Correlations between left ventricular rotation and twist and aortic stiffness – Results from the three-dimensional speckle-tracking echocardiographic MAGYAR-Healthy Study Domsik P., Kalapos A.; Lengyd C.; Orosz A.; Kerster T., Nemes A.;

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Introduction: Left ventricular (LV) twist is the wringing motion of the heart, and has an important, but not fully evaluated role in the systolic and diastolic LV interclockwise rotation of the LV apex. It is well-known that stiffening of the aortic wall leads to changes in blood pressures compromising coronary perfusion and wall leads to changes in blood pressures compromising coronary perfusion and wall leads to changes in blood pressures compromising coronary perfusion and wall leads to changes in blood pressures compromising coronary perfusion and wall leads to enaity subjects. The present study was designed to find a relationship between 3DSTE-derived LV rotation and twist and echocardiographic aortic elastic properties in healthy subjects.

Methods: The present study comprised 26 healthy volunteers (mean age. 36.0 ± Methods: The present study comprised 26 healthy volunteers (mean age. 36.0 ± Methods: The present study comprised 26 healthy volunteers (mean age. 36.0 ± Methods: The present study comprised 27 healthy volunteers (mean age. 36.0 ± Methods: The present study comprised 28 healthy volunteers (mean age. 36.0 ± Methods: The present study comprised 29 healthy volunteers (mean age. 36.0 ± Methods: The present study comprised 29 healthy volunteers and 3DSTE echo-cardiographic aortic stiffness parameters were calculated from systolic and diastolic ascending aortic diameter and blood pressure data.

Results: Mean aortic stiffness index (ASI) (4.08 ± 0.79) were in normal range, as well as basil (-2.24 ± 1.43 degrees) and apical LV rotation s (35.6 ± 1.43 degrees) and alical LV rotations (8.56 ± 1.43 degrees) and alical LV rotation s (35.6 ± 1.43 degrees) and apical LV rotation correlated with aortic and twist and echocardiographic aortic elastic properties in healthy volunteers.

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Holter ECG monitoring findings in patients with metabolic syndrome and critical limb ischaemia.

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Introduction: Metabolic syndrome (MS), known as insuline resistance syndrome with not accidental collective occurence of global and central obesity, carbohydrate at tolerance impairment, dyslipaemia, hypertension and other factors (components), is tolerance impairment, dyslipaemia, hypertension and other factors (components), is associated with higher cardovascular mortality and prevalence of ischaemic heart associated with higher cardovascular mortality and prevalence of the control ding to ankle-brachtal pressure index (ARI) is increasing with the decrease of ARI, dut also in patients with medial calcinosis (ARI above 1.3) is significantly increased. Medial calcinosis is often detected in patients with MS. DM and renal failure.

Medial calcinosis is often detected in patients with MS. DM and renal failure. In of the study: In a group of patients with MS. DM and renal failure. In of the study: In a group of patients with MS. DM and renal failure. In other ECG monitoring the occurence of complex forms of ventricular arrhythmias and myocardial ischaemia. To evaluate the contribution of Holter ECG monitoring in therapeutic management of this patients. (19 male and 9 female) with Raitients and Melhods: We investigated 28 patients (19 male and 9 female) with CLL, stage III or IV. Fornaine, indicated for reascalistation procedure, amputation or conservative treatment. The mean age was 69 years (range 56-86), Mean waist or conservative treatment of CLL was applied in II. 2 were on dietary regime. 5 were treated by antidabetic drugs and II by insulin. It was a proformed with an Marquette-Hellige, 3 channel device, mean recording duration was performed with an Marquette-Hellige, 3 channel device, mean recording duration was performed with an Marquette-Hellige, 3 channel device, mean recording duration was performed with an Marquette-Hellige, 3 channel device, mean recording duration was performed with an Marquette-Hellige, 3 channel device, mean recording duration was performed in I) patients, percutancous transluminal

Drug eluting balloon – a solution for recurrent multiple pulmonary vein stenoses complicating catheter ablation of paroxysmal refractory atrial fibrillation? A case report

Background: Radiofrequency ablation (REA) of ectopic foci within pulmonary veins and surrounding arrial tissue has become an effective treatment for refractory: pasand surrounding arrial tissue has become an effective treatment for refractory: pasands are all fibrillation (AE). Pulmonary veins to the reversible vascular and parenchymal pulmonary changes and occurring in 1-3% of our most health of patients have been RF ablated in our centre, 6 patients (3 after redshaftion) have of our most challenging PX case, we would like to demonstrate our learning curve in interventional management of this chronic multifocal latrogenic disease. In interventional management of this chronic multifocal latrogenic disease, and effort dysproe; cough, hemopoe). Through analysis returned with PYS symptomic (effort dysproe; cough, hemopoe). Through analysis is on our hospital 7 times within 42 months complaining of effort dysproe out on which with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary hippertension due to PVS confirmed by MRA or precisely depicted with pulmonary wents shown by MRA. Consequently a successful transcatherial recannisation of LIPV and DRA or precisely depicted with pulmonary states and sending of the ESPV (allowed, Following next 1) months an access of LSPV and the LSPV. After 7 months restenting of the ESPV (allowed, Following next 1) months an access of LSPV with pulmonary persent explains and precise and sending of the ESPV

Identificatin of a novel KCN/2 mutation causing Andersen-Tawil syndrome in a Hungartan patient

a Hungartan patient

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Background: Andersen-Tawil syndrome is a multisystem disorder mainly caused by mutations in the gene KCN/2 which encodes the inward rectifier K: channel, Kir2.1. The disease is characterised by centricular arrhythmias, periodic paralysis and demonstrate fearmers.

and dysmorphic features.

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Case history. The 15-year-old female patient presented to us with ventricular Case history. The 15-year-old female patient presented to us with ventricular arhythmias mainly manifesting as frequent ventricular presented to the stopped because of or sotalol, amiodarone was somehow effective but it had to be stopped because of or sotalol, amiodarone was somehow effective but it had to be stopped because of the pain or distingtion of stating for several days, was also noted in addition, typical Leg pain or dumbness, lasting for several days, was also noted in addition, typical Leg pain or dumbness, lasting for several days, was also noted in addition, typical Leg pain or fuluding low-set ears, hypertelorism and micrognathia raised the suspicion of Andersan-Tawi syndrome.

Suspicion of Andersan-Tawi syndrome suspicion of the MCN/12 gene was amplified and direct sequenced in four fragments. Sequencing revealed a three base-pair of codon 301 and feets the last two base pairs of codon 301 as the last pair of codon 303 and leads to a complete frame shift, leaving codon 301 as the last pair of codon 301 as the last two base pairs of codon 302 and the first base pair of codon 303 and leads to a complete frame shift, leaving codon 301 as the last pair of codon 302 and the first base pair of codon 303 and leads to a complete frame shift, leaving codon 301 as the last pair of codon 302 and the first base pair of codon 303 and leads to a complete frame shift, leaving codon 301 as the last two base pairs of codon 302 and the first base pair of codon 303 as the last two base pairs of codon 304 as the last two base pairs of codon 304 as the last two base pairs of codon 304 as the last two base pairs of codon 304 as the last pair of codon 304 and the first base points. The mutation is not predicted to lead to a truncated in the mutant sequence and the mutanton is not predicted to lead to a truncated in the mutant sequence and the mutant is not predicted to lead to a truncated i