NORSKE ABSTRAKTER PRESENTERT I BARCELONA

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1805 Effects of a decision aid and additional decisional counselling on cardiac risk reduction behaviour and health outcomes: randomised controlled trial

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Purpose: To evaluate the effects of a Decision Aid for cardiac patients with and without an additional individual decisional counselling on health outcomes and health-related quality of life mediated by adherence to cardiac risk reduction behaviour.

Methods: Design: Prospective, 3-group RCT with 4 repeated measures over 6 months.

Participants and setting: 363 patients referred to Cardiac Outpatient Clinic in Norway, being examined for coronary artery disease by an angiogram. Interventions: The intervention group I (N=121) received, for taking home, the Decision Aid prior to their scheduled angiogram; the intervention group II (N=121) in addition to the Decision Aid received an individual decisional counselling from a trained nurse counsellor in their homes prior to their angiogram; and the control group (N=121) who received "the usual care". Main outcome measures: Body Mass Index, cholesterol, blood pressure, amount of tobacco, and health-related quality of life (primary outcomes), the adherence to cardiac risk reduction behaviour (intermediate outcome), and knowledge, benefits and barriers of cardiac risk reduction behaviour, and health beliefs (mediating variables).

Results: There were no significant differences between intervention group I and the control group on any variables. Intervention group II however, had a significant decrease in Body Mass Index (p=.016), and significantly improved health-related quality of life on several dimensions: role functioning physical (p=.021), general health (p=.049), vitality (p=.025), role function limitation (p=.022) and disease perception (p=.006) compared to the control group six months after the intervention. There were no significant differences in adherence to cardiac risk reduction behaviour between any of the groups. There was a significant decrease in scores of barriers to cardiac risk reduction

behavior in intervention group II compared to the control group (p=.020) at two months following the angiogram.

Conclusions: In this study the Decision Aid alone did not improve health behaviours and outcomes. Combining a Decision Aid with additional decisional counselling supported patients to individually tailor their cardiac risk reduction behaviour to their health beliefs and preferences, resulting in better health outcomes and health-related quality of life. We do not know however, if these effects would have occurred by the counselling alone, without combining it with the Decision Aid.

P186 Global longitudinal strain before cardiac resynchronization therapy predicts mortality in heart failure patients

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Purpose: Cardiac resynchronization therapy (CRT) improves mortality in heart failure (HF) patients with wide QRS. Strain by speckle tracking echocardiography (STE) quantifies myocardial function and predicts prognosis. We aimed to prospectively explore the ability of different echocardiographic measures before CRT implantation to predict mortality in HF patients with CRT.

Methods: Echocardiography (2D) was performed before and 6 months after CRT implantation in HF patients fulfilling CRT indications. Left ventricular (LV) function was assessed as ejection fraction (EF), global longitudinal (GLS) and global circumferential (GCS) strain from 16 LV segments by STE. Response to CRT was defined as decline in end systolic volume ≥15 % at 6 months. Our composite endpoint was defined as death, heart transplantation or left ventricular assist device (LVAD) during 2 years from CRT implantation.

Results: We included 113 HF patients (64 ± 9 years, 24 % women, NYHA class 2.8 ± 0.4, 43 % ischemic cardiomyopathy). Eleven (10 %) endpoints occurred (7 deaths, 2 transplantations, 2 LVADs). Worse GLS before CRT was a marker of endpoint (-5.6 ± 3.3 % vs. -8.8 ± 3.8 %, p=0.009), while EF (22 ± 7 % vs. 28 ± 9 %, p=0.05) and GCS (-9.7 ± 2.9 % vs.-11.2 ± 3.0 %, p=0.12) were not. GLS before CRT predicted endpoint independently of CRT response (HR 1.21 (1.01-1.45), p=0.04). GLS worse than -8.0 % optimally predicted unfavourable outcome (log rank p=0.004) (Fig. 1).

Conclusions: Myocardial function by GLS before CRT predicted death, heart transplantation or LVAD 2 years after CRT in HF patients, independently of CRT response. EF and circumferential function were not markers of fatal outcome. Longitudinal myocardial function before CRT may have the greatest impact on outcome in CRT patients.

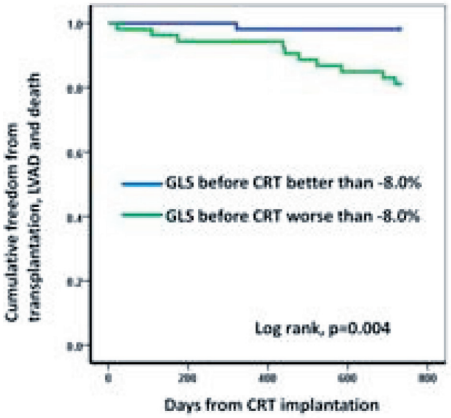

Figure 1. GLS and survival in CRT patients

P2966 Ventricular arrhythmias in subjects with ARVC are associated with increased cardiac volumes but not with ejection fraction by cardiac magnetic resonance imaging

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Purpose: Ventricular arrhythmias are frequent in patients with arrhythmogenic right ventricular cardiomyopathy (ARVC), but risk stratification is still challenging. We investigated the relation between ventricular arrhythmias and cardiac volumes and function by cardiac magnetic resonance imaging (MRI).

Methods: In total, 56 ARVC subjects (mean age 43 ± 16 years, 57 % male) were studied by cardiac MRI. We assessed end-diastolic volume indexed by body surface area (EDVI) and ejection fraction (EF) in the right (RV) and in the left ventricle (LV). Ventricular arrhythmias were defined as documented ventricular tachycardia or fibrillation or aborted cardiac arrest.

Results: Of the 56 included, 33 (59 %) were index patients fulfilling 2010 Task Force Criteria for ARVC diagnosis and 23 (41 %) were mutation positive family members. Ventricular arrhythmias had occurred in 29 (52 %). Indexed LV and RV volumes were increased in patients with ventricular arrhythmias compared to those without (LVEDVI: 80 ± 16 ml/m2 vs. 67 ± 18ml/m2, p=0.01 and RVEDVI 104 ± 36 ml/m2 vs. 69 ± 25 ml/m2, p<0.01) (Fig. 1). LV and RV function by EF did not differ in ARVC subjects with and without arrhythmic events (LVEF: 51 ± 8 % vs. 50 ± 9 %, p=0.66 and RVEF: 39 ± 14 % vs. 40 ± 11 %, p=0.77). Body surface area was similar in both groups (1.92 ± 0.18 m2 vs. 1.89 ± 0.24 m2, p=0.64).

Conclusions: ARVC subjects with ventricular arrhythmias had increased indexed RV and LV end-diastolic volumes compared those without, while myocardial function by EF did not differ in RV nor in LV. Risk stratification of ventricular arrhythmias in ARVC subjects by MRI should not rely on EF, but focus on increased volumes in RV and LV.

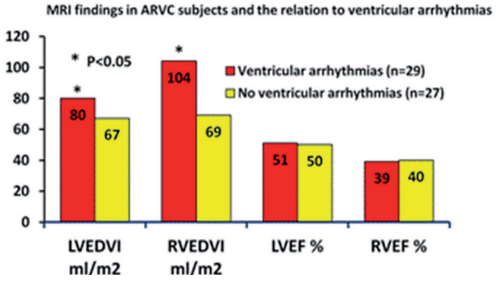

Figure 1

P2055 Aerobic interval training reduces the burden of atrial fibrillation

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Background: Exercise training is an effective treatment of important atrial fibrillation (AF) comorbidities. However, high levels of endurance exercise have been associated with increased prevalence of symptomatic AF. This study examines the effect of aerobic interval training in patients with AF.

Methods: 51 patients with paroxysmal or persistent AF were randomized to aerobic interval training (n=26) consisting of 4 times 4 min of treadmill exercise at 85-95 % of peak heart rate, 3d/week/12 weeks or a control group (n=25). An implantable loop recorder measured time in AF continuously from 4 weeks before to 4 weeks after the intervention period. Cardiac function, lipid status and quality of life were evaluated before and after the 12-week intervention-period.

Results: There was an increase in burden of AF in the control group, and a reduction in the exercise group (P=0.001 between groups). 16 patients in the control group and 3 patients in the exercise group experienced an increase in the burden of AF, whereas 5 patients in the control group and 10 patients in the exercise group experienced a decrease in AF burden. The rest had no change from pre to post. There was a trend towards fewer cardioversions (1 vs. 6, P=0.14) and hospital admissions (1 vs. 9, p=0.07) in the exercise group. There were no significant differences in use or changes of antiarrhythmic drugs between the groups. In the exercise group there was a significant increase in VO2Max (9.5 % vs. -1.1 %, p=0.002), left atrial ejection fraction during atrial systole (+1,3 percentage points (pp) vs. -2,5 pp, p=0.047) and left ventricular ejection fraction (+3.2 pp vs. -1.6 pp, p=0.03) by MRI, SF-36 measures of general health and vitality, and a significant decrease in cholesterol (-0.38 mmol/L vs. +0.13mmol/L, p=0.009) and triglyceride levels (-0.23 mmol/L vs. +0.18 mmol/L, p=0.008) compared to controls.

Conclusion: Aerobic interval training for 12 weeks clearly reduces the burden of AF in symptomatic patients. It is followed by a significant improvement in VO2Max, left atrial and ventricular function, cholesterol levels, and quality of life. Further studies are needed to evaluate the underlying mechanisms and the effect of exercise training for a longer period of time.

2239 Complement factor 5 blockade reduces porcine myocardial infarction size and improves cardiac function

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Purpose: Inhibition of the complement system has proven beneficial in various animal species but not in clinical studies of myocardial infarction (MI). Coversin (Ornithodoros

moubata complement inhibitor, OmCI) selectively blocks complement factor 5 (C5) activation and has been shown to dampen the inflammatory response in experimental sepsis. We hypothesized that correctly timed inhibition of C5 would reduce infarction size and improve ventricular function in a porcine model of MI.

Methods: Left anterior descending coronary artery was occluded for 40 min in 16 pigs (20 ± 1 kg) and reperfused for 240 min. Coversin or placebo was given intravenously 20 min after occlusion and throughout reperfusion. The extent of infarction was measured ex vivo as percentage of infarcted tissue in the area at risk using Evans blue and tetrazolium chloride staining, and magnetic resonance imaging (MRI). Tissue Doppler echocardiography was performed from apical 4-chamber view at baseline and end of reperfusion. Systolic displacement was measured as mean values from septal and lateral mitral annulus movements. Interleukin-1β (IL-1β) was analysed in myocardial microdialysis fluid. Values are presented as mean ± SD, and groups were compared with Student's t-test.

Results: Coversin significantly reduced MI by 39 % ($p<0.05$, Fig. 1A) and increased systolic displacement by 31 % ($p<0.01$, Fig. 1B). MRI correlated to histological findings (19 % reduced infarction in left ventricle, $R=0.92$, $p<0.01$). IL1-β was increased in the infarction area and was not different from baseline in coversin treated animals ($p<0.05$).

Conclusions: Selective C5 inhibition reduced size of infarction and improved ventricular function in a MI pig model. Coversin may thus have potential as a therapeutic agent in MI.

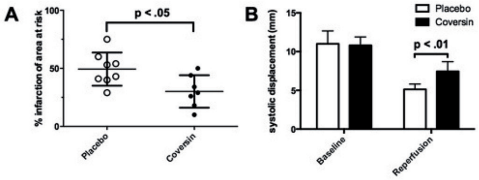

Figure 1. Effect of C5 blockade in MI.

P3205 Birth prevalence and time trends of congenital heart defects in Norway 1994-2009 - a CVDNOR project

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Purpose: In this study we present population-based birth prevalences and time trends of congenital heart defects (CHD) among live births, stillbirths, and terminated pregnancies during 16 years of follow-up.

Methods: CHD diagnoses among all births in Norway, 1994-2009, were ascertained from national health registries and databases; the Medical Birth Registry of Norway, the hospitals' Patient Administrative System, the National Hospital's clinical database for children with heart disease, and the Cause of Death Registry. Using a hierarchical classification system, individuals were assigned specific cardiac phenotypes. Time trends were analyzed using Joinpoint Regression Program.

Results: Among 954,413 births 13,081 were identified with CHD (137.1 per 10,000 births). Live birth prevalence was 133.2 per 10,000. Excluding preterm PDA, CHD prevalence per 10,000 births was 123.4; per year, the prevalence increased with 3.5 % (95 % confidence interval 2.5, 4.4) in 1994-2005, and declined with 9.8 % (-16.7, -2.4) from 2005 onwards. Severe CHD prevalence was 30.7 per 10,000; annual increase was 2.3 % (1.1, 3.5) in 1994-2004, and annual decrease 3.4 % (-6.6, -0.0) in 2004-2009. The prevalence of severe CHD in live births was 26.7 per 10,000; annual increase in 1994-2003 was 1.8 % (0.2, 3.4), and annual decrease in 2003-2009 was 4.6 % (-7.3, -1.8). Stillbirths and terminated pregnancies constituted an increasing proportion of births with severe heart defects; 3 % in 1994-2007, and 16.9 % in 2006-2009.

Conclusions: Although there has been a shift from diagnosing CHD in live births to prenatal diagnosis with the possibility for termination of pregnancy, this could not explain the decreasing live birth prevalence of total CHD and severe CHD from 2005 to 2009 in the present nationwide study.

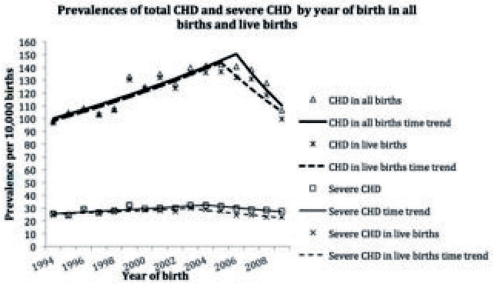

Figure 1

P3640 Impaired exercise capacity after anthracycline treatment in asymptomatic survivors of childhood acute lymphoblastic leukemia

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Purpose: Little is known about the effect of anthracycline therapy on future exercise capacity, measured by maximal oxygen uptake (VO2max), after treatment of childhood acute lymphoblastic leukemia (ALL). In a cross-sectional study of very long-term survivors of childhood ALL, mean 23 ± 8 years post diagnosis, we wanted to compare VO2max in survivors exposed and unexposed to anthracycline treatment.

Methods: Treatment data were collected from medical records. Echocardiography was performed in all survivors, before they performed maximal exercise on a bicycle, with continuous measurement of respiratory gas exchange and calculation of VO2max.

Results: Exposed survivors had received a cumulative isotoxic doxorubicin dose of median 120 mg/m2 (range 40-485). Unexposed survivors were older, thus having lower values of expected VO2max. Mean body weight and gender distribution did not differ between the treatment groups. In all, 55 % of exposed survivors had reduced VO2max, compared to only 17 % of unexposed survivors (p<0.001). VO2max did not have any statistical association with other treatment variables. However, we observed significant correlations between exercise capacity and measures of left ventricular function, such as ejection fraction (p=0.001), global longitudinal strain (0.015) and diastolic tissue Doppler velocities (p<0.001).

Conclusions: In survivors of childhood ALL, previous anthracycline treatment is associated with impaired exercise capacity, possibly related to late cardiotoxicity.

	All survivors	Exposed	Unexposed	p-value
	(n=132)	(n=103)	(n=29)	
Age at diagnosis (years)	6.3 ± 4.0	6.8 ± 4.2	4.7 ± 2.7	0.013
Age at exam (years)	29.4 ± 7.1	27.5 ± 6.4	36.1 ± 5.0	<0.001
Female (number)	65 (49 %)	48 (76 %)	17 (58 %)	0.253
Body weight (kg)	76.5 ± 17.9	76.0 ± 18.2	78.0 ± 17.2	0.599
VO2max (ml/kg/min.)	34.7 ± 8.4	34.5 ± 8.5	35.2 ± 8.3	0.711
VO2max (% of predicted)	85 ± 19	83 ± 19	94 ± 18	0.004
Reduced VO2max (number)	62 (47 %)	57 (55 %)	5 (17 %)	<0.001

P-values are for the comparison between survivors exposed and unexposed to anthracycline therapy. VO2max: maximal oxygen uptake.

P5510 Expression of genes in aspirated coronary thrombi in patients with acute myocardial infarction

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Purpose: Althought data about the clinical benefit of thrombus aspiration have exhibited controversial results, the knowledge of thrombus composition, particularly with respect to genetics, is of increasing interest. Reports until now have mainly focused on the structural and cellular components of aspirated coronary thrombi. We therefore aimed to investigate the genetic expression of selected mediators and proteases actively involved in plaque rupture, platelet and neutrophil cell activation, coagulation, fibrinolysis and inflammation in aspirated coronary thrombi from patients with acute myocardial infarction.

Methods: Coronary thrombi from 67 patients with acute myocardial infarction were investigated. RNA from aspirated coronary thrombi was isolated and gene expression arrays of selected markers were performed by a RT-PCR based method with relative quantification.

Results: Twenty of 22 selected markers were expressed in >50 % of the samples. CRP and IL12 were not expressed. The relative quantification of P-selectin correlated negatively to the ischemic time (p=0.01), while genes related to fibrinolysis (t-PA, u-PA, PAI-1), inflammation (PTX3, CXCL9, MCP-1, IL18, TNF-alfa) and to plaque instability (MMP-2 and TIMP-1) correlated positively to the ischemic time (all <0.05). When dichotomizing ischemic time into ≤ median (4.0h) and > median, the relative reduction of P-selectin was 0.7-fold, while the relative increase in t-PA was 2.2-fold, u-PA 5.8-fold, PAI-1 8.7-fold, PTX3 1.7-fold, CXCL9 3-fold, MCP-1 2.6-fold, IL18 2.3-fold, TNF-alfa 2-fold, MMP-9 2.8-fold and TIMP-1 3.2-fold. The presence of type 2 diabetes increased PAI-1 expression 3.2-fold, while the presence of hypertension reduced IL-8 and TIMP-1 to about half-fold. Smoking and overweight did not affect any markers.

Conclusions: Several pro-inflammatory markers and mediators were genetically expressed in aspirated coronary thrombi from patients with acute myocardial infarction. The genetic expression profile changed according to the ischemic time with a decrease in expression

of genes related to platelets and an increase in expression of genes related to fibrinolysis, inflammation and plaque instability, respectively. Expression of PAI-1 was significantly higher in patients with type 2 diabetes, possibly confirming the particular role of impaired fibrinolysis in type 2 diabetes. The presence of hypertension seemed to be associated with plaque instability.

P3264 Changes in aortic root diameter throughout normal pregnancy

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Purpose: During normal pregnancies in healthy women (HW) physiological changes facilitate the adaptations of the cardiovascular system. The aortic root diameter (AOD) may increase during pregnancy in patients with aortic pathology. The aim of this study was to investigate the AOD throughout pregnancy in HW.

Method: HW were examined using echocardiography with Doppler (GE Vingmed Vivid 7) at gestational weeks 14-16, 22-24, 36, and 6 months postpartum (PP). AOD was measured from parasternal longs axis 2D views at the sinus Valsalva in systole using "inner-egde-to-inner-egde" technique.

Results: (presented as mean ± SD): Study included 50 HW, aged 32 ± 5 years, 58 % nullipara. AOD showed statistical significant increase through pregnancy: from 2.5 ± 0.3cm in the 1st trimester, 2.6 ± 0.3cm by 2nd trimester, to 2.8 ± 0.2cm in the 3rd trimester ($p<0.05$). There was a statistical non-significant increase in AOD from the 3rd trimester to 2.80 ± 0.3 cm by 6 months PP (figure). Parity was a significant covariant factor ($p<0.05$) with multipara women exhibiting a larger AOD in the 3rd trimester, but not by the 1st trimester. End-diastolic ventricular diameter and cardiac output changed significantly throughout pregnancy with normalization to 1st trimester values at control 6 months PP. Systolic blood pressure (mmHg), however, changed during pregnancy: 108 ± 1, 104 ± 1, 109 ± 1 and further on to PP 112 ± 1 ($p<0.01$).

Conclusion: Our data demonstrate an increase in the AOD by approximately 3mm in HW during pregnancy. The AOD does not normalize the first six months PP. This is an important finding with implications for interpretation of the AOD in pregnant women.

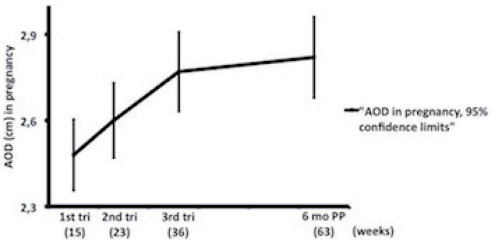

Figure: AOD throughout normal pregnancy

P3215 Living with recurrent atrial fibrillation: searching for control and appropriate support

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Background: Living with recurrent atrial fibrillation is often characterized by the unpredictability of incidence, symptom intensity and duration which may have major impact on patients' perception of control in daily life. In addition, patients may experience periods of high disease activity and frequent hospitalizations. However, little is known about how recurrent atrial fibrillation may influence patients' experience of control in daily life. Furthermore, studies examining patients' perception of support from healthcare professionals still seem to be sparse.

Purpose: To describe 1) how recurrent atrial fibrillation may influence patients' experience of control in daily life and 2) how patients perceive support from healthcare professionals.

Methods: A descriptive and explorative qualitative design, including semi-structured interviews with nine patients (eight men and one woman) living with recurrent atrial fibrillation, was used. The participants' mean age was 63 years and the duration of recurrent atrial fibrillation varied from 2-27 years. The interviews were audiotaped, transcribed verbatim and the data were analysed using Systematic Text Condensation.

Results: Two main themes were identified. The first theme "losing control in everyday life" was based on two subthemes: 1) living with unpredictability and 2) living with uncertainty. The second theme "seeking adequate support" was illustrated by the following subthemes: 1) need for consistent information and 2) need for assurance and emotional feedback.

Conclusion: Patients with recurrent atrial fibrillation experience loss of control in everyday life. The diminished control seems to be contributed

by the unpredictability and uncertainty associated with the disease. Support from healthcare professionals is perceived insufficient and inconsistent. The patients therefore need to be provided with more adapted information and an individualized schedule for treatment. Also, various psychosocial and emotional aspects associated with the disease must be addressed.

P3211Symptoms of anxiety and depression: the impact of diagnostic angiography

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Purpose: Coronary angiography is the gold standard diagnostic test for coronary heart disease (CHD) and the diagnostic results can have an immediate effect on symptoms experienced. Little is known, about the effect of having the diagnostic results on symptoms of anxiety and depression, and in this study the effects of diagnostic angiography on these symptoms was explored.

Method: The study included 780 consecutive patients, with established or suspected CHD undergoing examination both with invasive and CT Angiography. The trial was approved by the regional board of ethics. Data were collected at baseline and after six months. Symptoms of anxiety and depression were measured using the Hospital Anxiety and Depression Scale (HADS). The HADS has a range from 0-21 and optimal cut-off scores above 8 on both scales identify possible cases with anxiety and depression.

Results: The sample comprised 508 men and 272 women with mean age of 63.6 years (SD 10.8). A total of 468 (59.7 %) did not have previous CHD. The results indicated that 26 % had normal vessels; 20 % wall changes (20-49 %); and 53 % obstructive stenosis (≥50 %). At baseline a total of 23.6 % and 19.9 % in the sample had a score above 8 in the HADS anxiety and depression scales, respectively. After six months these proportions were reduced to 16.5 % and 14.6 % correspondingly. Among those with and without previous CHD there were no significant differences in baseline mean HADS anxiety or depression scores. Women had a significant higher baseline anxiety score than men (5.5 vs 4.8 p=0.02), otherwise there were no gender differences in scores or changes.

From baseline to six months there were significant improvements in the entire sample ($p<0.001$), in both the anxiety (mean change 0.93) and depression (mean change 0.93) scores. Analysing those without previous CHD, there were significant improvements over time in both anxiety and depression in those diagnosed with normal vessels; mean change 0.77 ($p=0.001$) and 0.63 ($p=0.016$) respectively. Likewise, in those with stenosis there were significant improvements; mean change 1.38 ($p<0.001$) and 0.81 ($p=0.001$), correspondingly. In those with wall changes, there were non-significant smaller improvements (mean change 0.49 and 0.53).

Conclusion: Undergoing diagnostic angiography significantly reduces symptoms of anxiety and depression in both those with stenosis and those with normal coronary arteries. The group being diagnosed with non-obstructive stenosis i.e. 20 -49 %, not eligible for revascularization, have a small however not significant improvements in symptoms of anxiety and depression.

P6560 Electro-mechanical alterations in patients with long QT syndrome

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Purpose: Long QT syndrome (LQTS) is an arrhythmogenic cardiac ion channelopathy which has been considered a purely electrical disease. However, recent reports have indicated mechanical abnormalities in LQTS patients. We aimed to explore systolic and diastolic function in LQTS patients.

Methods: We included 192 genotyped LQTS patients with no concomitant cardiac disease. Age and sex matched healthy individuals served as controls (n=59). By echocardiography, we assessed left ventricular (LV) ejection fraction (EF) and speckle tracking global longitudinal strain (GLS) (16 LV segments). E-wave, A-wave, E deceleration time and e' (mean of septal and lateral e') were recorded by Doppler. Left atrial volume index (LAVI) was calculated. Heart rate corrected QT interval (QTc) was assessed by 12-lead ECG.

Results: In the 192 LQTS subjects, systolic function by GLS and diastolic function by e' and E deceleration time were reduced compared to healthy (all $p<0.05$) (Table). LAVI was enlarged in LQTS ($p<0.01$). QTc and LAVI correlated in LQTS ($R=0.17$, $p<0.05$), but not in healthy ($R=0.33$, $p=0.13$).

Conclusion: LQTS patients had a subtle reduction in both systolic and diastolic function compared to healthy. LAVI was enlarged in LQTS, indicating longstanding diastolic alterations. Furthermore, prolonged QTc was related to increased LAVI

in LQTS, indicating an electro-mechanical association. These alterations may represent mechanical consequences of ion channel disease.

Table 1. Findings in LQTS and healthy individuals

	Healthy control (N=59)	LQTS mutation positive (N=192)	p-value
Age	37 ± 10	36 ± 16	0.43
Female (n (%))	31 (53)	117 (61)	0.25
Heart rate	66 ± 10	64 ± 12	0.09
QTc (ms)	391 ± 26	467 ± 40	<0.01
EF (%)	61 ± 5	61 ± 5	0.74
GLS (%)	−22.5 ± 1.9	−21.6 ± 2.0	<0.01
e' (cm/s)	12.2 ± 2.2	10.4 ± 2.7	<0.01
E deceleration time (ms)	158 ± 28	187 ± 41	<0.01
E/A	1.9 ± 0.6	1.7 ± 0.7	0.07
E/e'	6.4 ± 1.7	7.2 ± 2.2	0.06
LAVI (ml/m2)	26 ± 5	30 ± 8	<0.01

P1745 Cardiac function in very long-term survivors of childhood lymphoma

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Purpose: Childhood lymphoma survivors (CLSs) have markedly increased risk for cardiac disease, but data on left ventricular (LV) diastolic function in CLSs are conflicting. In this cross-sectional study of 125 adult CLSs, cardiac function was assessed by echocardiography and compared to healthy controls, matched for age, gender, body weight and systolic blood pressure.

Methods: LV systolic dysfunction was defined as ejection fraction <50 % (Simpson's method), or by fractional shortening <27 % for women and <25 % for men (M-mode measurement). LV diastolic dysfunction was defined as pulsed tissue Doppler peak early diastolic velocity (e') <8 cm/s in the septal, or <10 cm/s in the lateral mitral annulus. Increased LV filling pressure was defined as average E/e' ratio >13. Left heart valve dysfunction was defined as any stenosis, or any regurgitation graded as more than minimal, on the aortic or mitral valve.

Results: Previous diagnosis was Hodgkin's lymphoma in 81, and non-Hodgkin lymphoma in 44. Treatment included mediastinal radiotherapy (RT) in 66 (53 %), and anthracyclines in 92 (74 %). Mean time since diagnosis was 20.4 ± 8.6 years, and median age at exam was 33.0 years (range 19.0-54.5). CLSs did not have more LV systolic dysfunction than controls, but LV diastolic dysfunction was 7 times more frequent (p<0.001). After mediastinal RT, diastolic dysfunction was even more frequent, and 20 % had signs of elevated LV filling pressure in this groups, vs. 0 among the other CLSs (p<0.001). More than half of the CLSs treated with mediastinal RT had valvular dysfunction.

Conclusions: LV diastolic dysfunction occurs frequently in CLSs, particularly after mediastinal RT, whereas systolic dysfunction occurs infrequently, and equally frequent in CLSs and controls in this study.

See table.

Table1. Prevalence of cardiac abnormalities

	Survivors	Controls	p-value	Mediastinal RT	No mediastinal RT	p-value
	(n=125)	(n=125)		(n=66)	(n=59)	
LV systolic dysfunction	12 (10 %)	16 (13 %)	0.422	8 (12 %)	4 (7 %)	0.312
LV diastolic dysfunction	36 (29 %)	5 (4 %)	<0.001	24 (36 %)	12 (20 %)	0.048
Increased LV filling pressure	13 (10 %)	0	<0.001	13 (20 %)	0	<0.001
Valvular dysfunction	39 (31 %)	NA		36 (55 %)	3 (5 %)	<0.001
Abnormal LV dimension	15 (12 %)	10 (8 %)	0.312	13 (20 %)	2 (3 %)	0.005

LV, left ventricular; RT, radiotherapy; NA, not available.

P519 Impaired RV systolic function in lymphoma survivors after radiotherapy

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Purpose: Lymphoma survivors (LS) have increased cardiovascular disease burden, because of cardiotoxic treatment, in particular anthracyclines (AC) and radiotherapy (RT) involving the heart. Our aim in the present study was to assess RV systolic function after RT in this patient group.

Methods: All LS treated with high dose chemotherapy with autologous stem cell transplantation (HDT) in Norway in the period 1987–2008, aged ≥18yr at time of HDT were invited to a medical examination including echocardiography. This report includes 186 LS (66 % men) examined at our universitary hospital. All had received AC and 78 had additional radiotherapy involving the heart. Patients were categorized into three groups according to treatment: AC (n=108, age 58yr ± 12yr, 11yr ± 5yr since primary treatment), AC-MRT (mediastinal radiotherapy, median dose 31Gy, range 19-41Gy, n=39, age 47yr ± 12yr, 14yr ± 6yr since primary treatment) and AC-TBI (total

body irradiation, 13 Gy, n=39, age 56yr ± 10yr, 22yr ± 3yr since primary treatment). Conventional echocardiograms were obtained by Vivid 7 or E9 (GE Vingmed, Norway). RV global longitudinal strain (GLS, six segments) and RV-free wall GLS (three segments) by two-dimensional speckle tracking, and fractional area change (FAC) of the RV were all measured from the apical four chamber view. Analysis for differences between groups according to treatment were done by One-Way ANOVA.

Results: RV systolic function was significantly impaired in LS receiving AC + RT involving the heart compared with LS treated with AC, as judged by most parameters of RV systolic function (table 1). The treatmentgroups had comparable body mass index, p=0.96.

Conclusion: RV systolic function is impaired when RT involving the heart is added to AC in LS.

Table 1

Variable	MRT-AC (n=39)	TBI-AC (n=39)	AC (n=108)	p
FAC (%)	41 ± 5	43 ± 5	45 ± 5	<0.0001
RV GLS (%)	−21.3 ± 3.5	−21.2 ± 3.3	−23.1 ± 3.0	0.001
RV Free Wall GLS (%)	−25.5 ± 4.0	−26.3 ± 4.3	−27.6 ± 3.8	0.02
TAPSE (mm)	22 ± 5	22 ± 4	24 ± 4	0.05
s'RV (cm/s)	11.5 ± 2.8	12.3 ± 2.6	12.9 ± 2.8	0.03
TRP (mmHg)	21 ± 7	22 ± 12	22 ± 7	N

P1383 Treatment preferences assessed with standard gamble in patients reffered for aortic valve replacement

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Introduction: Symptomatic aortic stenosis (AS) is a condition with increased mortality, morbidity and rapid worsening of patient's health. Aortic valve replacement (AVR) is the only curable treatment. AVR should only be performed in motivated patients. Patients referred for surgery are increasingly old and frail with little known about their preferences for surgery and evaluation of health. Standard gamble (SG) assesses preferences by identifying the highest risk for treatment mortality patients are willing to take. The perioperative risk and uncertainty relating to the procedure lends the SG relevance for evaluating preferences in this condition.

Methods: Cross-sectional study of 505 patients with severe AS referred for AVR. The SG was interviewer-administered, other assessments included clinical variables, echocardiography with assessment of aortic valve severity and myocardial function, EQ-5D, Short Form-36 (SF-36) and self-reported questionnaire of AS-related symptoms. Patient's preferences were postulated to be more highly associated with assessments of health and AS related symptoms than with objective measures of disease severity. Patients with poorer self-assessed health and symptoms were hypothesized to have lower SG scores and hence willing to take more operative risk.

Results: 439 completed the SG interview with mean (SD) age 75 (11) years, 44 % women. Patients had a median SG score of 0.75 (interquartile range 0.50-0.95). SG scores were independent of sociodemographic variables, clinical variables, and hemodynamic valve parameters. In multiple logistic regression SF-36 mental health [OR 1.02 (1.00 – 1.03), p=0.02], EQ-VAS [OR: 1.03 (1.02 – 1.05), p<0.001], SF-36 health transition in last year [OR: 0.99 (0.98 – 1.00), p=0.05] and number of AS-related symptoms during last week [OR: 1.40 (1.14 – 1.72), p=0.001] were associated with SG score <0.75. The model accounted for 17 - 23 % of the total variance (pseudo R square).

Conclusions: Preferences for operation in patients with severe AS are not associated with clinical or echocardiographic assessment of the AS but with patient's assessment of their health, change in health and AS-related symptoms. This highlights the importance of assessing patient preferences as a means of informing clinical decision making. However, only a small amount of variation in SG-score is accounted for, emphasizing the importance of actually measuring preferences in pre-operative assessment of patients with severe AS.

P2988 Body mass index and changes in weight are associated with risk of atrial fibrillation and cardiovascular mortality: a longitudinal cohort study of 7,169 patients with newly diagnosed type 2 diabetes

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Purpose: Obesity is associated with an increased risk of type 2 diabetes (T2D) and cardiovascular (CV) disease. Our aim was to explore the impact of body mass index (BMI) at baseline, and weight

change, on the risk of atrial fibrillation (AF) and CV mortality in patients with T2D.

Methods: A total of 7,169 primary care patients with newly diagnosed T2D and without previous history of CV disease were grouped separately according to baseline BMI and their change in BMI within 18 months after diagnosis. The relative weight change was grouped as follows (1 BMI unit ~ 3.6 kg): "weight gain" (>1 BMI unit), "stable weight" (± 1 BMI unit) and "weight loss" (<1 BMI unit). Follow-up time was 9 years, and risks of AF and CV mortality were estimated using adjusted Cox regression models.

Results: Mean age was 60 years at time of diabetes diagnosis and patients were on average slightly obese (mean BMI 30.2 kg/m2). During follow-up, overweight and obese patients had significantly higher risk of AF, compared with those with normal BMI. For CV mortality there was an apparent "obesity paradox" situation with similar risk independent of BMI category. From time of T2D diagnosis, few patients (15 %) increased weight while the majority lost or maintained weight. Risks of both AF and CV mortality were significantly higher in patients increasing weight compared to weight stable patients.

Conclusions: Obesity/overweight or weight gain in patients with newly diagnosed T2D may be more hazardous than previously recognized, and efforts should be made to control weight in diabetes patients.

See table.

P2971 Emergency coronary angiography and revascularization in patients with out-of hospital cardiac arrest, relation to post ROSC ECG, a prospective observational study

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Purpose: The aim of this prospective cohort study was to evaluate the use of post-ROSC ECG to select patients with out-of-hospital-cardiac-arrest (OHCA) in need of immediate coronary angiography (ICA) and acute revascularization.

Methods: All patients with stable ROSC after OHCA were directly transferred to ICA, irrespective of ECG findings. Patients with a clear non-cardiac aetiology were excluded. Patients were classified into three groups according to post-ROSC ECG by two cardiologists blinded to the ICA results: 1) ST-elevation or new LBBB, 2) other signs suspected of coronary ischemia, or 3) no signs of coronary ischemia. In order to identify patients with an indication for ICA, based on either an acute occluded infarct related coronary artery (IRA) or a flow-limiting coronary stenosis, the coronary angiography were re-evaluated in all patients by an interventional cardiologist blinded to the post-ROSC ECG.

Results: A total of 124 patients were admitted to our hospital with ROSC after OHCA. The median age was 62 (IQR 54-69) years, 84 % were males, 82 % had an initial shockable rhythm. Of the 124 patients 88 (71 %) were classified with an indication for ICA according to the ECG (Table). Post-ROSC ECG had a sensitivity of 88 % in detecting patients with an indication for ICA. ST-elevation and LBBB alone had a sensitivity of 72 %. Acute PCI was performed in 63 (51 %) patients. If post-ROSC ST-elevation or new LBBB alone had been used to select patients for ICA, 19 (11+8) out of 124 (15 %) patients would have missed a successful PCI.

Conclusion: ECG findings early after OHCA are difficult to interpret and should not be used as strict selection criteria for referral to hospitals with acute PCI service. Even in the absence of ST-segment deviation or LBBB on post-ROSC ECG, flow limiting coronary lesions may be present and patients may benefit from acute revascularization.

Table 1

		Atrial fibrillation, n=287	CV mortality, n=203		
		HR	95 % CI	HR	95 % CI
Weight categories*					
Normal weight, n=750	BMI <25 kg/m2	Reference	Reference	Reference	Reference
Overweight, n=2579	BMI 25-30 kg/m2	1.85	1.10 to 3.09	0.72	0.47 to 1.12
Obese, n=3840	BMI >30 kg/m2	2.85	1.73 to 4.71	0.96	0.63 to 1.46
Weight change**					
Weight gain, n=1023	>1 BMI unit	1.53	1.10 to 2.12	1.84	1.28 to 2.66
Weight stable, n=3736	± 1 BMI unit	Reference	Reference	Reference	Reference
Weight loss, n=2410	>1 BMI unit	1.06	0.81 to 1.38	1.03	0.75 to 1.43

**Adjusted for age, gender and systolic blood pressure. **Additional adjustments for baseline BMI.*

Table: ECG groups and angiography evaluation (n=124)

	Classification based on post-ROSC ECG evaluation		
	Group 1* (n=68)	Group 2* (n=20)	Group 3* (n=36)
Classification based on coronary angiography evaluation			
Indication for ICA	47 (69 %)	11 (55 %)	8 (22 %)
Occluded IRA or thrombus	33	6	2
Flow limiting stenosis	14	5	6

**Group 1: ST-elevation or new LBBB. Group 2: Other signs of coronary ischemia. Group 3: No signs of coronary ischemia.*

P4912 Physical fitness predicts myocardial infarction and heart failure but only when age-adjusted heart rate response to maximal exercise is low

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Purpose: Physical fitness (PF) has previously been reported to be inversely associated with future CVD risk. We have recently shown that maximal heart rate response to exercise interacts with PF in CVD risk estimation. The present study investigates if PF predicts acute myocardial infarction (MI) and heart failure (HF) independently of heart rate response in 2014 healthy men during 35 yrs follow-up.

Methods: PF, total work divided by body weight (kJ/kg), and heart rate response (bpm), were calculated and age adjusted in 2,014 apparently healthy, middle-aged men after a maximal bicycle exercise test in 1972-75. Events of MI and HF were adjudicated by scrutiny of medical records in all country's hospitals. Risk estimations were analysed in tertiles of PF using univariable and multivariable Cox proportional hazards models.

Results: Crude incidence of MI and HF were 449 (22 %) and 99 (5 %). Incidences of MI and HF were highest in the lowest PF-tertile (T1). T1 was associated with increased risk of MI and HF compared with T3 both in univariable and multivariable analysis. After stratifying the men by heart rate response, the results for were statistically significant among the men with low heart rate response only (Table).

Conclusions: Low PF was independently associated with increased risk of MI and HF over 35 yrs in apparently healthy, middle-aged men. After stratification, the prognostic value of PF was confined to the subgroup with poor heart rate response. Thus, assessment of PF and peak heart rate response to exercise may be clinically useful when judging risk of future MI and HF in apparently healthy middle-aged men.

Table 1

Heart rate response	Event	Low physical fitness (T1)	High physical fitness (T3)
		n=677	n=665
All	MI	1.41 (1.11–1.79)	1
Low	MI	1.96 (1.19–3.47)	1
High	MI	1.14 (0.67–1.88)	1
All	HF	1.70 (1.04–2.82)	1
Low	HF	2.19 (1.03–5.40)	1
High	HF	1.28 (0.56–2.75)	1

Values are hazard ratios (95 % CI); MI, myocardial infarction; HF, heart failure. All hazard ratios are adjusted for baseline age, cholesterol, smoking, significant family history of CHD and systolic blood pressure.

P4914 Physical fitness predicts early but not late myocardial infarction; a 35-year follow-up study of 2,014 healthy middle-aged men

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Background: Physical fitness (PF) has previously been shown to predict cardiovascular (CV) death and disease. In the present study we aimed to investigate how baseline PF influenced risks of myocardial infarction (MI) during the first and last part of a 35 year observation of healthy middle-aged men.

Methods: Age adjusted PF, total work divided by body weight (kJ/kg), was calculated in 2,014 apparently healthy, middle-aged men after a maximal bicycle exercise ECG test in 1972-75. Incident myocardial infarction was registered in a nationwide scrutiny of charts in Norwegian hospitals, and early vs. late event was set before or after median MI-age (66 years). Impact of predictors and relative risks between baseline quartiles of PF were estimated using Cox proportional hazards models. When estimating risks of late MI, men with events before 66 years were excluded.

Results: During follow-up; we found 224 and 225 events of early- respectively late MI. Age adjusted PF at baseline was a significant predictor of early- but not late MI. Family history of CHD, baseline smoking status and cholesterol were significant predictors of early MI, while baseline blood pressure and cholesterol

were significant predictors of late MI. Lower PF-quartiles were associated with significantly increased risks of early MI than the highest PF quartile (Q4) in unadjusted, age adjusted and multivariable analysis. There were no differences in risks of late MI among the PF-quartiles (Table).

Conclusions: PF was independently associated with risk of early- but not late MI. Most classical CV risk factors were strong predictors of both early and late MI. Low PF at middle-age could be interpreted as a warning sign of an early rather than late MI.

Table: Hazard ratios (95 % CI)

	Early MI	Late MI
PF (1 SD)	0.76 (0.65–0.88)	0.98 (0.83–1.15)
PF Q1	2.18 (1.47–3.30)	1.28 (0.86–1.90)
PF Q2	1.53 (1.01–2.36)	0.99 (0.68–1.46)
PF Q3	1.36 (0.88–2.11)	1.19 (0.83–1.71)
PF Q4	Reference	Reference

PF (1SD), one SD increase of PF; PF Q1–4, quartiles by baseline PF. All hazard ratios are adjusted for baseline age, family history of CHD, cholesterol, smoking and blood pressure.

P2079 Serum uric acid is associated with mortality and heart failure hospitalizations in patients with complicated myocardial infarction: analysis from High Risk Myocardial Infarction Database Initiative

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Background: Serum uric acid (SUA) levels predict poor outcome in patients with stable coronary heart disease. Whether SUA predicts outcome in acute myocardial infarction (MI) complicated by reduced left ventricular (LV) function, heart failure (HF) or both is unknown.

Methods: We studied the association between baseline SUA and outcomes using univariable and multivariable Cox models in an individual-patient data meta-analysis of 4 large randomized trials of high-risk MI (CAPRICORN, EPHESUS, OPTIMAAL and VALIANT; N=28,771).

Results: SUA was available in 12,677 patients (median follow-up 1.9 years). Patients were separated into quartiles (Q) according to baseline SUA (Q1, 45-280; Q2, 281-344; Q3, 345-420; Q4, 420-1640 micromol/l). Patients in higher SUA quartiles were older, more symptomatic, and had more comorbidity. Renal failure prevalence was 10-fold higher in Q4 vs Q1. All-cause survival at 3-years was 86.8 % in Q1 vs 69.4 % in Q4 (fig. A). Most deaths were due to CV disease (fig. B). In univariable analysis, all-cause mortality rose across SUA quartiles (Hazard ratio (HR)=1.06, confidence interval (CI)=0.92-1.22 for Q; HR=1.57, CI=1.31-1.71 for Q3; HR=2.70, CI=2.23-2.89 for Q4; Q1 as reference). A similar trend was observed for CV mortality (HR=2.70, CI=2.35-3.10 for Q4 vs Q1) and HF hospitalization (HR=1.72, CI=1.61-1.84 for Q4 vs Q1). Multivariable analysis models adjusted for baseline characteristics consistently showed that SUA independently predicted all-cause mortality (HR=1.78, CI=1.51-2.09 for Q4 vs Q1), CV mortality and HF hospitalization. Analysis in patients without diuretics yielded similar results.

Conclusions: Elevated SUA was a strong and independent predictor of poor clinical outcomes in patients after acute high-risk MI complicated by reduced LV function, HF or both.

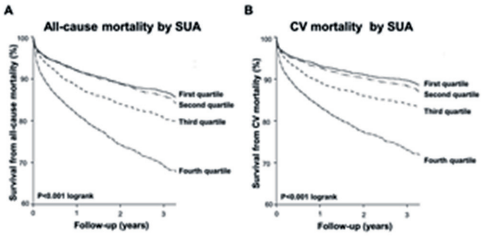

Figure: SUA and mortality in complicated MI.

P2053 Association between left atrial size and future atrial fibrillation: a 16 year follow up of 2369 women and men. Tromso study 1994-2010

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Purpose: Atrial fibrillation (AF) increases the lifetime risk of stroke and heart failure. Several risk factors of AF have been identified. The purpose of this study was to investigate the association between echocardiographic measurements with emphasis on left atrial (LA) size and future risk of incident AF in a large population-based cohort.

Methods: A random sample of 2369 participants in the Tromsø study in Northern Norway who were 50 years and older and without AF at baseline were followed from 1994 through 2010. LA size was indexed by body surface area

(BSA) and was categorized into three groups as normal (<2.2 cm/m2), moderately (2.2-2.79 cm/m2) and severely (>2.8 cm/m2) enlarged. To estimate sex-specific hazard ratios (HRs) for AF we used both age-adjusted and multivariable Cox proportional hazards regression models adjusted for age, systolic blood pressure, heart rate, body mass index, BSA, total and HDL cholesterol, and self-reported use of alcohol, smoking, coffee, physical activity, hypertension, prevalent coronary heart disease (CHD) and diabetes.

Results: Mean age at baseline was 62.6 years and 51.4 % were women. During follow-up we identified 462 cases of incident AF (193 women). LA size was associated with AF in both sexes. A moderately enlarged LA was in both women and men associated with 64 % increased risk for AF compared to subjects with normal LA size. In subjects with severely enlarged LA, we found HRs for AF of 4.4 (95 % CI, 2.6-7.4) in women and 3.9 (95 % CI, 2-7.6) in men compared with subjects with normal LA size (p-value for linear trend <0.001). Hypertension increased risk of AF (HR 1.6; 95 % CI, 1.3-1.9) in age and sex adjusted analysis. This relationship was somewhat weaker (HR 1.5; 95 % CI, 1.2-1.9) with additional adjustment for LA. Similarly, prevalent CHD was in age and sex adjusted analysis associated with AF (HR 2.4; 95 % CI, 1.9-3.0). When also adjusted for LA size, the HR was slightly attenuated (HR 2.2; 95 % CI, 1.7-2.9).

Conclusion: Enlarged LA size was independently associated with an increased risk of future AF in both sexes.

P2668 In-hospital and long-term mortality for patients 80 years or older with acute ST-segment elevation myocardial infarction. An eastern Norway cohort study 2005-2011

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Purpose: Patients ≥80 years with ST-elevation myocardial infarction (STEMI) are underrepresented in clinical trials, and little is known about long-term mortality of this population in the era of primary coronary intervention (PCI). Our hospital has offered primary PCI without age restrictions for several years. We aimed to study in-hospital and long-term mortality of STEMI patients ≥80 years compared to younger patients.

Methods: Single-centre observational cohort study. All consecutive STEMI-patients admitted to our hospital between 01.09.2005 and 31.12.2011 were included in a local registry. Predefined data including in-hospital mortality were registered. Vital status was obtained from the National Cause of Death Registry with censoring date 31.12.2011.

Results: A total of 4525 patients with a confirmed diagnosis of STEMI were registered; 600 patients (13 %) were ≥80 years. The percentage undergoing coronary angiography and PCI if indicated, was lower in patients ≥80 years compared with younger patients (83 % vs. 98 %), but there was no difference in symptom-to-balloon times. In the total cohort, in-hospital mortality was 4 % for patients <80 and 17 % for patients ≥80 years. In the invasively treated patients (96 % of all), inhospital mortality was 3.4 % in patients <80 years and 13 % in patients ≥80 years. The median follow-up time was 2.5 years. Survival in invasively treated patients ≥80 years was 66 % after 1 year and 58 % after 2 years (Figure). Factors associated with long-term mortality were prehospital resuscitation, elevated serum creatinine and PCI.

Conclusion: Mortality after STEMI was high in patients ≥80 years compared to younger patients. Although inhospital mortality was relatively low in patients treated invasively, only 58 % were alive after 2 years.

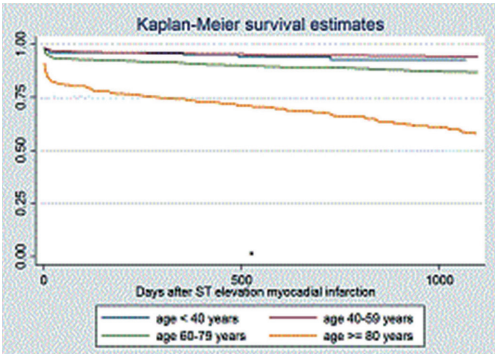

Figure 1

P728 Decreasing educational inequalities in percutaneous coronary intervention (PCI) utilization following an incident acute myocardial infarction (AMI) in Norway 2001-2009: a CVDNOR project

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Purpose: To explore national trends and educational differences in PCI utilization in patients with an incident AMI during 2001-2009.

Methods: Hospitalizations for an incident AMI in individuals 35-89 years were obtained from the Cardiovascular Disease in Norway (CVDNOR) project. Highest attained education was categorized into: basic (1st), secondary (2nd) and tertiary (3rd). Age-standardized rates of patients receiving PCI within 28 days were calculated and trends were analyzed by Joinpoint regression. Results are expressed as average annual percentage change (AAPC) in PCI rates. Educational differences were explored using multivariable Poisson regression.

Results: Of 104,836 patients (mean age (SD) 71.1 (12.7) years; 62.7 % men), 30.4 % underwent PCI within 28 days.

In men, PCI rates increased during 2001-2009 in all education levels (1st: AAPC=14.2 % [95 %CI; 11.1-17.4]; 2nd: AAPC=13.9 % [10.0-18.0]; 3rd: AAPC=9.9 % [7.1-12.8]). Similar trends were observed among women (1st: AAPC=13.8 % [95 %CI; 8.3, 19.5]; 2nd 10.6 % [95 %CI; 7.0, 14.4]; 3rd: AAPC=8.9 % [1.2, 17.2]).

Compared to patients with basic education, those with secondary and tertiary education had 12 % (RR=1.12, 95 % CI; 1.10-1.14) and 21 % (RR=1.21, 95 % CI; 1.18-1.24) higher rates of PCI within 28 days.

The observed gap in receiving PCI was continuously narrowing and differences became insignificant in 2009 ($p<0.01$ for interaction between calendar year and education levels) (Fig. 1).

Conclusion: PCI utilization in patients hospitalized for an incident AMI increased significantly in Norway during 2001-2009, especially among people with basic education. Patients with secondary and tertiary education had higher PCI rates compared to those with basic education, but these differences decreased over time.

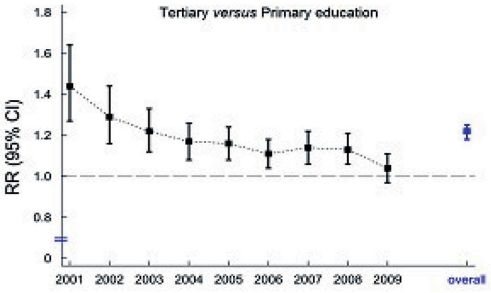

Figure 1. RR for receiving PCI after AMI

994 Everolimus initiation with early calcineurin inhibitor withdrawal reduced allograft vaculopathy in de-novo heart transplant recipients: results of the SCHEDULE trial

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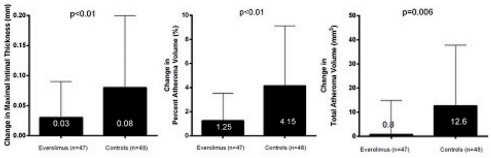
Purpose: Early initiation of everolimus after heart transplantation (HTx) with calcineurin inhibitors (CNI) has been shown to reduce cardiac allograft vasculopathy (CAV). The SCHEDULE trial demonstrated that CNI therapy can in fact be safely withdrawn early after HTx with a beneficial effect on renal function. The effect of this strategy on CAV has not been investigated previously and was a pre-specified endpoint of the SCHEDULE trial.

Methods: The SCHEDULE trial was a 12-month multicenter Scandinavian trial where 115 de-novo HTx recipients were randomized to everolimus with complete CNI withdrawal (EVE-group) 7-11 weeks after HTx or standard CNI therapy (CNI group). 95 (83 %) patients had matched intravascular ultrasound examinations allowing change in Maximal Intimal Thickness (MIT), Percent Atheroma Volume (PAV) and Total Atheroma Volume (TAV) to be assessed along with measurement of serum inflammatory markers.

Results: Mean recipient age was 49.9 ± 13.1 yrs. The EVE group (n=47) demonstrated significantly reduced CAV progression as compared to CNI (n=48) [ΔMIT 0.03 ± 0.06 and 0.08 ± 0.12 mm, ΔPAV 1.3 ± 2.3 and 4.2 ± 5.0 %, ΔTAV 0.8 ± 14.1 mm3 and 12.6 ± 25.2 mm3 (all p-values<0.01), respectively]. EVE patients had a significantly greater decline in soluble

tumor necrosis factor receptor (sTNR)-1 levels as compared to CNI (p=0.02) but there was no significant difference in change in levels of CRP, VCAM, VEGF, vWf, IL-8 (p>0.05).

Conclusion: Everolimus initiation and CNI withdrawal early after HTx significantly reduces CAV as assessed by IVUS. This strategy appears to have some impact on systemic inflammation as reflected by a significantly greater decline in sTNFR-1. Given the dramatically benefical effect of everolimus on CAV this novel CNI-free approach should be considered in all de-novo HTx patients.



Figur 1

36 Postsystolic shortening measured early in PCI-treated STEMI patients is a strong predictor of myocardial salvage and left ventricular recovery: A cross-modality imaging study

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Purpose: Assessment of potential recovery of myocardium at risk after reperfusion is difficult in patients with ST elevation myocardial infarction (STEMI). Previous studies have indicated that postsystolic shortening (PSS) is associated with myocardial recovery in patients with non-STEMI. However, little is known about a possible association between PSS and myocardial salvage in patients with STEMI. The aim of the study was to evaluate the association between myocardial strain measured by echocardiography in the acute stage and myocardial salvage measured by repeated cardiac magnetic resonance (CMR) imaging.

Methods: The study population consisted of 100 patients with first-time STEMI treated by primary PCI. Global longitudinal peak systolic strain (εSYS), and peak strain (εPEAK) were measured by two-dimensional speckle tracking echocardiography at a median of 2.4 (range 1-5) days after PCI. Postsystolic index (PSI) was calculated manually, PSI = [(εPEAK − εSYS):εPEAK] x 100. εSYS and PSI were dichotomized into two groups and defined as, those with low negative strain values (minimum to median) and those with high negative strain values (median to maximum). Myocardial salvage index (MSI), infarct size (IS) and ejection fraction (EF) were assessed by CMR performed both in the acute stage and after 4 months. MSI was defined as [(myocardium at risk − infarct size):myocardium at risk] x 100.

Results: Median values of εSYS and PSI were −13.1 and 9.6, respectively. Low negative εSYS range was from −3.2 to −13.1 and high negative εSYS range was from −13.2 to −22.3. Low PSI range was from 0.0 to 9.6 and high PSI range was 9.7 to 49.1.

Conclusion: Reduced negative systolic strain and a high degree of postsystolic shortening measured in the acute stage were associated with large infarct size, low ejection fraction and impaired myocardial salvage in STEMI patients treated by primary PCI.

See table

	Global longitudinal peak systolic strain (εSYS)			Post-systolic index (PSI)		
	Low εSYS	High εSYS	p-value	Low PSI	High PSI	p-value
Ejection fraction (%)	51.5	60.0	0.0002	60.0	51.0	0.0005
Infarct size (% of left ventricular mass	16.6	11.0	0.001	11.2	15.9	0.002
Myocardial salvage index	43.6	56.9	0.06	60.5	43.6	0.004

1233 Prevalence of risk factors at presentation and early mortality in patients 80 years or older with ST-segment elevation myocardial infarction

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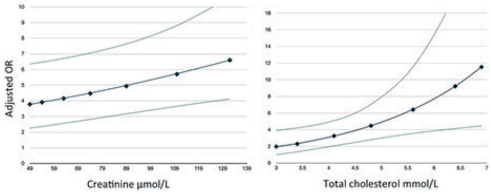
Purpose: Elderly patients with ST-segment elevation myocardial infarction (STEMI) are at high risk of complications and early mortality; still they are underrepresented in clinical trials and observational studies. We aimed to study risk profiles at presentation and early mortality in older (≥80 years) vs. younger (<80 years) STEMI-patients.

Methods: Prospective cohort study. The study population comprised 4092 consecutive STEMI patients admitted to our university hospital during 2006 to 2010. Baseline characteristics at admission were recorded as well as in-hospital mortality. Explanatory strategy was used in the analyses.

Results: Patients ≥80 years (n=536) were more likely to be female, have prior myocardial infarction, angina and stroke, but less likely to be current

smokers. The crude in-hospital mortality rate was 16.2 % in patients ≥80 years and 3.5 % in those <80 years (crude odds ratio 5.41, 95 % confidence interval, 4.0-7.3). The adjusted odds ratio for mortality for patients ≥80 vs. <80 years increased with increasing levels of serum creatinine and total cholesterol (Figure). In patients with low levels of serum creatinine and total cholesterol, the odds ratio was 3.01 (95 % confidence interval, 1.86-4.93; p=0.0001); increasing to 11.72 (95 % confidence interval 5.26-26.3; p=0.001) in patients with high levels of both risk factors.

Conclusion: High levels of serum cholesterol and creatinine were important risk factors for early mortality in elderly patients with STEMI. Depending on the levels of cholesterol and creatinine, in-hospital mortality in patients ≥80 years varied from a three-fold to an almost twelve-fold risk compared to younger patients.



Figur: Adjusted OR for mortality, ≥80 vs <80yrs

P3643 Impaired left ventricular diastolic function in adult survivors of childhood acute lymphoblastic leukemia treated with anthracyclines

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Purpose: Anthracycline therapy carries a dose dependent risk of late cardiotoxicity. Left ventricular (LV) diastolic dysfunction is thought to precede systolic dysfunction. Tissue Doppler imaging (TDI) with measurement of peak early diastolic velocities of the mitral annulus (e') is recommended to detect diastolic dysfunction. Several recent, smaller studies of childhood cancer survivors have failed to show reduced e' associated with anthracycline therapy. We wanted to test the hypothesis that antracycline therapy leads to LV diastolic dysfunction in the very long term.

Methods: In a cross-sectional study of 138 childhood acute lymphoblastic leukemia (ALL) survivors, mean 23 years after diagnosis, echocardiography with measurement of e' was performed, and compared to 138 randomly selected, healthy controls matched 1:1 for gender, age, body weight and systolic blood pressure.

Results: ALL survivors and controls had equal body weight and systolic blood pressure. Survivors had lower e' than controls. Anthracycline treated survivors (median cumulative dose 120 mg/m2) were younger than the other survivors, but e' did not differ between survivor groups (table). However, anthracycline treated survivors had lower e' than age-matched controls (p>0.001, table), whereas anthracycline naïve survivors and age-matched controls had equal e' (p>0.5). LV ejection fraction did not differ between survivors and controls, but was lower in anthracycline treated survivors compared to other survivors.

Conclusions: Anthracyclines lead to impaired LV diastolic function in the long term. As e' decreases with age, age-matched controls are necessary to detect the premature decline in diastolic function found after anthracycline therapy.

P5511 Markers of thrombin generation are associated with myocardial necrosis and left ventricular impairment in patients with ST-elevation myocardial infarction

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Introduction and aim: Thrombin generation and fibrin formation play an important role in intracoronary thrombus formation, which may lead to an acute myocardial infarction.

Aim of the present study was to investigate whether D-dimer, pro-thrombin fragment 1+2 (F1+2) and endogenous thrombin potential (ETP) as markers of in vivo and ex vivo thrombin generation, respectively, are associated with myocardial necrosis assessed by Troponin T (TnT), and left ventricular impairment assessed by left ventricular ejection fraction (LVEF) and NT-proBNP.

Methods: Patients with ST-elevation myocardial infarction (STEMI) from a cross sectional cohort study (n=993), referred for primary percutaneous coronary intervention (PCI) were included. Median age was 61 years (range 24-94), 80 % male. Blood samples were drawn the first morning after admission at a median time of 24 hours after onset of symptoms. D-dimer and F1+2 were determined by ELISA and ETP by the

Table 1

	Survivors (n=138)	Controls (n=138)	p-value	Anthracyclines (n=107)	Controls (n=107)	p-value	No anthracyclines (n=31)	p-value (vs other survivors)
Age at exam	29.5 ± 7.2	29.8 ± 7.4	0.667	27.5 ± 6.6	27.9 ± 7.0	0.618	36.3 ± 4.8	<0.001
Heart rate	66 ± 11	65 ± 11	0.319	67 ± 11	65 ± 11	0.132	63 ± 10	0.080
e' septum	11.1 ± 2.8	12.6 ± 2.5	<0.001	11.0 ± 2.7	13.0 ± 2.5	<0.001	11.1 ± 3.2	0.924
E/e' average	6.0 ± 1.5	5.6 ± 1.3	0.015	6.1 ± 1.5	5.4 ± 1.2	0.001	5.8 ± 1.3	0.454
EF	57 ± 6	57 ± 5	0.614	56 ± 4	57 ± 6	0.162	60 ± 5	<0.001

e': tissue Doppler peak early diastolic velocity of the mitral annulus; EF: ejection fraction.

CAT-assay. Patients on warfarin were excluded from analysis.

Results: In the total population levels of D-dimer, F1+2 and ETP (median (25,75 percentiles)) were 456 ng/L (287,796), 246 pmol/L (178,356), 1564 nM (1366,1743), respectively.

Significant correlations were found between both peak TnT and D-dimer (r=0.260, p<0.0001) and F1+2 (r=0.364, p<0.001) and between NT-proBNP and D-dimer (r=0.243, p=0.001) and F1+2 (r=0.120, p=0.0001). When dividing TnT and NT-proBNP levels into quartiles there were significant trends for increased levels of both markers across quartiles (all p<0.0001). No significant associations between TnT, NT-proBNP and ETP were found. When adjusting for relevant covariates (age, gender, BMI and NT-proBNP), both D-dimer and F1+2 remained significantly associated with peak TnT (both p=0.0001).

D-dimer remained significantly associated with NT-proBNP after adjustments (p=0.001), whereas the association between NT-proBNP and F1+2 was no longer statistically significant (p=0.301). A weak, but statistically significant inverse correlation was found between LVEF and D-dimer (r=0.160, p=0.0001) and F1+2 (r=0.090, p=0.011). When dichotomizing LVEF levels at 40 %, we observed significantly higher levels of both D-dimer (p=0.0001) and F1+2 (p=0.014) in the group with low EF (n=147). No difference in ETP levels was found.

Conclusion: In a large cohort of STEMI patients, levels of D-dimer and F1+2 were significantly associated with the extent of myocardial necrosis assessed by peak TnT. The high levels of these coagulation markers in patients with low LVEF and high NT-proBNP may indicate a hypercoagulable state in patients with impaired myocardial function.

P6551 Dual-acting angiotensin-receptor neprilysin-inhibition attenuates post-myocardial infarction cardiac remodelling and angiotensin-II-induced cardiorenal injury in vitro

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Background: Novel angiotensin-receptor neprilysin (NEP) inhibitors (ARNi) demonstrated benefits above single RAAS-blockade in early clinical trials, but mechanisms underlying cardiorenal protection by ARNi are unknown. We evaluated ARNi in cardiac remodelling after experimental myocardial infarction (MI), and in angiotensin-II (AngII) induced hypertrophy and fibrosis in cardiorenal cell lines.

Methods: One week after induction of MI, adult male rats were randomized and treated for four weeks with the ARNi LCZ696 (68 mg/kg body weight PO; ARNi-MI, n=11) or vehicle (VHC-MI, n=6). Echocardiography and organ weights served to assess cardiac structure and function at 5 weeks after MI. AngII-stimulated (100nM) 3[H]leucine-incorporation in cardiomyocyte over 60 h served to evaluate cardiac hypertrophy. 3[H]proline-incorporation in cardiac fibroblast or renal mesangial cells over 48 h (CF) served to evaluate collagen accumulation. Cells were pre-incubated with valsartan (VAL), NEP inhibitor LBQ657 (LBQ; 10μM), or both (ARNi).

Results: MI-ARNi had lower heart weights (1168 ± 35 vs 1319 ± 21 mg) and left ventricluar (LV) end-diastolic diameter (9.7 ± 0.2 vs 10.5 ± 0.3), and higher LV ejection fraction (60 ± 2 vs 47 ± 5 %) compared to MI-VHC (all p<0.05). Cell data, see table: ARNi at high dose abrogated AngII-stimulated

Table: Effects of ARNi in vitro

VAL (mM)	0.0	0.03	0.1	1.0
Cardiomyocytes: hypertrophy				
VAL alone	128 ± 2****	116 ± 1##	111 ± 2###	109 ± 3###
ARNi (VAL+ LBQ 1µM)	109 ± 2###	107 ± 3###	110 ± 3###	98 ± 3###
Cardiac fibroblasts: collagen accumulation				
VAL alone	211 ± 6****	164 ± 6###	144 ± 5###	113 ± 3###
ARNi (VAL+ LBQ 1µM)	170 ± 11###	123 ± 7###	115 ± 4###	101 ± 3###
Renal mesangial cells: collagen accumulation				
VAL alone	144 ± 6****	114 ± 5###	111 ± 6###	–
ARNi (VAL+ LBQ 1µM)	118 ± 4###	103 ± 7###	97 ± 13###	–

*Cell data normalized to unstimulated control (=100 %). ****p<0.0001 vs unstimulated control, ###p<0.001 vs stimulated control.*

cellular cardiac hypertrophy and cardiorenal fibrosis.

Conclusions: ARNi attenuates cardiac remodelling after MI, possibly by potent anti-fibrotic and anti-hypertrophic effects in cardiorenal tissues.

P5164Women's hearts - fixed and healthy after PCI?

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Purpose: To explore and describe the experiences of women with ischemic heart disease (angina pectoris and myocardial infarction) after Percutan Coronary Intervention (PCI). Furthermore, the study examined changes in daily life affected by the PCI and how women relate to heart-healthy lifestyle.

Methods: Data were collected through qualitative interviews in the respondents' home by using a semi-structured interview-guide reflecting a promotion of health perspective by the International Classification of Function (ICF) model and adherence to heart-healthy lifestyle approach in terms of the Health Belief Model. The interviews lasted for 1-1.5 hours. The data were analyzed in four steps with Malterud's modified version systematic text condensation (STC), based on Giorgi's phenomenological method of qualitative data analysis.

Results: A purposive sample of nine women aged 55-64 were interviewed in 2003. The women were characterized by living alone, low education, incapacitated, sick leave and additional diseases. Risk factors were identified as smoking (n=4), diabetes (n=1), high blood pressure (n=2) and heritage (n=5). Data analysis revealed four main categories: 1. "Experiences in the days before PCI" meaning trivializing cardiac symptoms, undertreated and underdiagnosed, 2. "Experiences with percutaneous coronary intervention" expressed as feeling "fixed" and healthy with few complications, by large being asymptomatic, requesting more information and follow-up, 3. "Experiences in the post-PCI" representing reactions of joy, appreciation of life and lability, consciousness in avoiding stress and focusing boundaries, and 4. "Compliance with heart-healthy lifestyle" in terms of concerning hereditary, symptom management and side effects of medications. Individual risk factors were given little attention, no one stopped smoking and the level of physical activity was generally low. Diet, however, was to a certain degree adjusted to heart friendly advice and recommendations.

Conclusion: PCI was experienced as a quick and uncomplicated treatment which made the women feeling repaired and "fixed". Being free from symptoms after PCI challenges the understanding of ischemic heart disease as a chronic illness. In all, lifestyle was only to a limited extent changed after PCI and the women lived largely as before. Individual health promoting approaches and systematic use of assessment tools, including socio-demographic data and risk assessment factors, may strengthen the focus on women's resources as well as opportunities and needs, to comply with recommendations on heart-healthy lifestyle.

P5381 Peroperative improvement in left ventricle longitudinal motion after transcatheter aortic valve implantation predicts better outcome

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Purpose: LV function is expected to improve immediately after transcatheter aortic valve implantation (TAVI) when afterload is reduced, but does not occur in all patients due to limited myocardial reserve. We hypothesized that peroperative improvement in systolic LV longitudinal motion after TAVI predicts better outcome.

Methods: 64 pts (mean age 81 (7) yrs) scheduled for TAVI were included. Transoesophageal 4 and 2ch echocardiograms were obtained immediately before and ~15 min after valve implantation. Peak systolic myocardial velocity (s') by tissue Doppler Imaging (TDI) was obtained from 8 basal segments, and averaged.

Pts were predefined responders for improved systolic function if TDI s' increased ≥20 % after the procedure. Outcome 3 months after TAVI was assessed by improvements in NYHA class, proBNP and systolic function (transthoracic s' (apical 4ch view)).

Results: 34 pts were classified responders and s' increased from 2.2 (0.8) to 3.1 (1.1) cm/s ($p<0.05$). In 29 pts s' remained unchanged at 2.4 (1.1) cm/s. Age, gender, preoperative NYHA class, proBNP, Euroscore and perioperative handling were similar in responders (R+) and non-responders (R−). Only responders had improved systolic function and reduced proBNP

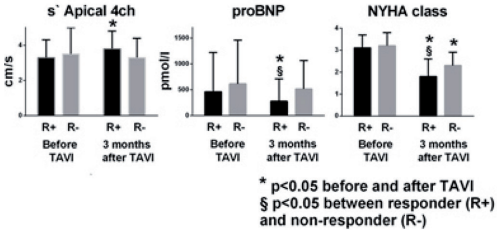

Figure 1

3 months after TAVI accompanied with a significantly better NYHA class compared to non-responders (Fig. 1).

Conclusion: Immediate improvement in longitudinal s' during the TAVI procedure predicted better outcome after 3 months with improved systolic function, proBNP and NYHA class. Peroperative TDI may be used to identify pts with less favorable outcome after TAVI.

P2673 Chemokine (CXC motif) ligand 16 (CXCL16) and osteoprotegerin (OPG) as predictors of outcome in patients with acute coronary syndromes (ACS) - a PLATO biomarker substudy

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Background: Early identification of patients with poor prognosis may help to optimize therapy in patients with acute coronary syndromes (ACS). Both CXCL16 and OPG may play a modulatory role in platelet-mediated vascular inflammation and, at least partly, represent different inflammatory pathways. We evaluated factors associated with OPG and CXCL16 levels, their relation to 1 year outcome and interactions with platelet inhibition in patients with ACS.

Methods: CXCL16 and OPG levels were determined in serum collected at randomization to ticagrelor or clopidogrel in 4185 patients with ACS in the PLATO (PLATelet inhibition and patient Outcomes) trial. Baseline characteristics by Chi-square test (categorical variables) or Kruskal Wallis test (continuous variables), and occurrence of the primary outcome, a composite of cardiovascular death, non-fatal myocardial infarction, or stroke (n=362), by Cox proportional hazards models were compared between groups stratified by quartiles of the respective biomarker.

Results: The median (interquartile range) of CXCL16 was 5.10 (4.4 – 6.0) and of OPG 2.6 (2.0 – 3.6). Patients with higher CXCL16 and OPG serum levels were older and more frequently were female and had a medical history including hypertension, diabetes mellitus, angina pectoris, heart failure, PCI, and chronic renal disease. In addition, patients with higher CXCL16 but lower OPG had a higher frequency of smoking, prior PCI, angina pectoris and ST-elevation MI. Higher CXCL16 levels were also associated with more previous MI and peripheral artery disease. Finally, higher CXCL16 and OPG were strongly associated with increased TIMI and GRACE risk scores and higher levels of troponin I, NT-proBNP and CRP. There was a higher incidence of the primary endpoint in the highest (12.1 %/year) versus the lowest (7.4 %/year) CXCL16 quartile (HR 1.63 95 % CI [1.22-2.18], p=0.004 in comparison to the lowest quartile). For OPG there was an increased risk of the primary outcome in the two higher quartiles (11.2 and 11.4 %/year) (HR 1.64[1.21-1.22] and 1.68 [1.24-2.28], p=0.0002 in comparison to the lowest quartile 6.8 %/year). No significant interaction between biomarkers and effect of randomized treatment on the primary outcome was observed.

Conclusions: In patients with ACS higher serum levels of CXCL16 and OPG are associated with clinical risk factors and adverse outcome but did not identify subgroups of patients with specific benefits from more intense antiplatelet therapy.

P1691 Secreted wingless (Wnt) antagonists and risk prediction in acute coronary syndrome (ACS) - a PLATO biomarker substudy

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Background: Recent studies suggest that the Wnt pathway may be implicated in vascular remodeling and atherogenesis. To gain further insight into the role of Wnt-related mediators in atherosclerotic disorders, we investigated the prognostic value of the Wnt pathway modulators serum secreted frizzled related protein (sFRP-3) and Dickopff-1 (DKK-1) for 1-year outcome in patients with acute coronary syndromes (ACS).

Methods: sFRP-3 and DKK-1 levels were determined in serum collected at randomization to ticagrelor or clopidogrel in 4201 patients with ACS in the PLATO (PLATelet inhibition and patient Outcomes) trial. Baseline characteristics by Chi-square test (categorical variables) or Kruskal Wallis test (continuous variables), and occurrence of the primary outcome, a composite of cardiovascular death, non-fatal myocardial infarction, or stroke (n=363), by Cox proportional hazards models were compared between groups stratified by quartiles of the respective biomarker.

Results: The median (interquartile range) of DKK-1 was 0.6 (0.2 – 1.3) and of sFRP-3 13.0 (3.3 – 36.8). Higher frequencies of hypertension, diabetes mellitus, angina, previous MI and heart failure and higher levels of CRP and NT-proBNP were observed with increasing quartiles of DKK-1 ($p<0.001$ for all, except diabetes $p<0.01$), and opposite trends were seen for sFRP-3 with the lowest risk factor frequencies and CRP and NT-proBNP levels in the top quartile ($p<0.001$ for all). Similarly, while there was a decrease in proportion of ST-elevation MI with increasing DKK-1 quartile (q1: 65 %, q4: 35 %, $p<0.001$), this pattern was opposite for sFRP-3 (q1: 38 %, q4: 72 %, $p<0.001$). Both DKK-1 and sFRP-3 showed transient association with troponin I with the highest levels in the mid quartiles. Baseline levels of DKK-1 and sFRP-3 were strongly inversely correlated ($r=-0.52$, $p<0.001$). Both biomarkers were moderately but significantly associated with the primary outcome ($p<0.05$) with an inverted U shaped association with risk, in particular for sFRP-3, where the lowest HR was found in the top quartile. No significant interaction between biomarkers and effect of randomized treatment on the primary outcome was observed.

Conclusions: Higher DKK-1 was less often seen in STEMI, while this trend was opposite trend for sFRP-3. There was a significant inverted U shaped prognostic pattern of Wnt anagonists concerning outcomes in ACS. This patterin indicates a complex role for the Wnt pathways in clinical atherosclerosis involving regulation of soluble Wnt modulators.

P1693 Pentraxin 3 (PTX3) predict adverse outcome in acute coronary syndromes - a PLATO biomarker substudy

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Purpose: PTX3 is expressed by the major cell types involved in atherosclerosis in response to inflammatory stimuli and may be regarded as a marker of local vascular inflammatory response. In the current study we investigated the association between levels of PTX3 and 1-year outcomes in patients with acute coronary syndromes (ACS).

Methods: PTX3 was determined in plasma sampled at randomization to ticagrelor or clopidogrel in 4191 patients with ACS enrolled in the PLATO (Platelet Inhibition and Patient Outcomes) trial. Patients were categorized based on quartiles of PTX3 levels and compared with respect to baseline characteristics (Chi-square or Kruskal Wallis tests for categorical and continuous variables, respectively), and to the primary composite endpoint of cardiovascular death, myocardial infarction (MI), or stroke (Cox proportional hazards models).

Results: The median (interquartile range) of PTX3 was 1.9 ng/mL (1.2 – 3.0 ng/mL). Higher PTX3 levels were associated with higher age, higher number of female gender, lower Body Mass Index, less smokers, higher frequency of chronic kidney disease ($p<0.001$ for all), and lower occurrence of ST-elevation MI ($p=0.0071$). Higher PTX3 levels were also associated with both TIMI and GRACE risk scores, and with higher Troponin I, NT-proBNP, and C-reactive Protein levels ($p<0.0001$ for all). No association was found with presence of diabetes mellitus or past medical history (e.g. coronary or peripheral artery disease, congestive heart failure, or stroke). Higher rates of the primary endpoint were found in the two upper PTX3 quartiles (Q3 and Q4: 10.5 and 11.7 %/year; HR [95 %CI]: 1.47 [1.08-1.98] and 1.64 [1.22-2.20], respectively) in

comparison to the lowest quartile (7.1 %/year), p=0.0006. There was no significant interaction between PTX3 levels and the effects of randomized treatment.

Conclusions: In patients with ACS, PTX3 levels are related to age and markers of myocardial damage and dysfunction, kidney dysfunction and inflammatory activity. High admission levels of PTX3 are associated with adverse clinical outcomes up to 12 months after ACS. The benefits of ticagrelor versus clopidogrel are independent of the PTX3 levels.

P1731 Chromogranin A is a potent risk marker in acute heart failure and directly associated with cardiomyocyte Ca2+ handling

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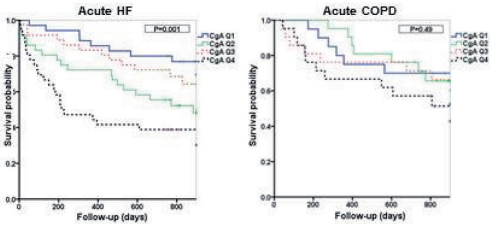
Background: Chromogranin A is considered as an unspecific prognostic biomarker, but given the established role of granin proteins on Ca2+ handling in non-cardiac cells, we hypothesized that CgA may (1) directly influence cardiomyocyte Ca2+ handling and (2) specifically be associated with mortality in patients with acute heart failure (HF).

Methods and results: We measured circulating CgA levels in patients with acute dyspnea due to HF (n=143) and chronic obstructive pulmonary disease (COPD, n=83) and explored the effect of the CgA fragment catestatin (Cts, CgA352-372) on cardiomyocyte Ca2+ handling. HF and COPD patients were matched on NYHA functional class (p=0.13) and mortality rates (median 813 days follow-up: 66 HF deaths [46 %] vs. 35 COPD deaths [42 %], p=0.56), but CgA levels were only associated with HF mortality (Fig). The association between CgA levels and HF mortality was also found after adjusting for other risk factors in multivariate Cox regression model: HR logCgA 1.43 (95 % CI 1.11-1.83), p=0.005. By experimental methods, Cts reduced Ca2+ sparks in all dimensions and Ca2+ spark and wave frequency. Cts also increased the magnitude of cardiomyocyte Ca2+ transients and induced larger and faster contractions of cardiomyocytes. Previously studies have found Cts to be internalized into cells by endocytosis and we observed a direct binding of Cts to the catalytic region of the Ca2+/calmodulin (CaM)-dependent protein kinase II d (CaMKIId), which is a nodal kinase regulating cardiomyocyte Ca2+ handling. Cts also attenuated CaMKIId autophosphorylation in



cells and perfused hearts and reduced CaMKIId activity in vitro.

Conclusions: CgA is a potent risk marker in patients with acute HF and directly associated with cardiomyocyte Ca2+ handling.

P4199 Aerobic interval exercise training improves the apnea-hypopnea index and self-reported sleepiness in obstructive sleep apnea patients

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Purpose: To investigate if 3 months of high intensity aerobic interval training improves the obstructive sleep apnea-hypopnea index (events h-1) in obese patients diagnosed with moderate to severe obstructive sleep apnea.

Methods: In a prospective randomized controlled study 30 Thirty obese (BMI = 37 ± 6 kg/m2) men and women (51 ± 9 years, height 177 ± 9 cm) diagnosed with obstructive sleep apnea for 5.7 ± 4.6 years (Apnea-hypopnea index (AHI index) = 42 ± 26) was randomized 1:1 to 12 weeks of supervised aerobic interval exercise training (AIT) or control (CON). AIT was performed as 4 x 4 minutes of treadmill running or walking at 90-95 % of maximal heart rate two times per week. CON continued with their normal lifestyle. Subjects were investigated at baseline and 12 weeks and sleep evaluation with respiratory polygraphy (blinded data evaluation), cardiopulmonary exercise testing, spirometry, blood biomarkers and self-reported sleepiness was investigated.

Results: Twenty-eight subjects completed the intervention period. Twenty-four subjects were regular CPAP users. The AHI-index was improved from 31.4 ± 21.7 to 23.9 ± 20.4 in the AIT group after 12 weeks of AIT ($p \leq 0.05$) and was unchanged in CON (50.3 ± 25.5 at

baseline and 46.6 ± 26.3 at 12 weeks). The Epworth self-reported sleepiness scale was improved from 10.0 ± 3.6 to 7.3 ± 3.7 in the AIT (p≤0.05) and was unchanged from baseline (5.9 ± 4.3) after CON. There was no change in body weight (120.5 ± 26.1 kg and 118.6 ± 23.1 kg in AIT/CON), average sleep oxygen saturation (92.4 ± 1.6 % and 92.6 ± 1.6 % in AIT/CON) or number of sleeping oxygen desaturation events per hour (36.9 ± 18.6 event·h1- and 55.8 ± 28.6 event·h1- in AIT/CON) from baseline to after the intervention period. Maximal oxygen uptake improved from 28.2 ± 7.4 ml·kg1-·min1- to 30.2 ± 7.7 ml·kg1-·min1- in the AIT group (p≤0.05), and was unchanged from baseline (27.0 ± 7.3 ml·kg1-·min1-) in CON. Pulmonary function was unchanged in both groups (Baseline FVC of 4.3 ± 0.9 L and 4.4 ± 0.8 L, and baseline FEV1 of 3.4 ± 0.6 L and 3.4 ± 0.6 L in the AIT and CON respectively). Leptin was significantly reduced after AIT (from 1747 ± 1419 pmol·l1- to 1412 ± 1047 pmol·l1- (p≤0.05), with no change in CON (1656 pmol·l-1 at baseline).

Conclusion: Twelve weeks of aerobic interval exercise training two times per week significantly improves the AHI index, self-reported sleepiness and maximal oxygen uptake in obese sleep apnea patients without any change in body weight.

P2663 Cardiac troponin I for prediction of clinical outcomes and cardiac function through three months follow-up after primary percutaneous coronary intervention for ST-elevation myocardial infarction

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Purpose: Circulating levels of cardiac troponin I (cTnI) following ST-elevation myocardial infarction (STEMI) are associated with infarct size and chronic left ventricular dysfunction, but the relation to clinical endpoints and biochemical measures of global cardiac function remains less well-defined. The objective of this study was to investigate whether various cTnI variables were associated with clinical outcomes and cardiac function through three months follow-up after primary percutaneous coronary intervention (PCI) for STEMI.

Methods: A substudy of the PROTECTION AMI trial. CTnI was measured at several time points during the index hospitalisation and patients were followed for three months before reassessment including N-terminal pro-B-type natriuretic peptide (NT-proBNP) and left ventricular ejection fraction (LVEF) measurements. In multivariable regression models with a composite clinical endpoint (death, cardiogenic shock during the index hospitalization, congestive heart failure, or serious arrhythmia), NT-proBNP >118 pmol/L or LVEF <40 % as dependent variables, different cTnI variables (admission, several post-PCI single-points, estimated peak and estimated area under curve (AUC) were tested after adjustments for age, gender, hypertension, diabetes, prior heart failure, prior MI, systolic blood pressure, heart rate, weight, Killip class category, infarct location and time from symptom onset to PCI.

Results: 1066 patients all receiving PCI within six hours were included. The median (quartile 1-3) cTnI levels were 0.4 (0.1-0.4) ug/L at admission, 33.1 (12.8-72.1) ug/L after 16-24 hours, 9.1 (3.9-17.5) ug/L after 70-80 hours and 1349 (486.1-3003) ug*hr/L for AUC. In the multivariable models for predicting clinical events, NT-proBNP >118 pmol/L or LVEF <40 %, all post-PCI single-points and estimated cTnI variables were found to be independently associated to the dependent variables (p for all <0.001). The C-statistic of the clinical endpoint model improved from 0.767 (baseline model with clinical risk factors) to 0.832 when cTnI at 16-24 hours was added and 0.843 when cTnI AUC was added. Respectively, for the NT-proBNP model from 0.789 to 0.860 and 0.858, and for the LVEF model from 0.683 to 0.841 and 0.854. Quantified by the integrated discrimination improvement the addition of these cTnI variables significantly bettered prediction (p for all <0.001).

Conclusions: CTnI measurements taken after primary PCI for STEMI are associated with clinical outcomes and cardiac function through three months follow-up. These results suggest that cTnI is a useful risk stratification tool in STEMI patients.

P877 Short duration of symptoms at presentation is associated with left ventricular recovery in dilated cardiomyopathy: results from a prospective cohort study

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Purpose: Outcome data in dilated cardiomyopathy (DCM) stem from research performed

Table: Predictors of delta EF

Variable	Baseline value	Univariate r	p-value	Multivariate β	p-value
Age (years)	51 ± 14	−0.18	0.08	0.03	0.77
Gender (%M/F)	73/27	0.06	0.70	0.02	0.77
Body mass index	28 ± 5	−0.14	0.18	−0.10	0.27
Systolic blood pressure	116 ± 20	−0.11	0.27	0.11	0.27
Heart rate	75 ± 16	0.34	0.001	0.13	0.21
Duration of symptoms (days)	208 (93-477)	−0.45	<0.001	−0.45	<0.001
LVEF (%)	26 ± 10	−0.56	<0.001	−0.57	<0.001
Peak VO2 (% of expected value)	69 ± 22	−0.20	0.06	0.05	0.65
Pulmonary capillary wedge pressure (mmHg)	15 ± 8	0.23	0.03	−0.12	0.25
NT-proBNP (pg/ml)	1332 (584-2901)	0.30	0.003	0.02	0.88

prior to the widespread use of inhibitors of the renin-angiotensin-aldosterone axis, beta blockers, cardiac resynchronization therapy (CRT) and left ventricular (LV) assist devices (LVAD). We report prospective outcome data from a cohort of patients with DCM treated according to current guidelines.

Methods: We included 102 patients with idiopathic DCM and LV ejection fraction (LVEF) <40 %. Baseline (BL) work-up included echocardiography, measurement of peak oxygen consumption (peak VO2) and right-sided heart catheterisation. Follow-up (FU) was performed after 12 months. Predictors of the absolute change in LVEF (delta EF) were analysed by regression analysis.

Results: At FU, two patients were transplanted and one was on an LVAD. Four patients did not show. In the others, LVEF had increased by 13 ± 13 percentage points (p<0.001). BL data and predictors of delta EF are presented in the Table. LV function improved more in patients with severely reduced LVEF at BL. Parameters associated with LVEF, such as LV filling pressure and biomarkers, were likewise univariate predictors of delta EF, but not in multivariate analysis. Apart from LVEF, only symptom duration at BL was independently associated with delta EF.

Conclusion: Most patients with DCM experience a substantial increase in LVEF over 12 months. Patients with a severely reduced LVEF at presentation have a greater potential for LV recovery. A short duration of symptoms is an independent predictor of improvement in LVEF.

See table.

P880 Assessment of regional cardiac function might improve discrimination between left ventricular non-compaction and hypertrophic cardiomyopathies

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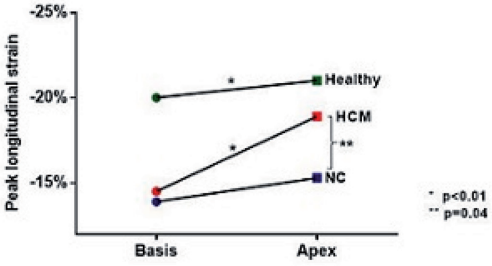
Purpose: Modern imaging technology has resulted in an improved detection of left ventricle non-compaction cardiomyopathy (LVNC). Hypertrophic cardiomyopathy (HCM) shares several morphological features with LVNC. However, prognosis and treatment strategy in LVNC is very different from HCM. We aimed to evaluate if regional LV myocardial function in these diseases may help discrimination between LVNC and HCM.

Methods: We studied 15 patients with LVNC (age 50 ± 15), 25 patients with HCM (age 44 ± 15) and 25 healthy controls (age 40 ± 13), diagnosed according to current guidelines. Global longitudinal strain (GLS) by echocardiography was calculated from a 16 LV segments model with speckle tracking technique. LV basal (6 segments) and apical (4 segments) longitudinal strains were averaged.

Results: Patients with LVNC and HCM had reduced LV function compared to healthy by EF (37 ± 12 % vs. 55 ± 5 % vs. 61 ± 5 %, p<0.001) and by GLS (-11.5 ± 10 % vs. -16.3 ± 3.1 % vs.-21.2 ± 1.8, p<0.001). LVNC had reduced function compared with HCM (EF: p<0.001 and GLS, p=0.04). LV apical strain was worse in LVNC compared to HCM (-15.3 ± 6.7 % vs.-18.9 ± 4.4 %, p=0.04), while LV basal strain did not differ (-13.9 ± 5.7 % vs.-14.5 ± 3.9 %, p=0.69). Function increased from base to apex in HCM (-14.5 ± 3.8 vs. -18.9 ± 4.4 %, p<0.001) and in healthy controls (-20.2 ± 1.6 % vs.-21.6 ± 2.7 %, p=0.008), as opposed to a more homogeneously decreased function in LVNC (-13.9 ± 5.7 %, vs.-15.3 ± 6.7 %, p=0.34) (Figure).



Differences in regional longitudinal strain in apex vs. basis in left ventricle between groups

Conclusion: This study demonstrated a basal to apical gradient with relatively preserved apical function in HCM, while function was homogenously reduced in LVNC. These characteristics may help to discriminate between these two cardiomyopathies.

P6522 Cardiac biomarker ST2 reflects haemodynamic stress in dilated cardiomyopathy: results from a prospective cohort study

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Purpose: The cardiac biomarker soluble ST2 (sST2) is associated with adverse outcome in heart failure (HF). In experimental studies, sST2 expression is induced by myocardial stress and pro-inflammatory stimuli. Determinants of sST2 levels in HF patients have not been well described. We assessed the associations between sST2 levels and haemodynamic parameters reflecting right and left ventricular pre- and afterload in patients with dilated cardiomyopathy (DCM).

Methods: We prospectively recruited 102 patients with left ventricular ejection fraction (LVEF) <40 % and a diagnosis of idiopathic DCM based on patient history, echocardiography and coronary angiography. Work-up included right sided heart catheterisation. Subsequently, heart transplantation and death were recorded. Soluble ST2 was measured by a highly sensitive immunoassay. Determinants of sST2 were assessed by linear regression analyses.

Results: Population characteristics and their association with sST2 in uni- and multivariate analyses are presented in the Table. Levels of sST2 were higher in patients with severe symptoms (NYHA III-IV) even after adjustment for LVEF (p=0.02). In multiple regression, only gender, heart rate and right atrial pressure remained independent predictors of sST2. After a median of 3.6 years, 12 patients were dead or heart transplanted. Baseline sST2 was higher in these patients than in survivors (p=0.05).

Conclusion: In DCM, sST2 is independently associated with an elevated heart rate and venous congestion. Our results imply that sST2 reflects haemodynamic decompensation in DCM.

See table

P842 The effect of exercise on left ventricular twist in patients with type 2 diabetes and diastolic dysfunction

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Purpose: Type 2 diabetes mellitus (T2DM) is associated with diastolic dysfunction (DD). Left ventricular (LV) twist and untwist are important for normal LV function, and have been found to be altered in patients with diastolic dysfunction. We sought to find out how the effect of high intensity interval exercise (HIIE) alter LV twist parameters in patients with T2DM (duration <10 years) and DD, compared to moderate intensity exercise (MIE), in accordance to International Diabetes Federation recommendations.

Methods: A total of 36 patients (mean age 57 years, 22 male) with T2DM and DD (early diastolic tissue velocity (e') <8 cm/s) were randomized to either HIIE (4x4minutes) at 90-95 % of maximal heart rate 3 times/week (n=19) or MIE for 210 minutes/week (n=17). LV twist and untwist were measured by two-dimensional speckle tracking echocardiography pre and post the 12-week period of exercise.

Results: Diastolic function improved significantly in both groups after 12 weeks of exercise (HIIE mean difference e' 1.8 ± 1.1, P<0.001 versus MIE 0.5 ± 0.7 cm/s, P=0.017). The amplitude of twist, twist rate and untwist rate decreased, but the time to peak untwist rate from aortic valve closure was shorter after exercise (Table).

Conclusions: In T2D patients with diastolic dysfunction, exercise shortened the time to peak

Table 1. Predictors of sST2

Variable	Baseline value	Univariate r	p-value	Multivariate β	p-value
Age (years)	51 ± 14	−0.19	0.05	−0.44	0.66
Gender (% male)	73		0.003	2.39	0.02
Systolic BP (mmHg)	116 ± 20	−0.28	0.004	−0.40	0.69
Heart rate (bpm)	75 ± 16	0.55	<0.001	3.61	0.001
Right atrial pressure (mmHg)	7 ± 5	0.44	<0.001	2.34	0.02
Mean pulmonary artery pressure (mmHg)	24 ± 10	0.33	0.001	−0.92	0.36
Pulmonary capillary wedge pressure (mmHg)	15 ± 9	0.36	<0.001	0.32	0.75
Cardiac output (l/min)	4.9 ± 1.5	−0.35	<0.001	−1.10	0.27
Left ventricular ejection fraction (%)	26 ± 10	−0.43	<0.001	−0.37	0.71
NYHA class III-IV (%)	25		<0.001	1.07	0.29

Table: Left ventricular twist parameters

Parameters	Moderate intensity exercise			High intensity interval exercise		
	Baseline	Post 12 weeks	P	Baseline	Post 12 weeks	P
Peak twist °	13.3 (4.6)	9.3 (5.9)	0.024	12.5 (3.6)	9.4 (3.9)	0.010
Peak twist rate, °/s	95.8 (29.8)	67.9 (26.8)	0.033	78,7 (27.3)	58.1 (21.3)	0.019
Peak untwist rate, °/s	−98.9 (37.0)	−69.7 (17.2)	0.008	−87.0 (28.7)	−67.3 (37.2)	0.046
Time to peak early apical untwist rate, % of diastole	18.2 (9.9)	12.4 (6.4)	0.044	21.3 (9.8)	12.4 (8.7)	0.001
Time to peak early basal untwist rate, % of diastole	21.9 (17.3)	13.6 (9.3)	0.046	18.6 (9.1)	9.1 (5.6)	0.02
Time to peak early untwist rate, % of diastole	24.3 (14.4)	13.1 (13.4)	0.081	22.0 (18.0)	9.9 (11.9)	0.003

Values are expressed as mean (standard deviation), % of diastole – the total length of the diastolic period was set to 100 % and the time to peak early untwist rate was definded as the time from AVC to peak untwist rate divided by the diastolic period.

untwist rate as well as improved the diastolic dysfunction, especially in the high intensity exercise group.

See table

P3431 The presence of toll-like receptor 9 protects cardiac function in a diastolic heart failure model

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Purpose: Immune activation is important in the pathogenesis of heart failure (HF). Toll-like receptor 9 (TLR9) activity influences the pathogenesis of various cardiovascular disorders. Studies on TLR9 in HF have employed experimental systolic HF models but the consequence of TLR9 in diastolic HF is unknown. We investigated the role of TLR9 in a murine diastolic HF model caused by cardiomyocyte SERCA2a deletion.

Methods: We engaged in a 3-generation breeding strategy using αMHC-MerCreMer Serca2a flox/flox mice crossed with TLR9−/− mice to generate comparable mouse lines. Diastolic HF was induced both spatially and temporally (Tamoxiphen induced gene-recombination at 8-10 weeks) by KO of cardiac myocyte SERCA2a. Two substudies were undertaken; 1) a 12 week survival study registering death or signs of severe morbidity (leading to euthanasia) and 2) a study with MRI and echocardiography at baseline, 3 weeks and 6 weeks. Finally, tissue and blood was harvested.

Results: All mice depleted of SERCA2a, but none with the SERCA2a gene intact, reached our pre-specified end-parameter within 73 days. The lack of TLR9 in this diastolic HF model led to significant reduction in survival with a median life expectancy of 62.5 days as compared to 58 days (p=0.007). Serial imaging demonstrated an earlier onset of left ventricular restrictive filling abnormalities in the HF group depleted of TLR9. Significantly lower EF and CO, larger left atria, lower end-systolic and end-diastolic left ventricular volumes were detected.

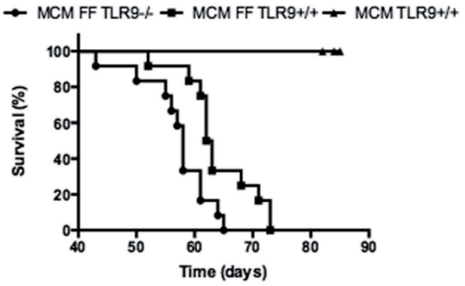

Figure 1. Survival in days after HF induction.

Conclusion: The lack of TLR9 aggravates the development of diastolic HF induced by SERCA2a KO. These findings may add to the understanding of molecular mechanisms governing the progression of diastolic HF.

P417 Diastolic function during exercise: effects of a 12 week high-intensity exercise program in patients with a recent myocardial infarction

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Purpose: Reduced left ventricular (LV) diastolic function is a negative prognostic marker after acute myocardial infarction. High-intensity interval training can improve functional capacity in myocardial infarction patients, but it is not known whether it can improve diastolic function. Our aim was to study whether a twelve week high-intensity interval training program could improve diastolic function in patients with a relatively recent myocardial infarction.

Methods: Thirty patients (27 males, 3 females, mean age = 56 (8) years, mean time from infarction = 78 (45) days) performed high-intensity interval training twice a week for 12 weeks. Each training session consisted of four 4-minute intervals at 85-95 % of peak heart rate, separated by 4-minute active breaks at 70 %. A cardiopulmonary exercise test was performed to determine peak oxygen uptake (VO2peak). Echocardiography, including color tissue Doppler of the LV, was performed during supine rest and during an upright bicycle exercise test (peak load 75 Watt).

Results: There was a significant increase in VO2peak from baseline to follow-up (35 (7) vs. 39 (7) ml/kg/min), p<0.001). There was a significant correlation between VO2peak and mitral annular early diastolic velocity (e') at peak exercise, and the relationship was present both at baseline and follow-up (r=0.48, p=0.007, and r=0.41, p=0.03). There was a trend towards an increase of e' at 75 Watt from baseline to follow-up (8.1 (1.6) vs. 8.5 (1.7) cm/s, p=0.06), but no change in e' at rest (7.2 (1.9) vs. 7.3 (1.6) cm/s, p=0.43). There was no change in E/e' at rest (10 (2) vs. 10 (2), p=0.84), E/e' at 75 Watt (11 (3) vs. 11 (3), p=0.70) or E/A ratio at rest (1.2 (0.3) vs. 1.1 (0.3), p=0.41). There were no changes in variables describing left ventricular systolic function: mitral annular systolic velocity (s') at rest (6.0 (1.0) vs. 6.0 (0.9) cm/s, p=0.93), s' at 75 Watt (7.9 (1.1) vs. 8.0 (1.3) cm/s, p=0.64), or mitral annular plane systolic excursion at rest (13 (3) vs. 13 (2) mm, p=0.38). Heart rate was unchanged at rest (59 (7) vs. 57 (8), p=0.13) and at 75 Watt (94 (13) vs. 91 (11), p=0.12).

Conclusion: The present study shows that left ventricular diastolic function during exercise is related to VO2peak. Interestingly, we found a trend towards an improved diastolic function after exercise training, even in a population with a relatively well preserved systolic and diastolic function. The results demonstrate the importance of obtaining measurements during exercise when evaluating the effects of an exercise training intervention.

P822 No effect of levosimendan on inflammation in ST-elevation myocardial infarction complicated by acute heart failure: a substudy of the LEAF-trial

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Treatment with levosimendan in patients with decompensated chronic heart failure (HF) has been shown to reduce levels of pro-inflammatory cytokines suggesting possible anti-inflammatory effects. Such potentially beneficial effects of levosimendan have so far not been studied in patients with de novo HF complicating acute ST-elevation myocardial infarction (STEMI).

A total of 61 patients developing clinical signs of HF within 48 hours after a primary PCI-treated STEMI (including cardiogenic shock), were randomised double-blind to a 25 hours infusion of levosimendan or placebo in the LEvosimendan in Acute heart Failure following myocardial infarction (LEAF) trial. The pre-specified endpoints in this substudy were change in levels of pro-inflammatory cytokines and adhesion molecules from baseline to day 1, day 5 and 6 weeks.

The two treatment groups (levosimendan and placebo) were not significantly different with respect to baseline characteristics (Table 1). Patients were characterised by large STEMI and severe HF at inclusion, all except one with new onset HF. There were no statistically significant between groups differences in changes from baseline of IL-6, CRP, sIL-6R, sgp130, MCP-1, IL-8, MMP-9, sICAM-1, sVCAM-1 and TNF-a at the pre-specified endpoints.

In patients with acute HF complicating a primary PCI-treated STEMI, levosimendan treatment did not affect levels of inflammatory markers during the first 6 weeks as compared to placebo. The results differ from previous studies on patients with decompensated HF and may reflect that the inflammatory response pattern in patients with

Table 1. Baseline characteristics

	Levosimendan (n=30)	Placebo (n=31)
Age	66 (56-74)	62 (56-74)
Cardiogenic shock, n (%)	4 (13)	5 (16)
IABP at baseline, n (%)	8 (27)	9 (29)
Hours from start of symptoms to PCI	3 (2-8)	3 (2-6)
Hours from PCI to to study infusion	24 (14-33)	22 (14-26)
TnT peak, ng/L	12195 (7990-16187)	11828 (5670-18640)
NT-proBNP (pmol/L)	386 (297-597)	474 (248-922)
LVEF at baseline, %	43 (38-49)	40 (33-47)

Data are presented as median (IQR). IABP, intra-aortic counter-pulsation.

de novo HF and acute STEMI are mostly influenced by myocardial injury.

See table

P517 Right ventricular function in adult survivors of childhood cancer

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Purpose: Right ventricular (RV) function is strongly associated with prognosis in several cardiac conditions, but data on RV function in childhood cancer survivors (CCS) are very limited.

Methods: Echocardiograms were obtained by a high-end digital scanner (Vivid 7 or E9, GE). RV function was evaluated measuring tricuspid lateral annular systolic velocity (s') using pulsed tissue Doppler. Logistic regression analyses with RV systolic dysfunction (s' <10 cm/s) as dependent variable, and gender, age at exam, cancer diagnosis, previous treatment exposure (anthracyclines, cyclophosphamide, iv methotrexate, mediastinal radiotherapy, spinal radiotherapy), left ventricular (LV) function and valvular function as covariates, were performed. CCS were compared 1:1 to healthy, matched controls.

Results: In this cross-sectional study, 235 CCS were examined mean 22 ± 8 years after diagnosis, which was Hodgkin's lymphoma in 56, non-Hodgkin lymphoma in 43 and acute lymphoblastic leukemia in 136 of the survivors. Mean age at exam was 31 ± 8 years. CCS had lower s' than controls (12.2 ± 2.0 vs. 13.1 ± 1.8 cm/s, p<0.001). RV systolic dysfunction was found in 8 % of CCS and 2 % of controls (p=0.004). In multivariate analysis, RV systolic dysfunction was correlated with LV systolic dysfunction (odds ratio (OR) 3.3, 95 % confidence interval (CI) 1.1-10.6, p=0.041), and trended to be correlated with LV diastolic dysfunction (OR 3.0, 95 % CI 1.0-9.6, p=0.064). Neither age, gender, diagnosis nor previous treatment exposure were correlated with RV systolic dysfunction.

Conclusion: Long-term CCS have impaired RV systolic function compared to healthy controls. Exploratory analyses indicate an association between RV and LV dysfunction, but not between RV dysfunction and previous anti-cancer treatment. Prospective studies are needed to determine the prognostic value of RV dysfunction in CCS.

P497 Static lung hyperinflation and increase of right ventricular volume reduce LV mass and volume in patients with COPD

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Aim: We and others have demonstrated impaired LV function in COPD. In the present study we aimed to elucidate the impact of lung hyperinflation and the size of right ventricle (RV) on the left ventricle (LV) in patients with stable COPD, where LV cardiovascular disease was thoroughly excluded in advance.

Methods: 112 outpatients with stable COPD in GOLD stages I-IV and 34 controls were included. The COPD patients were divided in two groups by mean pulmonary artery pressure ≥ or <25 mmHg; those with pulmonary hypertension (COPD-PH) and those without (COPD-non-PH). RV and LV end-diastolic volumes were measured by magnetic resonance and 3D echo imaging. To evaluate the importance of hyperinflation, residual volume (RV % predicted) was measured by body plethysmography.

Results: There was a marked and significant increase in RV end-diastolic volume from the controls, 57 ± 7, to COPD-non-PH, 71 ± 15 and to COPD-PH, 74 ± 15 ml/m2 (p<0.01 for all), but no change in LV volumes. The end-diastolic LV/RV volume ratio decreased significantly (p<0.01 for all) from the controls, 1.03, to COPD-non-PH, 0.88, and to COPD-PH, 0.78. Linear regression showed that one standard deviation (1-SD) increase in RV % predicted (62 %) was significantly associated with a 5.5 g decline in LV mass, (95 % CI -8.5, -2.4, p<0.001). In the same way, an increase of 1-SD of RV volume (1.35 l) was related to a decline in LV mass of 5.4 g. Both regression analyses were adjusted for hypertension, diabetes, total cholesterol level and pack years of smoking. Body size and age, gender and height were adjusted for by means of BSA for LV mass and by use of predicted values of RV (accounting for height, age and gender). Systolic blood pressure was not associated with LV mass change.

Conclusion: The present study has demonstrated that both static lung hyperinflation and RV volume have a negative impact on LV mass and structure in stable COPD patients, where LV cardiovascular disease was excluded. These findings might be mechanisms for the observed subclinical impairment of LV function in these patients.

P4188A delayed increase in high sensitive Troponin I following high-intensity endurance cycling competition may have a potential role in the detection of unrecognized coronary artery disease

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Purpose: An increase in circulating cardiac Troponin levels may be observed following prolonged intense physical exercise. The precise cause and clinical significance of this troponin increase is unknown. The objective of this study was to describe the pattern of high-sensitive cardiac Troponin I (hs-cTnI) release following long-term cycling competition in presumably healthy amateur athletes.

Methods: Leisure sport cyclists without known coronary artery disease or cardiovascular medical treatment completing the 91 km mountain bike race were included in the study. Blood samples and rest ECG were aquired at 4 timepoints: 24 hours prior to the race, and at 0, 3 and 24 hours following the race.

Results: A total of 97 cyclists, 74 (76 %) males, mean age 43 (36-49) years, completing the race in 4:22 ± 0:52 (h:min) with a mean heart rate of 156 ± 16.7 bpm were included. Mean hs-cTnI value prior to the race was 4.0 ± 3.7 ng/l (upper limit of normal: 30.0 ng/l).No patient had rest ECG or symptoms suggestive of coronary artery disease (CAD) during the race or for the first 24 hours following the race. Following the race, there was an increase in hs-cTnI in all participants that competed in the race. The mean hs-cTnI value immediately following the race was 61.4 ± 54.8 ng/l, peaking at 3 hours to 90.8 ± 113.9 ng/l, declining at 24 hours to 46.9 ± 215.2 ng/l. All values were highly significantly different from baseline (p<0.0001). In 3 out of 4 individuals with the highest hs-cTnI levels, significant CAD was detected by CT coronary angiography. Revascularization was performed in two of these. No CAD was detected in individuals with a max hs-cTnI level <370 ng/l.

Conclusions: Hs-cTnI levels increased in all participants following the competition. Highly elevated hs-cTnI following the competition identified several cyclists with previously unrecognized significant CAD. These findings may suggest a potential role for hs-cTnI in the detection and monitoring of CAD among persons participating in prolonged high intensity endurance activity.

P3667 Heart rate as an independent predictor of mortality in patients with heart failure: results from the Norwegian Heart Failure registry

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Purpose: To evaluate whether heart rate is an independent predictor of mortality in patients with heart failure (HF).

Methods: Hospital outpatients with HF were enrolled at stable follow-up with a measurement of heart rate and extensive demographic data, and followed for all cause mortality with complete update for all patients.

Results: There were 4890 patients (Sinus rhythm 2974), with mean age 70.4 ± 11.8 years, 28.5 % women, included from 2000 to 2012 in the analyses. The median follow-up was 58 months in survivors. Adherence to evidence-based medication was high: >90 % was prescribed a beta blocker and an ACE-I/ARB and 27 % an aldosterone blocker. In multivariate linear regression heart rate was related to type of rhythm, age, daily diuretic dose, gender, presence COLD/asthma, beta blocker use, NYHA class, coronary artery disease as the main cause for HF, diabetes mellitus, and use of ACE-I/ARB. In univariate Cox regression analyses of time to mortality heart rate was not a predictor (HR 1.004; 95 % CI 1.000-1.008; P=0.069). However, in multivariate Cox regression analyses heart rate was a strong predictor of mortality (HR 1.006, 95 % CI 1.001-1.010; P=0.008). This was adjusted for age, beta blocker use, NYHA class, daily dose diuretic, diabetes mellitus, gender, coronary artery disease as the main reason for HF, ACE-I/

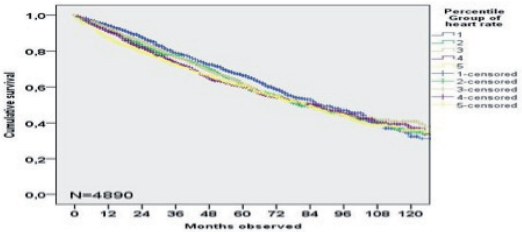

Figure: Kaplan-Meier curve of quintiles of HR.

ARB use. Similarly for patients with sinus rhythm this was also significant (HR 1.009, 95 % CI 1.003-1.014; P=0.004).). This result was adjusted for age, beta blocker use, NYHA class, daily dose diuretic, diabetes mellitus and gender.

Conclusion: In this cohort of patients receiving optimal medical treatment at specialized outpatient HF clinics, an increased heart rate was as strong independent predictor of an increased risk of death independent of the type of rhythm.

P3459 Automatic measurement of aortic annulus diameter in 3-dimensional transoesophageal echocardiography

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Purpose: Transcatheter aortic valve implantation involves percutaneously implanting a biomechanical Aortic Valve (AV) to treat severe aortic stenosis. In order to select a proper device, precise sizing of the AV annulus is critical.

Methods: We developed a novel and fully automatic segmentation method to measure the AV annulus geometry, operating on 3-dimensional transesophageal echocardiographic (TEE) acquisitions from mid-esophagus with the transducer array aligned with the long axis of the Left Ventricle (LV).

The novel segmentation algorithm can be summarized by the following steps: The LV Outflow Tract (LVOT) long axis and AV hinge-point plane are automatically estimated. The LVOT, AV and Aortic Root (AR) are then tracked over the cardiac cycle by combining edge detection with a compact geometric representation in a Kalman filter.

The method was validated on 3D TEE recordings of 16 patients with varying severity of aortic disease. Measurements of the AV annulus were done by two independent experienced echocardiologists for comparison. The annulus was traced in mid systole in a plane intersecting the lowest insertion point of all aortic cusps.

Results: The automatic method measured the AV annulus with mean computation time 9.9 s. Comparison against two manual observers showed agreements (mean ± SD) of -0.35 ± 1.6 mm (r=0.87) and -0.23 ± 2.3 mm (r=0.74) for perimeter-derived diameters and 0.40 ± 1.6 mm (r=0.86) and 0.46 ± 2.3 mm (r=0.74) for area-derived diameters. The corresponding interobserver agreements were -0.12 ± 2.1 mm (r=0.77) and -0.16 ± 2.1 mm (r=0.76).

Conclusions: We demonstrated the feasibility of an efficient and fully automatic measurement of the AV annulus in patients with AV disease. The algorithm provided robust measurements indistinguishable from those done by cardiologists.

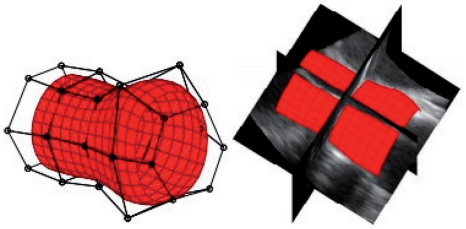

Figure: Geometric representation of LVOT and AR.

P3596 Higher levels of cardiac troponin t in patients with stable angina pectoris predict increased risk of myocardial infarction

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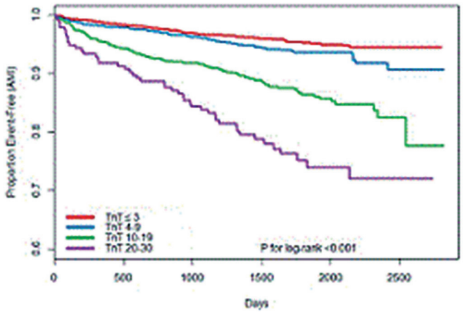
Purpose: Previous studies among patients with stable angina pectoris (SAP) have shown that cardiac troponin T (hs-cTnT) concentrations as determined by highly sensitive assays are significantly associated with risk of cardiovascular death, but the association with acute myocardial infarction (AMI) is less clear. We assessed the relationship between baseline hs-cTnT levels and risk of AMI in SAP patients.

Methods: 3882 patients who underwent elective coronary angiography 2000 – 2004 were followed to subsequent AMI or end of 2006. Univariate and multivariate survival analyses according to hs-cTnT groups (≤3 ng/L; n=1796, 4-9; n=1199, 10-19; n=689 and 20-30; n=198) were studied by Kaplan Meier plots and by Cox regression.

Results: The population consisted of 2773 (71.4 %) males with a median age of 61.7 years, of which 286 (7.4 %) experienced an AMI. Kaplan Meier plots revealed a strong, graded association between hs-cTnT categories and risk of AMI (Figure 1). In a Cox model adjusted for age, sex, body mass index, hypertension, diabetes mellitus, smoking, Apo A1, Apo B, Lp(a) and CRP, hazard ratios (HRs) (95 % confidence intervals [CIs]) were 1.05 (0.75 – 1.46), 1.94 (1.38 – 2.73) and 3.25 (2.13 - 4.95) when comparing the 2nd, 3rd and 4th to the 1st Hs-cTnT group, respectively (P for trend <0.001). The linear association remained significant (P for trend <0.001) even

after adjusting for the number of significantly stenosed coronary arteries (0-3), left ventricular ejection fraction (%), estimated glomerular filtration rate (mL/min/1.73m2), medication and previous peripheral vascular disease, percutaneous intervention or coronary bypass surgery.



Conclusion: In patients with SAP, higher levels of hs-cTnT are associated with an increased risk of subsequent AMI.

P4517 Coronary atheroma regression and plaque characteristics after aerobic exercise assessed by intravascular ultrasound: a randomized controlled trial

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Purpose: To investigate effects of high intensity aerobic interval training (AIT) versus moderate continuous training (MCT) on coronary atherosclerosis in patients with significant coronary artery disease on optimal medical treatment.

Methods: Thirty-six patients were randomized to AIT (intervals at ≈90 % of peak heart rate) or MCT (continuous exercise at ≈ 70 % of peak heart rate) 3 times a week for 12 weeks following intracoronary stent implantation. Grayscale and radiofrequency intracoronary ultrasound (IVUS) was performed at baseline and follow-up. The primary endpoint was changes in plaque burden and plaque composition. Separate lesions were classified in terms of plaque vulnerability using radiofrequency IVUS criteria. IVUS recordings were analysed off line in an independent CoreLab.

Results: Necrotic core was reduced in both groups in defined coronary segments (AIT -3.2 %, MCT -2.7 %, both p<0.05) and in separate lesions (median change -2.3 % and -0.15 mm3, p<0.05). Plaque burden was reduced by 10.7 % in separate lesions independent of intervention group (p=0.06). No significant differences in IVUS parameters were found between exercise groups. A minority of separate lesions were transformed in terms of plaque vulnerability during follow-up with large individual differences between and within patients. Figure 1 illustrates the transformation from a thin-cap fibroatheroma to intimal medial thickening in a patient undergoing AIT.

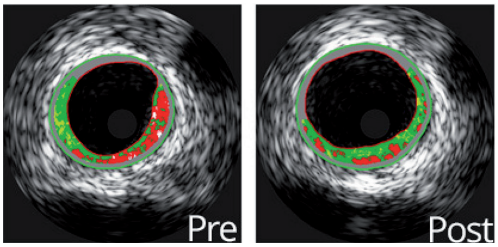

Figure: RF-IVUS images pre and post exercise.

Conclusions: Changes in coronary artery plaque structure or morphology did not differ between patients undergoing AIT or MCT. The combination of regular aerobic exercise and optimal medical treatment for 12 weeks induced a moderate regression of necrotic core and plaque burden in IVUS-defined coronary lesions.

P6152 Soluble ST2 is associated with reduced myocardial function and arrhythmic events in patients with arrhythmogenic right ventricular cardiomyopathy (ARVC)

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Purpose: Risk stratification for ventricular arrhythmias in arrhythmogenic right ventricular cardiomyopathy (ARVC) remains challenging. Soluble ST2 (sST2), a member of the interleukin 1 cytokine family, can be induced in mechanically overloaded cardiomyocytes and is elevated in patients with left ventricular (LV) heart failure. In ARVC patients, right ventricular (RV) function is predominantly affected. We wanted to explore if the plasma concentration of sST2 was associated with reduced myocardial function and arrhythmic events in patients with ARVC.

Methods: We included patients with ARVC and their mutation positive family members. sST2 was determined by ELISA in plasma collected at time of echocardiographic examination. Myocardial function was assessed by echocardiography including strain by speckle tracking technique. RV function was assessed by RV fractional area change (FAC) and by RV global strain (average longitudinal strain from 6 RV segments). LV function was assessed by ejection fraction (LVEF) and LV global strain (average longitudinal strain in 16 LV segments).

Results: We included 46 ARVC mutation positive subjects (age 41 ± 15 years, 21 female), of whom 22 had previous ventricular arrhythmia and 24 had no arrhythmic events. sST2 was elevated in those with arrhythmias compared to those without (34 ± 13 ng/mL vs. 26 ± 7 ng/mL, p=0.009). sST2 correlated with RV function by RV global strain (R=0.51, p=0.001) and RVFAC (R=0.36, p=0.02) and with LV function by LVEF (R=-0.43, p=0.003) and LV global strain (R=0.48, p=0.001). ROC analyses for sST2 showed C-statistics of 0.70; 95 % CI 0.55-0.86. A sST2 level of 30 ng/mL identified ARVC patients with ventricular arrhythmias with a sensitivity of 50 % and a specificity of 88 %.

Conclusions: Soluble ST2 was elevated in ARVC patients with arrhythmic events and correlated well with RV and LV function. sST2 may be of additive value in risk stratification for ventricular arrhythmias in ARVC.

P4300 Plasma YKL-40 levels are elevated and predict mortality in patients with aortic stenosis

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Purpose: Valve calcification and inflammation play key roles in the development of aortic stenosis (AS). A microarray study recently demonstrated that YKL-40, mainly produced by macrophages, neutrophils and vascular smooth muscle cells, was one of the most up-regulated mRNA transcripts in aortic calcified vs. non-calcified valves. We hypothesized that circulating levels of YKL-40 would be upregulated and could predict all-cause mortality in patients with severe symptomatic AS.

Methods: We measured plasma levels of YKL-40 in 136 patients with symptomatic severe AS and 46 healthy controls and its relation with transvalvular gradients, valve area, valve calcification (as estimated by ultrasound backscatter) and indices of heart failure as assessed by echocardiography and its relations to all-cause mortality (n=35) during long-term follow-up (median 4.6 years).

Results: Plasma YKL-40 levels were markedly increased in patients with AS (median [25th, 75th percentile]: 34 ng/ml [21,61] vs. 8 ng/ml [6,11], p=0.003). The strongest determinants of plasma YKL-40 in symptomatic AS were CRP (Beta=0.46, p<0.001), age (Beta=0.34, p<0.001) and ultrasound back scatter (Beta=0.28, p=0.002). Univariate analysis on quartiles of YKL-40 divided into quartiles demonstrated a non-linear association with all-cause mortality with a particularly enhanced risk in quartile 4 (HR 5.09 [95 %CI: 1.88-13.73] p=0.001) compared to quartile 1. Increased YKL-40 (i.e. quartile 4) was associated with all-cause mortality also after adjustment for conventional risk factors (i.e. age, type 2 diabetes, eGFR, LVEF, valve area, atrial fibrillation, troponin T and NT-proBNP) (HR 2.48 [1.15-5.34] p=0.020).

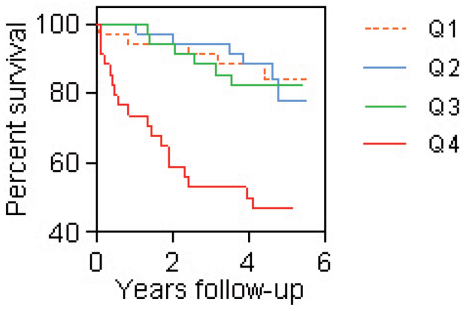

Figure: Kaplan-Meier.

Conclusion: Circulating YKL-40 is increased in severe symptomatic AS and enhanced levels are associated with decreased long-term survival.

P574 Cardiac resynchronization therapy in left bundle branch block improves right ventricular function

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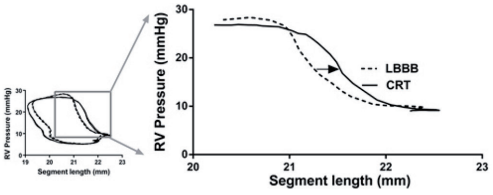
Purpose: Right ventricular (RV) function has been recognized as a predictor of clinical response to cardiac resynchronization therapy (CRT) during left bundle branch block (LBBB). In

an experimental setting, we aimed to study the impact of CRT on RV function during LBBB.

Methods: In 6 anaesthetised dogs with LBBB induced by radio frequency ablation, we applied CRT with one electrode on the right side of the interventricular septum and one epicardially on the LV lateral wall. RV pressure was measured by a micromanometer in the RV cavity and segmental length (SL) by sonomicrometry in the RV free wall. The area of the RV pressure-SL loop was used as an index of regional work in the RV free wall. Pre-ejection RV shortening, measured at 50 % increase of RV pressure, was calculated in percentage of peak systolic shortening.

Results: Induction of LBBB was associated with a reduction in RV free wall work from 41 ± 13 to 29 ± 16 mmHg*mm (P<0.05). This was in part due to distortion of the pressure-SL loop with marked pre-ejection shortening (33 ± 14 %) of total shortening. CRT increased segmental work to 41 ± 15 mmHg*mm, P<0.05 and RV dP/dt max increased from 361 ± 78 to 446 ± 76mmHg/s (P<0.05). Neither maximum RV pressure (28 ± 3 vs. 27 ± 3mmHg, NS) nor total shortening (8 ± 3 vs. 8 ± 3 %, NS) was changed by CRT. However, the RV pre-ejection shortening decreased substantially to 13 ± 12 % (P<0.05 vs. LBBB) of total shortening (figure).



Figure

Conclusions: During LBBB there is ineffective contraction in the RV free wall as approximately 1/3 of the contraction occurs during low pressure prior to ejection. The efficiency was improved by CRT, which markedly increased regional work in the RV free wall. The findings suggest that improvement in RV function may be important for success of CRT in LBBB.

P4572 Effect of ischemic postconditioning on myocardial strain measured by two-dimensional speckle tracking in primary PCI-treated STEMI patients

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Purpose: Effect of ischemic postconditioning (IPost) on reperfusion injury has been evaluated both in experimental and clinical trials in patients with acute ST elevation myocardial infarction (STEMI). We have recently reported no significant effect of IPost on infarct size measured after 4 months in the randomized POSTEMI trial. The aim of this study was to study the effect of IPost on reperfusion injury and early myocardial recovery measured by myocardial strain.

Methods: Patients with first-time STEMI, symptom duration <6 h, TIMI flow 0-1 in the infarct related artery (IRA) and successful opening (TIMI 2-3) were included. Patients were randomized to either IPost or control and treated by primary PCI. IPost was performed by 4 cycles of 1 min balloon occlusion of IRA, starting 1 min after opening and separated by 1 min reperfusion intervals Global longitudinal peak systolic strain (εSYS, peak negative strain in systole) and peak strain (εPEAK, peak negative strain in diastole) were measured by two-dimensional speckle tracking echocardiography at a median of 2.4 (range 1-5) days after PCI. Post-systolic index (PSI) as a measure of postsystolic shortening, was calculated manually, PSI = (εPEAK - εSYS)/ εPEAK x 100.

Results: A total of 100 patients, median age 61 (range 38 – 87) years, 85 % males and 56 % with anterior wall infarction were included in the strain analysis, 45 in the IPost and 55 in the control group. Peak systolic strain was reduced to – 13.1 (IQR -16.3, - 11.2). Postsystolic shortening was present in 98 out of 100 patients. No significant between-group differences were found in myocardial strain or post-systolic shortening (measured as εSYS, εPEAK, or PSI) in the IPost group compared to control.

Table: Relationship between myocardial strain measurements and IPost

Strain measurements	IPost group (n=45)	Control group (n=55)	p-value
Peak-systolic strain (εSYS)	−12.9 (−16.5; −11.0)	−13.1 (−16.0; −11.3)	0.80
Peak-strain (εPEAK)	−15.1 (−18.0; −12.8)	−15.2 (−17.0; −13.5)	0.86
Post-systolic index (PSI)	9.1 (5.8; 14.9)	10.0 (4.7; 18.5)	0.83

Data are median values (25 and 75 percentiles).

Conclusion: Ischemic postconditioning did not influence early myocardial recovery, measured as peak systolic strain or postsystolic shortening.

P4642 Feasibility and accuracy of nurse performed pocket-size ultrasound imaging of the pleura and vena cava inferior to assess volume state in patients with heart failure in an outpatient clinic

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Purpose: Pleural effusion in heart failure (HF) patients correlate well with decompensation and assessment of the dimension and collapsibility of the inferior vena cava (IVC) may help to identify both decompensation as well as dehydration. Follow-up in multidisciplinary HF clinics are shown to improve patient treatment. As this may improve treatment of HF we aimed to study the feasibility and accuracy of routinely nurse performed pocket-size ultrasound imaging of the pleura and vena cava inferior after specific training.

Methods: Patients in an outpatient HF clinic at a non-university hospital were included. Before the study start two specialized nurses underwent a specific training period, with cardiologists as supervisors, with respect to assessment of the pleural cavities and dimension and collapsibility of the IVC. They used a PSID with B-mode and colour flow imaging, and measured dimensions on the PSID. The dimension of the IVC was measured in supine position both end-expiratory and after sniff in sagittal axis. The amount of PLE was measured in sitting position as the dimension of the echo-free space between the diaphragm and basal lung. Reference echocardiography was performed by one of four cardiologists in all patients by high-end echocardiography, including assessment of both pleural cavities.

Results: Mean (SD) age in 62 (48 % women) patients was 74 (12) years, ejection fraction 34 (13) %, NYHA 2.4 (0.6), and N-terminal pro brain natriuretic peptide (proBNP) 3761 (3072) ng/l. Time consumption for PSID examination performed by nurses was median 5 minutes, and reference examination was performed immediatetely. By PSID examination IVC was assessed in all patients, and some amount of pleural effusion was detected in 36 pleural cavities in 23 patients (reference; 39 and 26, respectively). Correlation (95 % CI) for the measurements of pleural effusion, end-expiratory and end-inspiratory IVC was 0.97 (0.91-1.00), 0.89 (0.81-0.95) and 0.79 (0.57-0.93), respectively. Coefficient of variation for end-expiratory IVC was 10 % and 95 % limit of agreement was -6 to 7 mm, respectively. The very few undetected cases (by nurses) with pleural effusions by PSID were in patients were the amount was classified as minor and located only in the costodiaphragmal recess.

Conclusions: Specialized nurses were after dedicated training able to reliably classify volume state by assessing both the pleural cavities and the dimension and collapsibility of the inferior vena cava with an excellent agreement with high-end ultrasound examinations done by cardiologists. This may improve follow-up of heart failure patients.

P2419 High physical fitness; associated with increased or reduced atrial fibrillation risk in middle-aged men?

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Purpose: Smaller studies have demonstrated increased atrial fibrillation (AF) risk among long-endurance trained athletes. However, limited and inconsistent data are available concerning level of fitness and AF risk in the general population. We aimed to investigate associations between measured physical fitness and incident AF in men.

Methods: In 1972-1975, as part of a prospective cardiovascular survey, 1997 healthy middle-aged Norwegian men were tested with a symptom-limited bicycle exercise ECG test. Physical fitness was calculated as total exercise work capacity divided by body weight. Incident AF was documented by scrutiny of medical records in all Norwegian hospitals. Risk estimations were analysed in quartiles of age-adjusted PF using adjusted Cox proportional hazards models.

Results: During 35 years of follow-up, 253 men developed AF (13 %). Men with age-adjusted physical fitness in the upper quartile had significantly reduced long-term AF risk in multivariate analyses compared with the rest of the cohort. Further analyses in deciles and subgroups with baseline elevated blood pressure, showed that the men with the very highest fitness had slightly increased risk compared to men with high (but not extreme) fitness.

Conclusions: High physical fitness in healthy middle-aged men was associated with approximately 25 % lower long-term risk of incident AF. Men with very high fitness, however, seemed to have a slightly increased risk compared to those with more moderate high fitness, especially if their resting blood pressure was elevated.

Table 1. Risk of AF in hazard ratios according to quartiles of age-adjusted physical fitness

Age-adjusted physical fitness	AF events	Hazard ratio*	p-value
Highest quartile (n=510) vs. the remaining quartiles (n=1487)	65 (12.7 %) 188 (12.6 %)	0.74 (0.55–1.00)	0.047

**Multivariable adjusted for age, systolic blood pressure, left ventricular hypertrophy, resting heart rate, height, relative heart volume (by x-ray), body mass index (BMI) and including the significant interaction term BMI × relative heart volume.*

4104 Renal sympathetic denervation is inferior to adjusted drug treatment in patients with true treatment resistant hypertension, a randomized controlled trial

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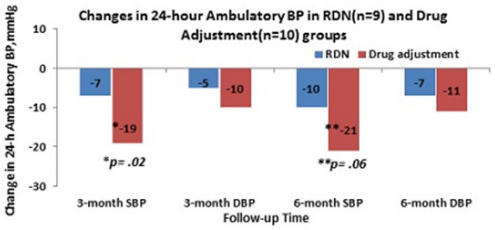
Purpose: Renal sympathetic denervation (RDN) has been introduced as a new treatment of hypertension that is resistant to drug treatment (TRH). However, the randomized and controlled documentation that RDN lowers blood pressure (BP) is limited. We aimed to investigate the BP lowering effect of RDN versus clinically adjusted drug treatment in true TRH after excluding patients with poor drug adherence.

Methods: Patients with apparent TRH (n=65) were referred specifically for RDN and those with secondary and spurious hypertension (n=26) were excluded. TRH was defined as office systolic BP>140 mmHg despite maximally tolerated doses of at least 3 antihypertensive drugs including a diuretic. Additionally, ambulatory daytime systolic BP>135 mmHg following witnessed intake of antihypertensive drugs was required, after which 20 patients had normalized BP, indicating poor drug adherence. Patients with true TRH were randomized and underwent RDN (n=9) versus adjusted drug treatment (n=10).

Results: 24-hour ambulatory systolic and diastolic BPs in the drug adjustment group changed from 151±12/85±6 mmHg (±SD) at baseline to 130±12/74±7 mmHg at 6 months (p=0.001 and p<0.0005, systolic and diastolic BP, respectively), and in the RDN group from 149±9/89±15 to 139±10/82±4 mmHg (p=0.02 and p=0.01, respectively). The absolute reduction in systolic BPs were higher in the drug adjustment group at 3 and 6 months compared to RDN group (p=0.02 and p=0.06, respectively). Pulse pressure, daytime and nighttime ambulatory BPs changed in parallel to the 24-hour ambulatory BPs.



Conclusions: Our data suggest that RDN has inferior lowering effects compared to adjusted drug treatment in patients with true treatment resistant hypertension after excluding patients with confounding poor drug adherence.