# ABSTRACTS PRESENTERT PÅ VÅRMØTET

## P-001. Elevated serum osteoprotegerin levels measured early after acute ST-elevation myocardial infarction predict final infarct size.

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Purpose: Increased serum osteoprotegerin (OPG), a member of the tumor necrosis factor receptor superfamily, has been shown to be associated with increased mortality and heart failure development in patients with acute coronary syndromes and correlated to markers of myocardial necrosis like troponin I. The aim of the present study was to elucidate a possible association between serum OPG measured acutely in patients with ST-elevation myocardial infarction (STEMI) and final infarct size. Methods: Serum OPG was measured in fasting blood samples from 199 patients (median age 58 years, 18 % women) with acute STEMI, within 24 hours after a primary percutaneous coronary intervention (PCI). After 3 months, final infarct size expressed as percent of left ventricular mass was assessed at rest by SPECT imaging. The outcome variable final infarct size was dichotomized using the 75 % percentile as cut off value (large infarct size ≥ 29.0 %). A multivariable logistic regression analysis was performed adjusting for covariates including age, gender, body mass index, smoking, treated hypertension, multivessel disease, serum cholesterol, C reactive protein and creatinine. Continuous variables are presented as median values [interquartile range]. Results: Median OPG concentration was 1.4 [1.0, 2.1] ng mL-1. After adjustment for covariates, baseline OPG (as a continuous variable) was an independent predictor of large infarct size measured at 3 months (OR per 1-SD increase in OPG level 2.5 [1.7,3.6]; p<0.001). Patients with high OPG level (> median) at baseline had larger infarct size at 3 months compared to patients with low OPG levels (< median) (25 [8,40] vs. 6 [0,19] % of left ventricular mass, respectively, p<0.0001). A high OPG level was also associated with a 7 fold increase in the odds of developing a large myocardial infarct (OR 7.0 [95% CI 3.2,15.5]; p<0.001). After adjustment for potential confounders, the adjusted OR was 6.6 [2.9,15.2]; p<0.001. Conclusion: High levels of circulating OPG measured the first morning after a PCI treated acute STEMI were strongly associated with final infarct size.

## P-002. TNF-α antagonists reduce carotid intima media progression rate and improve aortic stiffness in patients with inflammatory arthropathies.

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Introduction: Patients with chronic inflammatory arthropathies such as rheumatoid arthritis (RA), ankylosing spondylitis (AS) and psoriatic arthritis (PsA) have increased cardiovascular morbidity and mortality. Results from observational studies indicate that anti-TNF-α therapy reduces the incident of myocardial infarction in patients with inflammatory arthropathies. Carotid intima media thickness (cIMT) and aortic stiffness are validated cardiovascular risk predictors. Thus, the aim of the current study was to examine the effect of one year treatment with Tumor Necrosis Factor (TNF)-α antagonists on cIMT and aortic stiffness in patients with inflammatory arthropathies.

Methods: Fifty-five patients with active RA, AS or PsA and clinical indication for anti-TNF-α therapy were included. 36 patients started with anti-TNF-α therapy and were compared with a non-treatment group of 19 patients. cIMT was measured at baseline, after 6 and 12 months with the Art.Lab system. Aortic stiffness was assessed as carotid-femoral pulse wave velocity (PWV) with the Sphygmocor device at baseline, after 3, 6, 9 and 12 months. Furthermore, clinical and biochemical inflammatory markers were measured at each visit.

Results: Mean (SD) age in the treatment/control group was 47.2 (12.2) / 51.2.0 (14.1) years (P=0.53) and 42.9 / 50.0 % (P=0.63) were females. Baseline CRP was 12.1 (15.9) mg/L (P=0.58) and the baseline

disease activity score (DAS28) was 3.96 (1.13) / 4.17 (1.23) (P=0.67). After 12 months, cIMT was reduced in the treatment group compared to control group (-5 (45) µm versus 23 (48) µm, respectively; P=0.01), and PWV was improved in the treatment group, but not in the control group (-0.52 ± 0.80 m/s versus 0.04 ± 0.48 m/s, respectively; P=0.001). CRP and the Disease Activity Score (DAS28) were significantly reduced in the treatment group after 12 months (-8.2 (19.2) mg/L P<0.001 and -0.82 (1.18) P=0.004).

Conclusion: These findings indicate that anti-TNF-α therapy improves cIMT and aortic stiffness in patients with inflammatory arthropathies concurrent with reduction in inflammatory markers.

## P-003. Anxiety, Depression and Sleep in Permanent Atrial Fibrillation

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*Introduction*
Patients with atrial fibrillation (AF) have reduced health-related quality of life (HRQoL). One third of AF patients referred to a specialist clinic reported increased anxiety and depression. We hypothesized that patients with permanent AF might have poorer sleep and more symptoms of anxiety and depression than subjects in normal rhythm.

*Methods*
In a cross-sectional design we compared patients with permanent AF to a control group in sinus rhythm, all recruited from the Asker and Bærum Atrial fibrillation study which included an ECG screening in a 75 year-old age cohort in two Norwegian municipalities with high educational level. The main outcome variables were Hospital anxiety and depression scale (HADS) scores and Pittsburg sleep quality index (PSQI) score. The Life orientation test-revised (LOT-R), the Symptom checklist frequency and severity (SCL) and the Short form 36 (SF-36) were also completed. The Brief illness perception questionnaire (BIPQ) was administered to AF patients only.

*Results*
Twenty-seven subjects with permanent AF (median duration 5 years, range 0.6-25 years) and 71 subjects in sinus rhythm participated, with similar proportions of women (26 and 28 %). Neither the scores for HADS anxiety, HADS depression, PSQI, LOT-R, SCL, nor the scores for SF-36 Mental health, Emotional role or Bodily pain differed significantly between the groups. Emotional impact of illness by BIPQ were comparable to the BIPQ score in another study, in subjects having a cold. AF patients had, however, significantly poorer scores for SF-36 Physical functioning, Physical role, General health, Vitality and Social functioning compared to subjects in sinus rhythm.

*Conclusion*
Reduced physical HRQoL in permanent AF does not seem to affect anxiety, depression, sleep or emotional impact of illness in stable elderly patients with AF. Adjusted expectations to HRQoL in patients with stable chronic disease may explain why these well-adapted AF patients tolerate their impaired physical HRQoL without a deterioration also of mental health.

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## P-004. Scandinavian Candesartan Acute Stroke Trial (SCAST)

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Introduction: It has long been a controversy whether high blood pressure should be lowered in the acute phase of stroke. ACCESS (Stroke 2003;34:1699) suggested a beneficial effect of the angiotensin receptor blocker candesartan in the acute phase of stroke, but these findings need to be confirmed in new, large trials.

Methods: SCAST is an international randomising, placebo-controlled, double blinded trial of candesartan in acute stroke. Patients presenting within 30 hours of stroke (ischaemic or haemorrhagic) and with systolic blood pressure > 140 mm Hg are randomly assigned to candesartan or placebo for 7 days (doses increasing from 4 to 16 mg once daily). The follow-up period is 6 months. Primary effect variables: i) Death or major disability at 6 months; ii) Vascular death, myocardial infarction or stroke during the first 6 months.

Status/results: Patient recruitment was stopped on February 28th 2010. A total of 2,026 patients have been included at >100 centres in Norway, Sweden, Denmark, Belgium, Estonia, Finland, Lithuania, Poland and Germany. The final visit of the last

patient will be in September 2010. Characteristics at baseline: Mean age 72 years; blood pressure 172/89 mm Hg; ischaemic stroke 85%; haemorrhagic stroke 15%. Funding: Basic funding from Norwegian health authorities. Trial drugs and unconditional grants from AstraZeneca and Takeda. Participating centres receive a limited economical compensation.

Discussion/conclusion: SCAST is to date the largest trial of blood pressure lowering treatment in the acute phase of stroke, and the first large trial with an angiotensin receptor blocker for this indication.

## P-005. Ulik treningsrespons på hjertet mellom afrikanske og kaukasiske mannlige fotballspillere på elitenivå

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Hensikt. Tidligere studier har vist at mannlige kaukasiske fotballspillere har økt venstre ventrikkel (VV) masse og eksentrisk remodellering av VV. Andre har vist at afrikanske idrettsutøvere har enda større strukturelle endringer som følge av trening enn kaukasere. Hensikten med denne studien var derfor å teste dette i en større studie av profesjonelle fotballspillere. Vi ønsket samtidig å undersøke om det var andre treningsrelaterte forskjeller mellom kaukasere og afrikanere i remodelleringen av hjertets fire kamre.

Metode. 555 mannlige norske elite fotballspillere (509 kaukasere og 46 afrikanere), og 46 kaukasiske utrente kontroller ble undersøkt med ekkokardiografi. Følgende parameter ble målt i endediastole: VV indre diameter (VVIDd) og tykkelse av VV septum og bakre vegg (VVSd og VVBVd). I tilegg ble VV masse kalkulert ut fra formelen: 0,8 x (1.04[(VVIDd + VVBVd + VVSd) $^3$ - (VVIDd) $^3$]) + 0,6g. Relative veggtykkelse (RVT) ble regnet ut fra: (2*VVBVd/VVIDd). VV endediastolisk volum (VVedV) og endesystolisk volum av venstre atrium (VAesV) ble beregnet med heholdsvis Simpson`s og areal-lengde metode. Endediastolisk areal av høyre ventrikkel (HVedA) og høyre atriums endesystolisk areal (HAesA) ble målt i apikal 4-kammer vindu. Kroppsmasseindeks (BMI) og overflateareal (BSA) ble beregnet. Alle ekkokardiografianalyser ble utført blindet av samme person.

Resultat. Det var ingen signifikant forskjell i alder, BMI, BSA eller blodtrykk mellom de aktuelle gruppene. 37 av fotballspillerne hadde en VV masse/BSA over 115g/m2.

Konklusjon. Kaukasiske fotballspillere hadde signifikant større ventrikler sammenlignet med spillere av afrikansk opprinnelse. Til tross for dette var det ingen signifikant forskjell i VV masse mellom de to etniske gruppene på grunn av en mer konsentrisk remodellering av VV hos afrikanere enn hos kaukasere. Det var ingen signifikant forskjell i atriestørrelse mellom gruppene.

## P-006. Connective tissue growth factor (CCN2/CTGF) attenuates left ventricular remodeling after myocardial infarction and prevents ischemic heart failure

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Introduction: Myocardial CCN2/CTGF - connective tissue growth factor is induced in experimental models of heart failure as well as in human heart failure. However, its pathophysiological role in the development of ischemic heart failure remains unresolved.

Methods: Transgenic mice with cardiac-restricted overexpression of CTGF (Tg-CTGF) were compared with nontransgenic littermate control mice (NLC). Myocardial infarction (MI) was induced by ligation of the left coronary artery in Tg-CTGF (n=22) and NLC mice (n=21) and left ventricular (LV) remo-

| | VV mass (g) | RVT | VVedV (ml) | VAesV (ml) | HVedA (cm2) | HAesA (cm2) |
|---|---|---|---|---|---|---|
| Kaukasiske kontroller, n = 46 | 149,7 ± 35,6 †# | 0,31 ± 0,06 †# | 124,7 ± 23,9 † | 55,6 ± 20,0 †# | 24,2 ± 4,0 † | 17,6 ± 3,9 †# |
| Kaukasiske fotballspillere, n = 509 | 181,6 ± 34,4 * | 0,33 ± 0,06 *# | 146,9 ± 27,8 *# | 73,6 ± 20,8 * | 27,5 ± 4,9 *# | 21,5 ± 3,9 * |
| Afrikanske fotballspillere, n = 46 | 181,3 ± 37,5 * | 0,37 ± 0,06 *† | 131,1 ± 24,1 † | 72,2 ± 20,5 * | 24,8 ± 3,8 † | 21,6 ± 3,7 * |

**: p<0.005 vs. kontroller, †: p<0.005 vs. Kaukasiske fotballspillere, #: p<0.005 vs. Afrikanske fotballspillere*

deling and cardiac function was assessed after 4 weeks. Area at risk was estimated in a separate group of animals after perfusion with Evans blue dye, and was similar among Tg-CTGF and NLC mice. In addition, serum levels of CTGF (s-CTGF) were measured in 42 patients admitted to hospital for ST-elevation myocardial infarction (MI), 2 days, 1 week, 2 months and 1 year after percutaneous coronary intervention (PCI). Cardiac magnetic resonance imaging was performed at the same time points to determine infarct size and LV ejection fraction (EF).

Results: During the 4 weeks follow-up, there was significantly better survival in Tg-CTGF mice as compared to NLC mice; 63.6% vs. 38.1%, p<0.05. In vivo pressure-volume analysis after 4 weeks displayed preserved cardiac performance in Tg-CTGF mice, as measured by dp/dt max, LV end-diastolic and end-systolic pressure as well as cardiac output, and end-point analysis after excision of the hearts revealed attenuation of cardiac hypertrophy and pulmonary congestion in Tg-CTGF mice vs NLC mice (Heart weight/body weight ratio; 5.3±0.2mg/g, n=14 vs 8.0±0.9mg/g, n=9, p<0.05). Also, markers of myocardial remodeling, i.e. myocardial BNP and beta-myosin heavy chain mRNA levels, measured by real time qPCR analysis, were significantly less up-regulated in Tg-CTGF than in NLC hearts. Interestingly, in patients in which s-CTGF levels increased from day 2 after PCI until 2 months after PCI (n=21), infarct healing was significantly improved and LV remodeling attenuated one year after the ischemic event. Consistently, EF was also significantly higher in these patients after one year, as compared to patients with unaltered or decreased s-CTGF levels (n=21).

Conclusion: CTGF prevents development of experimental ischemic heart failure in mice, and increase in s-CTGF levels in patients after MI is associated with attenuated LV remodeling and improved cardiac function. These results may indicate cardioprotective effects of CTGF in ischemic heart failure.

## P-007. Influence of ST-segment Recovery on Infarct Size and Ejection Fraction in Patients With STEMI Receiving Primary Percutaneous Coronary Intervention

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Introduction: In ST-elevation myocardial infarction patients treated with fibrinolytics, electrocardiogram-derived measures of ST-segment recovery guide therapy decisions and predict infarct size. The clinical utility of ST-segment recovery is less

well defined in patients receiving mechanical reperfusion.

Methods: We studied 144 patients treated with primary percutaneous coronary intervention (pPCI). We aimed to define the association between infarct size as determined by cardiac magnetic resonance (cMR) and different metrics of ST-segment recovery. Electrocardiograms were assessed at baseline and 90 minutes. 3 methods for calculating and categorizing ST-segment recovery were employed: (1) Summed ST-segment deviation (STD) resolution analyzed in 3 categories (≥70 %, ≥30% to <70%, < 30%) (2) Single-lead STD resolution analyzed in same 3 categories; (3) worst-lead residual STD analyzed in 3 categories (< 1 mm, 1 to < 2 mm, and ≥ 2 mm). Infarct size and ejection fraction were assessed at 4 months by cMR.

Results: All 3 ST-segment recovery algorithms predicted final infarct size and cardiac function. Worst-lead residual STD performed equal to or better than more complex methods, and identified large subgroups at either end of the risk spectrum (table 1); with adjusted odds ratios for infarct size above median (reference < 1 mm): 1 to < 2 mm, 2.3 (95% confidence interval 0.8, 5.9); ≥ 2 mm, 6.3 (1.7 ¨C 23.7), c-index 0.781.

Conclusion: An electrocardiogram obtained early after pPCI analyzed by a simple algorithm provides prognostic information on final infarct size and cardiac function.

| Groups | Infarct size (% LV, IQR) | LVEF (%, IQR) |
|---|---|---|
| Worst-lead residual STD 90 minutes | | |
| < 1 mm (n=44) | 7.7 (10.8) | 50 (9) |
| ≥1 mm to < 2 mm (n=59) | 13.1 (13.6) | 51 (10) |
| ≥2 mm (n=41) | 24.6 (21.1) | 44 (11) |
| ∑ STD resolution at 90 minutes | | |
| ≥70% (n=88) | 10.7 (15.7) | 50 (10) |
| ≥30% to < 70 % (n=37) | 15.1 (24.0) | 48 (10) |
| < 30% (n=19) | 21.2 (33.2) | 45 (10) |
| Single-lead STD resolution at 90 minutes | | |
| ≥70% (n=71) | 9.2 (16.7) | 50 (10) |
| ≥30% to < 70 % (n=53) | 14.0 (17.0) | 48 (10) |
| < 30% (n=20) | 20.0 (17.5) | 47 (11) |

## P-008. Prognostic value of oral glucose tolerance testing in patients with a primary PCI treated ST elevation myocardial infarction.

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Introduction: Patients with acute myocardial infarction and newly detected abnormal glucose regulation (AGR) have been shown to have a less favourable prognosis compared to patients with normal glucose regulation. We have previously shown that a very early oral glucose tolerance test (OGTT) after an acute ST elevation myocardial infarction (STEMI) did not provide reliable information about long-term glucometabolic state. The aims of the present study were to relate AGR (classified by an OGTT both early and late after an acute STEMI) to clinical outcome.

Methods: Patients (n=224, median age 58 years) with a primary percutaneous coronary intervention (PCI) treated STEMI without previously known diabetes were included and followed for clinical outcome, defined as the sum of all-cause mortality, non-fatal myocardial re-infarction, recurrent ischemia causing hospital admission, and stroke. The patients were classified by a standardised 75 g OGTT at two time points, first, at 16.5 hours (median time) after admission, then at a 3 months follow-up (201 patients, one was dead and 22 were unwilling to repeat the OGTT). Based on the OGTT results, the patients were categorised according to the WHO criteria and the term AGR was defined as the sum of impaired fasting glucose, impaired glucose tolerance and type 2-diabetes.

Results: The number of STEMI patients with newly diagnosed AGR in-hospital and three months later were 105 and 50 patients, respectively. During the follow up time of median 33 months, 58 (25.9%) patients experienced a new clinical event. There were six deaths, 14 non-fatal re-infarction, 34 recurrent ischemia, and four strokes. By use of Kaplan-Meyer analysis the probability of a new clinical event was found similar in patients with abnormal and normal glucose regulation, both when classified in-hospital and re-classified three months later (Log–Rank p=0.383 and p=0.264, respectively).

Conclusion: In a primary PCI treated STEMI population without previously known diabetes, abnormal glucose regulation was not associated with a poor clinical outcome after three years follow-up, regardless of whether they were classified early in-hospital or three months later.

## P-009. Hjertestans hos ung kvinne med pacemaker

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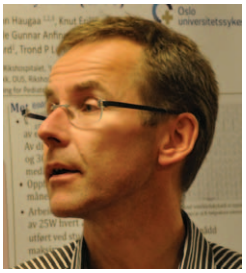

Innledning. Elektrodeperforasjon er en alvorlig, men sjelden pacemakerkomplikasjon. Dette er en dramatisk kasuistikk om en ung kvinne med medfødt hjertefeil og pacemaker-behov grunnet AV-blokk. Få måneder etter implantasjon perforerte ventrikkelelektroden og pasienten fikk hjertestans.

Bakgrunn. Pasienten var en 20 år gammel kvinne med medfødt transposisjon av de store kar. Som en konsekvens av operasjon med atrial switch (a.m Senning) i spedbarnstiden var hennes høyre ventrikkel den systemiske ventrikkel. Foruten lett nedsatt fysisk kapasitet var hun velfungerende fram til hun 19 år gammel utviklet 2.- og 3.grads AV-blokk med bradykardi. Hun fikk derfor implantert en to-kammer pacemaker med skrue-elektroder i høyre atrium og i apex av venstre ventrikkel (funksjonelt høyre).

Hjertestans. Tre måneder senere opplevde pasienten skarpe smerter i venstre hemithorax, og atter en uke senere falt hun om med hjertestans. Hun ble vellykket resuscitert. Rytmeanalyse viste AV-blokk grad 2, med rikelig innslag av pace-spikes uten kapring. Rtg thorax viste ventrikkelelektroden langt ut i venstre hemithorax og gav inntrykk av at den passerte hjerteskyggen. CT bekreftet at ledning hadde penetrert. Ekkokardiografi påviste kun fysiologisk mengde perikardvæske. Pacemakertest viste manglende eksitasjon av ventrikkelen på 7,5V@1,5ms, og minnet avslørte stigning i terskelverdien siste uke før hjertestansen. Lagrede EGM viste ventrikkelflimmer i forbindelse med hjertestansen. Pasienten hadde nå 2.grads AV-blokk med 2:1 overledning og ventrikkelfrekvens 40-50. Hun ble behandlet med hypotermi for å begrense omfanget av iskemisk skade etter hjertestans, og hun ble sikret mot ytterligere bradykardi med transvenøs temporær pacemaker. Det neste døgnet forløp uten komplikasjoner, men spørsmålet var nå nevrologisk utkomme. Etter endt hypotermi våknet pasienten uten cerebrale sekveler. Hun ble overført Rikshospitalet for hjertekirurgisk backup

ved pacemakerrevisjon. Ny elektrode ble implantert septalt i venstre ventrikkel før gammel ledning ble trukket ut. Ved kontroll ved lokalsykehuset 2 mnd senere hadde hun kommet seg flott, var mentalt adekvat og uten nevrologiske utfall. Pacemakeren ble kontrollert med normale funn.

Diskusjon. Årsak til perforasjonen er usikker. Distale del av elektroden er stiv, og mulig mekanisme er stempeleffekt mot veggen, som tilslutt ledet til penetrasjon gjennom myokard. Anamnesen med skarpe smerter i hemithorax en uke før hjertestansen, samt stigende terskelverdi, sannsynliggjør at prosessen var i gang allerede på dette tidspunktet. Manglende perikardblødning kan mulig forklares med at ledningen tettet sitt egen hull, i tillegg til at perforasjonen var i et lavtrykks-system. Perforasjonen i seg selv kan ha irritert myokard og utløst ventrikkelflimmer. Alternativt kan erstatingsrytmen ha blitt svekket sekundært til kontinuerlig ventrikkelpacing og idet pacemakeren perforerte inntrådte pace-svikt, påfølgende asystoli og derigjennom ventrikkelflimmer.

## P-010. Ingen endring av endotelfunksjon eller inflammasjonsstatus hos pasienter med familiær hyperkolesterolemi behandlet med statiner

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Bakgrunn: Familiær hyperkolesterolemi (FH) er assosiert med økt forekomst av prematur aterosklerose, og aterosklerose er forbundet med inflammasjon og endotel dysfunksjon. Det er også vist at pasienter med ubehandlet FH har endotel dysfunksjon. Vi ønsket derfor å undersøke om endotelfunksjon og inflammasjon var forskjellig hos friske kontrollpersoner og FH-pasienter behandlet med statiner.

Metoder: Pasienter med genetisk verifisert FH behandlet med statiner (n=14) ble sammenlignet med 11 friske alders- og kjønnsmatchede kontrollpersoner. Endotelfunksjonen ble undersøkt ved hjelp av Endo-PAT® systemet fra Itamar Medical Ltd. (Caesarea, Israel). Fastende blodprøver ble tatt og 27 forskjellige cytokiner, kjemokiner og vekstfaktorer ble analysert i tillegg til rutinemessige blodprøver inklusive lipidprofil.

Resultater: Det var ingen statistisk forskjell mellom FH gruppen og kontrollgruppen vedrørende alder, vekt, blodtrykk eller BMI. In vivo måling av endotelfunksjonen ble gjort ved hjelp av Endo-PAT systemet, og gjennomsnittelig reaktiv hyperemi index (RHI) var 1.58 og 1.93 (p=n.s.) i henholdsvis kontrollgruppen og FH-gruppen. Det var ingen forskjeller mellom gruppene vedrørende TNF-α (32 vs. 26 pg/mL), IL-10 (8 vs. 7), IL-6 (17 vs. 9), IL 10 (8 vs. 7), IL-1ß (1.9 vs. 1.2), IL-1ra (88 vs. 101), MCP-1 (35 vs. 45) (alle p=n.s). Det var heller ingen forskjell for noen av de andre inflammasjonsmarkørene som ble testet. Verdiene for HDL-kolesterol, LDL-kolesterol, triglyserider, APO-A, APO-B, Lp(a), homocysteine, HbA1c, trombocytter og fibrinogen var også statistisk like for de to gruppene.

Konklusjon: Endotelfunksjon målt ved RH-PAT og inflammasjonsstatus målt ved biomarkører var like for pasienter med familiær hyperkolesterolemi behandlet med statiner sammenlignet med friske kontroll personer. En mulig forklaring er en anti-inflammatorisk effekt av statin-behandlingen. Ideelt sett burde vi også undersøkt pasienter med FH uten behandling med statiner, men dette anså vi som uetisk.

## P-011. Arbeidsinduserte ventrikulære arytmier debuterer ved lavere frekvenser etter behandling med betablokkere hos pasienter med katekolaminerg polymorf ventrikkel-takykardi (CPVT)

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Bakgrunn: Katekolaminerg polymorf ventrikkeltakykardi (CPVT) er en arvelig hjertesykdom som disponerer for anstrengelsesutløste ventrikulære arytmier. Standardbehandling av pasienter med CPVT er i dag betablokkere som har vist redusert arytmiforekomst. Betablokkerbehandling kan imidlertid ha utilstrekkelig beskyttende effekt, og implantasjon av hjertestarter må vurderes individuelt. I denne studien ønsket vi å undersøke betablokkeres effekt på forekomsten av arbeidsinduserte arytmier hos CPVT-pasienter.

Metoder: 36 CPVT-pasienter ble fulgt i 24 (8-288) mnd. Seks av disse var indekspasienter og de resterende var mutasjonspositive familiemedlemmer identifisert via genetisk kaskadescreening. Klinisk undersøkelse, inkludert arbeids-EKG (50 W med økning av 25 W hvert 2. minutt), ble utført ved studiestart og 3 måneder etter at maksimal dose betablokker var oppnådd. Hjertefrekvens og arbeidsbelastning ble registrert i hvile, ved maksimal

belastning og ved debut av spredte ventrikulære ekstrasystoler samt ved mest alvorlige arytmi.

Resultater: Arbeids-EKG utløste ventrikulære ekstrasystoler hos 27 (75 %) ubehandlede CPVT-pasienter. Hvilepuls og makspuls ble redusert av behandlingen med betablokker (begge p<0,001). Både de ventrikulære ekstrasystolene og de mest alvorlige arytmiene debuterte ved lavere frekvenser ved behandling med betablokker enn uten behandling (p= 0,01), men ved tilsvarende arbeidsintensitet. Behandlingen med betablokker undertrykket forekomsten av arbeidsindusert ikke-vedvarende ventrikkeltakykardi hos 4 av 6 mutasjonsbærere (p=0,08), men forekomsten av mindre alvorlige arytmier ble ikke affisert.

Konklusjon: Arbeidsinduserte arytmier debuterte ved lavere hjertefrekvens ved betablokker-behandling enn før behandling hos CPVT-mutasjonsbærere. Det så ut til at betablokkere reduserte forekomsten av de mest alvorlige arytmiene ved arbeidsbelastning, mens de mindre alvorlige arytmiene ikke ble affisert. Den beskyttende effekten av betablokker ved CPVT kan derfor ikke tilskrives negativ kronotrop effekt alene.

## P-012. NT-proBNP Correlates Strongly With Ejection Fraction Measured by MRI in Patients Treated for ST-Elevation Myocardial Infarction

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Objectives: Several studies have demonstrated that proBNP obtained in the acute phase of myocardial infarction predicts long-term morbidity and mortality. We aimed to study the relation between NT-ProBNP and ejection fraction (EF) by MRI in the acute phase and during stable conditions after modern treatment of acute ST-elevation myocardial infarction (STEMI).

Methods: This was a sub-study of the NORwegian District Study on ST-Elevation Myocardial Infarction (NORDISTEMI) in which patients received thrombolysis and a majority of them also early invasive treatment. NT-proBNP was measured both at 3 days and 3 months after index infarction, and MRI was performed after 3 months (n=160). The association between NT-proBNP and EF was estimated by linear regression after log transforming NT-proBNP and controlling for confounders, i.e. age, anterior myocardial infarction, body mass index, HbA1c, hypertension, gender and smoking habits.

Results: The table shows the relation between MRI EF and log-NTproBNP at, a) 3 days and b) at 3 months after STEMI.

Conclusion: NT-proBNP and EF measured with MRI during stable conditions at three months following STEMI are rather closely related, but also NT-proBNP after 3 days are highly significantly related to MRI EF at 3 months. Our data suggest that NTproBNP may be a robust and useful marker of left ventricular function following modern treatment of acute myocardial infarction.

| LogNT-proBNP vs. MRI EF at 3 months (n=160) | β-coefficient | $^2$ | p-value |
|---|---|---|---|
| LogNT-proBNP at 3 days | -9.21 | 0.22 | <0.0001 |
| LogNT-proBNP at 3 months | -13.29 | 0.42 | <0.0001 |

## P-013. Increased heart rate variability parameters during non-directive meditation

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Purpose: To explore changes in heart rate variability (HRV) during non-directive meditation. Concentrative Zen techniques have shown increase of parasympathetic activity. Techniques based on free open mental attitude (non-directive) appear more effective against mental stress and hypertension.

Material and Methods: 27 middle-aged [mean age 53.1 (34–63)] subjects (14 males) were studied. Inclusion criteria were freedom from cardiovascular disease, no vasoactive or psychotropic medication, sinus rhythm, and informed consent. Registrations were obtained between 9 AM and 1 PM. The subjects first rested sitting for 20 minutes in a quiet room with closed eyes followed by 20 minutes of Acem meditation. Each subject served as his/her own control. Haemodynamic and autonomic data were collected non-invasively. HRV parameters were estimated by power spectral analyses. P-values of changes in HRV parameters between rest and meditation were calculated by paired t-test

| | N | HEART RATE | RESPIRATORY RATE | LF HRV (ms2) | HF HRV (ms2) | PSD (ms2) |
|---|---|---|---|---|---|---|
| REST | 27 | 72 | 17 | 308 | 245 | 7560 |
| MEDITATION | 27 | 69 | 16 | 531 | 624 | 14450 |
| p-value | | 0.005 | 0.045 | 0.014 | 0.013 | 0.012 |

or Wilcoxon matched pairs signed rank sum test, depending on distribution.

Results: Table depicts results based on mean variables. In the low frequency (LF) band, the minimum, mean, and maximum HRV increased during meditation (p = 0.023, 0,014, and 0.006, respectively). Similar results were observed in the high frequency (HF) band (p= 0.006, 0.013, and 0.22), and in the power spectral density (PSD) (p=0.094, 0.012 and 0.039). No statistically significant differences between rest and meditation occurred in the very low frequency (VLF) component, in the LF and HF normalised units (nu), or in the LF/HF ratio.

Conclusion: Non-directive meditation showed significant increase in HRV. No difference in the LFnu and HFnu and LF/HF ratio indicates that the balance between sympathetic and parasympathetic activity is maintained. These findings indicate that non-directive meditation by middle aged people helps to preserve HRV and may contribute toward reduction of cardiovascular morbidity and risk

## P-014. Subclinical impairment of LV function in patients with obstructive sleep apnoea syndrome (OSAS)

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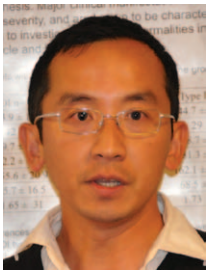

Aims: Obstructive sleep apnoea syndrome (OSAS) is associated with obesity, hypertension and heart failure. The aim of the present study was to find out if there was subclinical impairment of LV function in obese, asymptomatic and recently diagnosed OSAS patients without any recognized heart disease.

Methods: 40 consecutive patients with OSAS verified by polysomnigraphy were compared with 41 voluntary healthy controls. OSAS patients were divided into lean (BMI, body-mass-index ≤ 28; "OSAS-lean", n=16) or obese (BMI > 28; "OSAS-fat", n=24). LV systolic function was determined using 2D echo including 2D longitudinal global strain recorded from three apical views. LV diastolic function was assessed by the ratio between early diastolic- transmitral Doppler velocity (E) and the average of septal and lateral mitral tissue Doppler imaging velocities (e`). All analyses were performed by one investigator blinded to the study groups.

Results: Healthy controls and "OSAS-lean" were identical for height and body weight, while "OSAS-fat" had greater body weight. "OSAS-fat" had lower minimal nocturnal oxygen saturation and greater apopnea-hypopnea-index than "OSAS-lean". While systolic blood pressure (mmHg) was similar in "OSAS-lean" and controls (130±3 and 128±3), it was significantly elevated in "OSAS-fat" (141±3). Heart rate was increased in "OSAS-lean" (73±2 bpm) and "OSAS-fat" (74±3 bpm) compared to controls (64±1). LV dimensions were similar in all three groups, while LV ejection fraction (Simpson) was slightly but significantly lower in both OSAS groups. Septal, posterior, and relative wall thickness were all increased in "OSAS-lean", and further augmented in "OSAS-fat". Peak longitudinal global LV strain values were significantly and similarly reduced in "OSAS-lean" (-16.0±0.4%) and OSAS-fat (-15.5±0.5%) compared to controls (-18.0±0.3%). Moreover, LV global strain was inversely correlated with apopnea-hypopnea-index (R=0.55; P<0.00001). E/é ratio was significantly increased in "OSAS-fat" (10.1±0.8) as compared to "OSAS-lean" (8.4±0.7) and controls (7.7±0.3).

Conclusion: The present study demonstrates sublicinical impairment of LV systolic and diastolic function in obese asymptomatic patients with OSAS as compared to lean OSAS patients and controls. Allthough increased blood pressure might have an impact on our findings, the strong association between systolic 2D strain and the apnoea-hypnoea index indicates decreased oxygen saturation to be of importance for the impairment of LV function in patients with OSAS.

## P-015. Left ventricle dimensions in adults with osteogenesis imperfecta in Norway Substudy of the Norwegian Survey on Adults with OI

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Background: Osteogenesis imperfecta (OI) is a heterogeneous hereditary connective tissue disorder caused by genetic defects in type I collagen synthesis. The aim of this study was to investigate cardiac abnormalities in adults with osteogenesis imper-

fecta (OI), in particular dimensions of the left ventricle and LV mass.

Methods: The echocardiographic survey included 99 adults with OI divided in three clinical types, I, III and IV, and 52 controls. Blood pressure and body surface area (BSA) were measured, and LV end-diastolic dimension (LVIDd) and mass were calculated by standard 2D echocardiography and corrected for BSA.

Results: Hypertension was registered in 37 individuals (37.4%). The OI group had significantly lower BSA than the control individuals, 1.7 ± 0.3 vs. 1.9 ± 0.2 $m^2$ ($p<0.05$). LVIDd and LV mass were significantly larger in the OI group when compared to the controls, 2.98 ± .64 vs. 2.59 ± .26 cm/m2 ($p<0.05$) and 97.3 ± 30.1 vs. 73.3 ± 18.0 g/m2 ($p<0.05$), respectively. Type III OI showed significantly enlarged LVIDd as compared to OI type I and IV, 4.33 ± 1.10 vs. 2.83 ± .33 ($p<0.05$), vs. 2.85 ± .37cm/m2 ($p<0.05$), respectively. After exclusion of OI patients with hypertension, LV- mass and end-diastolic diameter were still increased in the OI group as compared to the controls, 73.3 ± 18.0 g/$m^2$ ($p < 0.001$), and 3.0 ± 0.5 vs. 2.6 ± 0.3 cm ($p < 0.001$), respectively. In the multivariate regression analysis systolic blood pressure and OI remained as significant predictors of LV- mass, while female gender was the only significant predictor for LV diameter.

Conclusions: We found for the first time increased LV- mass and internal dimension in adult OI patients as compared to controls. The changes in LV seemed to be more pronounced in patients with OI type III compared to OI types I and IV. We consider these changes to be partly caused by augmented blood pressure and partly by OI itself. ■