) and there were 4 female patients. Tissues were processed for chromogranin A by means of biotin-streptavidin immunohistochemistry.

Results: Echocardiographically measured mean aortic valve area was 0.7 cm^2 and mean pressure gradient through aortic valve was 53 ± 7 mm HG. In all patients left ventricular ejection fraction was more than 55% and right atrial area less than 18 cm^2.

In one case we didn`t find any signs of chromogranin A in right atrial tissues. There were some blood vessels which few endothelial cells contained chromogranin in the other four specimens. In three cases factor positive cells particularly were pronounced in epicardium and endocardium, especially in regions with cube shaped epithelial cells.

Conclusions: Chromogranin A is characteristic factor in right atrial tissue (endothelium, endocardium, epicardium) of patients with severe aortic valve stenosis. Presence of factor proves idea about possibility of human heart cells to change a phenotype and produce neuroendocrine hormones for tissue remodelation.

P-18

Cardiotropic Action of a Stable Nitric Oxide Donor in Heart Failure

Valery Kapelko, Vladimir Lakomkin, Elena Lukoshkova, Alexander Abramov, Vladimir Ermishkin, Vladimir Gramovich, Oleg Vyborov, Nidas Undrovinas, Vladimir Shirinsky

Russian Cardiological Research and Productive Complex, Russia

Background: Nitric oxide is involved in a number of cellular metabolic processes. The activity of tissue NO synthases are depressed in heart failure, thus the supplementation of NO to the diseased myocardium may be beneficial.

Objective: We used dinitrosyl iron complexes (DNIC) with ligand glutathione (commercial name 'Oxacom') to study their cardiotropic action in heart failure. Oxacom is known to induce a prolonged hypotensive effect both in animals and healthy volunteers.

Materials And Methods: Rats were injected twice with isoproterenol in varied doses, 85–180 mg/kg. The blood pressure (BP), left ventricular (LV) pressure and ECG were recorded after 2–8 weeks in acute experiments preceded by echocardiographic study.

Results: Echocardiograhic study revealed obvious signs of heart failure, namely LV dilatation, lower ejection fraction, especially in rats that received higher cumulative isoproterenol doses (300–360 mg/kg). Cardiomyocytes isolated from failing hearts responded to electrostimulation by arrhythmic contractions and by significantly slowed and incomplete removal of Ca++ from the myoplasm. The decreased maximal rates of LV pressure development and fall as well as lowered indices of myocardial contractility and relaxability were detected. Intravenous bolus injection of oxacom caused an immediate BP decrease by 20–30 mm HG followed by a slow recovery. The LV systolic pressure did not change but the maximal rate of LV pressure development and contractility index increased by 20–26% while the isovolumic relaxation constant rose 1.5-fold associated with decreased LV diastolic pressure. These changes gradually normalized within 10–15 minutes.

Conclusion: Results suggest that prolonged NO donation exerts positive inotropic action in heart failure mainly by relaxability improvement.

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Prediabetes and Diabetes Are Not Related to Endothelial Dysfunction among Patients with Unstable Coronary Syndromes

Linda Bjork Kristinsdottir, Steinar Orri Hafþórsson, Erna Sif Oskarsdottir, Gudmundur Thorgeirsson, Vilmundur Gudnason, Sigurdur Sigurdsson, Isleifur Olafsson, Thorarinn Arni Bjarnason, Karl Andersen, Erna Sif Arnardottir

Landspitali – The National University Hospital of Iceland, Reykjavik, Iceland

Introduction: Approximately two thirds of patients with Acute Coronary Syndromes (ACS) have undiagnosed diabetes or prediabetes. The aim of this study was to determine whether disturbances in glucose metabolism are related to endothelial dysfunction in patient with ACS.

Methods: Patients with ACS but no known disturbance of glucose metabolism were consecutively included in a single center university hospital setting. A standard oral glucose tolerance test and measurements of fasting plasma glucose and HbA1c were performed 3–5 days after hospitalization, and repeated 8–12 weeks later. Carotid ultrasound was also performed to determine the extent of plaque formation in each patient. Assessment of endothelial dysfunction was done with EndoPAT and presented as the Reactive Hyperemia Index (RHI).

Results: Ninety-two patients were consecutively included (mean age 63.5 years, 79% male). Medians of RHI were 1.85 (IQR: 1.59–2.25), 1.78 (IQR: 1.60–2.27) and 1.85 (IQR: 1.40–3.43) in pa-