of age and apolipoprotein A1 for the development of LVDD, while multivariate analysis revealed apolipoprotein A1 as an independent predictor. E/E’ correlated significantly with age, HDL cholesterol, and apolipoprotein A1.

**Conclusion:** LVDD was detected in 10.3% T2D patients without arterial hypertension, ischemic heart disease or other cardiac disease. Higher apolipoprotein A1 was recognized as independent predictor of LVDD. Early detection of LVDD in T2D may be useful to halt the progression of myocardial lesions to heart failure.

**P 22: Omega-3 polyunsaturated fatty acids alleviate heart pump function in Type 1 diabetic cardiomyopathy**

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**Background and aims:** The term “diabetic cardiomyopathy”, which was first proposed by Rubler in 1972 describes myocardial dysfunction in the absence of coronary heart disease, hypertension or heart valve defects. However, from 70s of previous century is known about the omega-3 polyunsaturated fatty acids (PUFAs), which are potent cardioprotectors. Despite the success of diabetology, the mechanisms of omega-3 PUFAs influence on the heart function in diabetes mellitus remains unresolved. In this study we aimed to evaluate the effects of omega-3 PUFAs on cardiac performance in rats with experimental diabetes.

**Materials and Methods:** Experiments were fulfilled on male Wistar rats, weighing 140-270 g, who were divided into 3 groups: 1 - control, 2 - rats with streptozotocin-induced diabetes (50 mg/kg), 3 - rats with streptozotocin-induced diabetes, which received 45% of omega-3 PUFAs-containing drug Epadol during 4 weeks. Rats were anesthetized with Urethane, fixed and right carotid artery was prepared. Ultraminiature catheter 2F (‘Millar Instruments’, USA) was injected through the right carotid artery retrograde into the left ventricle. The fatty acids composition in heart homogenates was determined by gas chromatography.

**Results:** We demonstrate that omega-3 fatty acid treatment of diabetic rats modifies fatty acid composition of heart by increasing omega-3 PUFAs content. Additionally, supplemented omega-3 fatty acids significantly prevent body and heart weight loss and reduce blood glucose level, induced by streptozotocin. Our data indicates the impairment of the heart pump function in animals with diabetes. We established that cardiac output declined to 36.78% (P<0,05), ejection fraction to 29,12% (P<0,05), stroke volume to 70,13% (P<0,05). Omega-3 PUFAs under similar conditions restores pump function - increases ejection fraction to 35,37% (P<0,05), stroke volume at 40,76 (P<0,05), stroke work at 68,02% (P<0,05) and cardiac output at 15,32%. In 2 group of rats diastolic dysfunction was found - the time constants of active relaxation Tau (Weiss) reduced to 50,63% (P<0,05) and Tau (Glantz) to 59,77%, the minimum rate of myocardial relaxation dP/dtmin slightly increased. Effect omega-3 PUFAs on diastolic function is characterized by the increase of Tau (Weiss) to 64,39% (P<0,05) and Tau (Glantz) to 11,32% (P<0,05) and a slight decrease value of dP/dtmin. Omega-3 PUFAs had almost no impact on the maximum rate of pressure increase in dP/dt max under diabetes.

**Conclusion:** These novel data indicate that modification of fatty acid composition of cardiac membranes by omega-3 fatty acids prevents the pump, diastolic function alterations induced by streptozotocin. It is assumed that omega-3 PUFAs treatment will lead to a decreased risk of cardiovascular complications in diabetes. These results indicate the prospects of further studies to use omega-3 PUFAs in the treatment of diabetes.

**P 23: The possible contribution of thyroid hormones to insulin effect on beta adrenoceptor mediated cardiac responses in diabetic rats.**

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**Aims:** As is well known, diabetes-induced cardiovascular complications are leading factors of morbidity and mortality. Among them atherosclerosis, hypertension and heart failure are most important ones. The very first problem seen in diabetic heart is the decrease of beta adrenoceptor mediated cardiac responses. The decrease of catecholamine stimulated beta adrenoceptor responsiveness is mostly associated with diminished receptor sensitivity and density. Insulin treatment of diabetic rats has been demonstrated to correct these parameters. However, the improving effect of insulin is shown to be abolished in thyroidectomized diabetic rats. Although the effect of the relationship between these two hormones has already been studied, their exact role on beta adrenoceptor mediated cardiac responses is uncertain. Thus in the present study, we aimed to determine the possible relationship between insulin and thyroid hormones to normalization of diabetes induced cardiac abnormalities.

**Methods:** Male Sprague-Dawley rats were used for this study. Ten days after surgical thyroidectomy, diabetes was induced with 38mg/kg streptozotocin by a single intravenous tail-vein injection. After 6-weeks of diabetes induction, some groups (D-I, TxD-I) were treated with insulin (5-20U/kg/day, subcutan), some other groups (TxD-I-T2,5, TxD-I-T5) were administered insulin + thyroid hormone combination (2,5 or 5ug/kg/day T3 via osmotic minipump) for 2 weeks. Cardiac function was determined by the assessment of in vivo pressure-volume (PV) analysis and in vitro left ventricular papillary muscle experiments (isoproterenol, 0,001U/ml; forskoline, 3uM). mRNA expressions of β1-AR and SERCA were evaluated by QPCR.

**Results:** Heart rate and end systolic pressure were markedly reduced in diabetic (D) and thyroidectomized diabetic (TxD) groups; however, they were improved with insulin treatment in D group. On the other hand, in TxD group, only insulin and 5ug/kg T3 combination (TxD-I-T5) ameliorated these parameters. End diastolic pressure and ejection fraction were found to be unchanged between the groups. Rate of contraction and relaxation, time constant of left ventricle pressure decay were reduced in D and TxD groups. These parameters were corrected significantly in D-I and TxD-I-T5 groups. Cardiac index was reduced in D and TxD groups. This parameter was not increased markedly in any of the treated groups. End systolic volume index was raised in D and TxD groups, and reduced only in D-I group. End diastolic pressure index was also increased in D and TxD groups. It was significantly decreased in D-I and TxD-I-T5 group. Isoproterenol, a nonselective β-AR agonist, induced concentration-dependent positive inotropic effects on papillary muscles of control rats (C). Maximum response (Emax) was markedly diminished in D and TxD groups. These parameters were ameliorated in D-I group significantly. Furthermore, in TxD-I-T5 group, the contractile response was enhanced compared to TxD group. Forskolin induced positive inotropic effect in all groups. The response was decreased in D and TxD groups. Insulin treatment improved this decrease in D-I and TxD-I-T5 groups. β1-AR mRNA levels were decreased both in D and TxD groups. Insulin treatment increased β1-AR mRNA levels. In TxD-I group insulin did not increase β1-AR mRNA levels. On the other hand, in TxD-I-T5 group β1-AR mRNA levels were significantly increased compared to TxD, TxD-I and TxD-I-T2.5 groups. SERCA mRNA levels were also found to be reduced in D and TxD groups. Insulin treatment did not correct the decrease in mRNA levels of SERCA of D and TxD groups. In TxD-I-T5 group,