Methods: 990 patients with suspected NSTE-ACS were consecutively included from Sept. 2015 to Feb. 2017. Serum samples were collected at 0 (cTn0), 1 (n=481; results were not reported to clinical care), 3 and 8–12 hours. The final diagnosis was adjudicated by two independent cardiologists based on all available clinical, laboratory (i.e. hs-cTnT (Roche Diagnostics) used in clinical routine) and imaging data. All samples were measured by the hs-cTnT and hs-cTnI (Abbott Diagnostics) assays. All patients were evaluated by the HEART-Score and a value of >3 was regarded as a significant clinical risk that would overrule the biochemical ESC rule out criteria.
Results: The prevalence of NSTEMI was 13%, UAP 11%, non-ACS cardiac disease 8%, non-cardiac chest pain 58%, and 10% had other diseases. When a HEART score ≤3 were added as a clinical criterion to the ESC biochemical criteria, the number of ACS-patients who were incorrectly ruled out decreased 3–8.5 times (Table 1). Conversely, the percentages of non-cardiac chest pain patients who were correctly ruled out were 6–23% lower. Figure 1 shows the ROC-AUC of the cTn0 and HEART-Score in ACS vs. non-ACS patients.

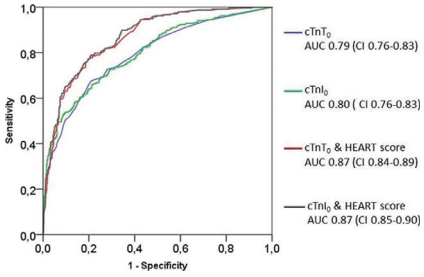

Figure 1

Conclusion: The diagnostics accuracy of the ESC Cardiac algorithms for early identification of unselected chest pain patients with NSTE-ACS was markedly improved by the use of a clinical score.
Funding Acknowledgements: Founded by Helse Bergen and Helse Stavanger

P1740
Diagnostic evaluation of the new Sgx clarity ultra-sensitivity troponin I assay in patients with suspected myocardial infarction

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Background: Cardiac troponin is the gold standard for diagnostic evaluation of patients with suspected myocardial infarction (MI). Recently, 0/1h algorithms based on high-sensitivity troponin assays (hs-Tn) have been introduced and allow a rapid decision-making in the emergency department. In the present analyses, we aimed to evaluate the diagnostic performance of a newly developed ultra-sensitivity troponin I (us-TnI) assay in patients with suspected MI.
Methods: Patients presenting with suspected MI to the emergency department were included. The final diagnosis was adjudicated by two physicians and based on all available clinical, imaging and laboratory results, including hs-TnT but excluding us-TnI. Patients with ST-elevation MI were excluded. Us-TnI measurements were performed directly on admission and after one hour. Us-TnI was measured using the Sgx Clarity™ cTnI Assay, with a limit of detection of 0.08ng/L. All patients were followed for up to one year to assess rates of mortality and incident MI. The diagnostic performance was evaluated by calculation of the area under the ROC (AUC), negative predictive value (NPV) and positive predictive value (PPV) using admission us-TnI concentrations and the 0/1h change. The dataset was 1:1 split into a derivation and validation population depending on the admission date. A NPV above 99.5% and for rule-in a PPV above 80% was targeted in the derivation population. The selected algorithm was then applied to the validation population.
Results: In the derivation dataset 767 patients were included and 155 patients were diagnosed with having MI. The AUC of an admission us-TnI was 0.92 and increased to 0.95 after one hour. For rule-out of MI an us-TnI <1ng/L dircetly on admission resulted in a NPV of 100.0% (CI 98.2–100.0). Using serial sampling an admission us-TnI <2ng/L and a 0–1h delta <1ng/L resulted in a NPV of 99.7% (CI 98.4- 100.0) and ruled-out MI in 46.8% of all patients (Figure 1). The respective one-year rate of death or MI was 0.6%. For rule-in of MI an us-TnI ^{3} 25ng/L on admission or a 0–1h delta ^{3} 6 ng/L resulted in a PPV of 81.3% (CI 73.7,87.5) and ruled-in MI in 18.5% of all patients. The one-year rate of death or MI was 12.7%. Results were similar in 767 patients from the validation cohort (Admission NPV 99.5%, 0/1h NPV 99.7%, 0/1h PPV 77.7%).
Conclusion: Application of an us-TnI assay allows the accurate triage of a large proportion of patients with suspected MI using a 0/1h algorithm.

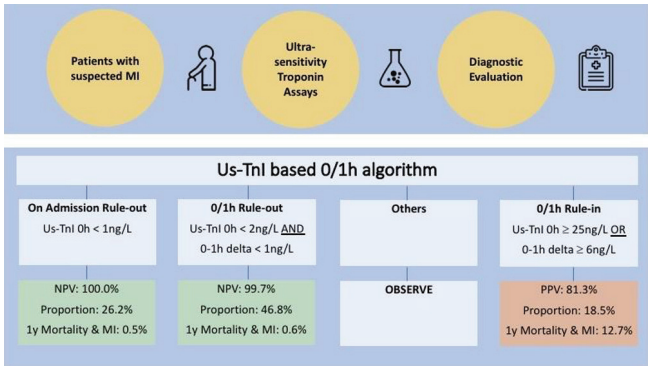

Figure 1. Central Illustration using us-TnI in patients with suspected MI. 0/1 hour algorithm to rule-out or to rule-in patients with suspected MI

P1741
Copeptin plasma level in type 1 and type 2 myocardial infarctions

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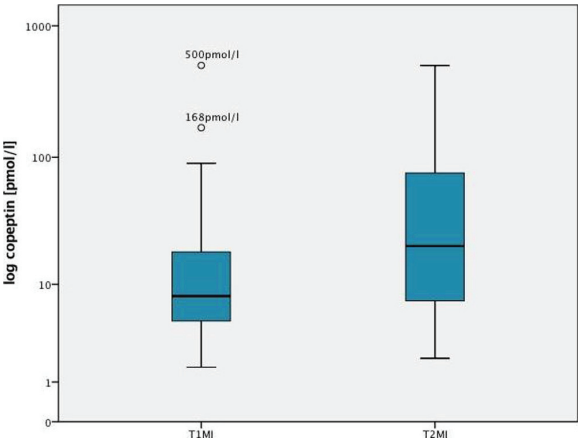
Background: During the last years, distinguishing between type 1 (T1MI) and type 2 myocardial infarction (T2MI) by use of biomarkers became a matter of clinical interest. This study aimed to investigate whether copeptin plasma levels can help to differentiate between T1MI and T2MI.
Methods: In a retrospective analysis, 959 unselected consecutive patients with chest discomfort and suspicion of acute MI were evaluated. Patients diagnosed with ST-elevation MI were excluded from the analysis. The remaining patients were classified into T1MI, T2MI, and no-MI, using clinical assessment and coronary angiography. Copeptin concentrations were measured using Thermo Scientific BRAHMS Copeptin ultrasensitive Kryptor assay and compared between both MI subtypes. Furthermore, univariable and multivariable regression analyses for significant confounders were performed.
Results: After exclusion of 848 patients (747 no MI and 102 STEMI), 111 (11.6%) subjects with NSTE-ACS were included in the analysis. Of those, 62 (55.9%) were classified by clinical means as T1MI and 49 (44.1%) as T2MI. The Mann-Whitney-U test revealed a significant difference in copeptin plasma concentrations between T1MI and T2MI patients (7.95 pmol/l [IQR 13.53] vs 20.45 pmol/l [IQR 85.74]; p=0.002) (Figure). Univariable logistic regression model for copeptin as a predictor for T2MI was statistically significant (OR 1.007 [95% CI 1.001–1.013]; p=0.023). After adjustment for the significant confounder (heart rate) elevated copeptin levels remained significantly associated with the diagnosis of T2MI (OR 1.017 [95% CI 1.004–1.029]; p=0.008).



Copeptin concentrations in T1MI and T2MI

Conclusion: Compared to T1MI patients copeptin levels were significantly higher in patients with T2MI. This association persisted after correction for significant confounders. A more pronounced elevation of copeptin levels might help in differentiating between patients with T1MI and T2MI in combination with clinical judgement.

P1742
Effects of sleep-disordered breathing on myocardial stress and renal function in patients with acute myocardial infarction

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Background: Sleep-disordered breathing (SDB) can have multiple adverse effects on the cardiovascular system.
Purpose: In this study, we aimed to examine our hypothesis that SDB may increase myocardial stress and impair renal function, depending on the severity of SDB, in patients with acute myocardial infarction (MI).

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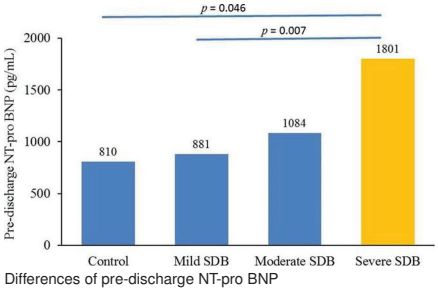
Methods: We recruited 776 patients who underwent primary percutaneous coronary intervention. Patients underwent polysomnography at 12 days, and they were divided into non-SDB, mild SDB, moderate SDB, and severe SDB groups based on their apnea-hypopnea index (AHI) (0–4.9, 5.0–14.9, 15.0–29.9, and ≥30 events/h, respectively). The N-terminal pro-brain natriuretic peptide (NT-pro BNP) and creatinine levels were measured at the time of the hospital visit and at 12 days.
Results: There was no difference in the baseline creatinine and NT-pro BNP levels across the 4 groups. Patients with severe SDB exhibited significantly higher pre-discharge creatinine and NT-pro BNP levels compared with the control and mild SDB patients (Creatinine: 1.04±0.49 mg/dL vs. 0.94±0.27 mg/dL, p=0.029, 1.04±0.49 mg/dL vs. 0.93±0.26 mg/dL, p=0.003, respectively; NT-pro BNP: 1801±3201 pg/mL vs. 810±1051 pg/mL, p=0.046, 1801±3201 vs. 881±1280, p=0.007, respectively). Multiple regression analyses adjusted for age, time to admission, anterior infarct, final Thrombolysis in Myocardial Infarction flow grade, peak creatine kinase level, Killip class, and AHI showed that AHI positively correlated with the pre-discharge creatinine levels, NT-pro BNP, and delta NT-pro BNP levels.



Differences of pre-discharge NT-pro BNP

Conclusions: Severe SDB increases myocardial stress, as estimated using the NT-pro BNP levels, and impair renal function.

ACUTE CARDIAC CARE

P1743
Hyperglycemia treatment in patients with acute heart diseases: is it possible to reset the risk of hypoglicemia?

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Rationale: In diabetic or non-diabetic hyperglycaemic patients hospitalized for acute cardiovascular diseases, the occurrence of hypoglycaemia increases the risk of mortality and morbidity without a reduction in events related to a tighter glycemic control. The guidelines about this topic agree with the exclusion of an intensive treatment, but are very discordant in recommending a conventional (blood glucose <180 mg/dl) or milder control (blood glucose <200 mg/dl).
Purpose: We adopted a protocol of mild blood glucose control based on nursing management, called "BBC200" (corrected basal-bolus with glycemic target <200), with the aim of maintaining average blood glucose <200 mg/dl and indication to intravenous insulin only for glucose >350 mg/dl, and we evaluated its efficacy and safety in patients with acute heart diseases.
Materials and methods: We used the BBC200 protocol in 1,256 hyperglycaemic, clinically stabilized patients (mean age 74±12 years) admitted in Intensive Care Unit (ICU) for acute coronary syndrome or acute heart failure. A retrospective analysis was carried out concerning the evaluation of the onset of episodes of hypoglycemia (blood glucose <70 mg/dl) and therapeutic failure (persistent hyperglycemia, with values >240 mg/dl).
Results: Mean blood glucose at admission was 231±72 mg/dl, while during treatment it was 169±50 mg/dl. The need for intravenous infusion of insulin occurred in 5 cases (0.2%). There was only one case of severe hypoglycemia (≤40 mg/dl), for an error of administration and 2 cases of moderate hypoglycemia (41–70 mg/dl), with a total hypoglycemia rate of 0.13%. Transient therapeutic failure was found in 27% of cases.

BBC200 Protocol	
Basal	Insulin glargine administration s.c. h 22:00: 0.1 U/kg if blood sugar >200 mg/dl
Bolus	Administration of rapid acting insulin s.c. with blood glucose assessment for breakfast, lunch and dinner: 1 U per 10 mg glucose increase >200 mg/dl before the meal
Glycemia >350 mg/dl	15 U of rapid acting insulin s.c. and call the doctor. If persistence of glycemia >350 mg/dl for more than 6 hours insulin e.v. is recommended

The table shows the scheme for the basal–bolus insulin administration.

Conclusions: In ICU hyperglycemic inpatients, the simple, intuitive and economical "BBC200" protocol could lead to an hypoglycemic risk very close to zero (0.13%), with a significant reduction in hypoglicemia related clinical events, and a modest increase in persistent hyperglycemic phenomena.

P1744
Hypothermia-induced diastolic dysfunction in ventricular trabeculae from human failing explanted hearts is caused by elongated contraction-relaxation cycle time and is worsened by increased heart rate

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Background: Acute myocardial infarction and ventricular arrhythmias are the most common causes of cardiac arrest. Targeted temperature management (TTM) is part of the standardized treatment for cardiac arrest patients that remain unconscious after admission. Hypothermia decreases cerebral oxygen consumption and induces physiological bradycardia reducing cardiac output; however, the effects of hypothermia on myocardial contractile function are not fully elucidated.
Purpose: Determine the effects of hypothermia on heart contractile function during different stimulation frequencies. It was hypothesized that cooling heart tissue to temperatures obtained during TTH will lead to impaired relaxation and increased diastolic tension and that increased heart rate will potentiate these effects. Second, beta-adrenergic receptor (βAR) stimulation could ameliorate the diastolic dysfunction during hypothermia.
Methods: Human left ventricular trabeculae obtained from explanted hearts from patients with terminal heart failure were stimulated at a frequency of 0.5 Hz and contraction-relaxation cycles (CRC) were recorded. Maximal developed force (Fmax), maximal rate of development of force ((dF/dt)max), time to peak force (TPF), time to 80% relaxation (TR80) and relaxation time (RT=TR80-TPF) were measured at 37–33–31–29°C. At these temperatures, stimulation frequency was increased from 0.5 to 1.0 to 1.5 Hz. At 1.5 Hz, concentration-response curves for βAR agonist isoproterenol were performed.
Results: Fmax, TPF and RT increased when temperature was lowered, whereas the (dF/dt)max decreased. At all temperatures, frequency to 1.0 and 1.5 Hz increased Fmax and (dF/dt)max, whereas TPF and RT decreased. At 31 and 29°C, diastolic tension increased at 1.5 Hz, which was ameliorated by βAR stimulation. The sensitivity to the βAR agonist isoproterenol increased by ~one log unit at 33°C and lower. At all temperatures, maximal βAR stimulation increased Fmax, (dF/dt)max and systolic tension, whereas diastolic tension was decreased progressively with lowering temperature. βAR stimulation reduced TPF and RT to the same extent at all temperatures, despite the elongated CRC.
Conclusion: Diastolic tension increased at higher stimulation frequency during hypothermia, indicating incomplete relaxation which may limit the volume of blood filling the ventricle. We suggest that diastolic dysfunction often reported during hypothermia results from an elongated CRC decreasing the time for ventricular filling during diastole. During hypothermia, physiological bradycardia protects the heart from diastolic dysfunction and increasing the heart rate should be avoided. In the event of insufficient cardiac output leading to organ hypoxia during hypothermia, low dose stimulation with a βAR agonist might be therapeutically beneficial, since it would increase stroke volume by enhancing contractile force generation while increasing the rate of relaxation, increasing the time in diastole.
Funding Acknowledgements: South-Eastern Norway Regional Health Authority, The Norwegian Council on Cardiovascular Diseases, The Research Council of Norway

P1745
Routine blood tests are strong predictors of mortality among patients with suspected heart disease in the emergency department

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Background: Patients with symptoms suspicious of heart disease are frequent in the emergency departments (ED). However, these symptoms (chest pains, palpitations or dyspnea) can be very unspecific. In example, recent studies have shown that less than 1 in 10 patients with chest pain have an acute coronary syndrome. In order to avoid unnecessary admissions without the risk of missing a serious heart condition early risk prediction is essential. Novel biomarkers have been suggested as a way of identifying the patients at high risk of adverse events. However, these can be expensive. Routine samples might have the same impact, but the use of routine markers as a risk prediction tool in patients with suspected heart disease has not previously been examined.
Purpose: To investigate the predictive strength of 8 routine laboratory tests (albumin, hemoglobin, potassium, sodium, thrombocytes, leukocytes, creatinine, and C-reactive protein) on all patients suspected of heart disease admitted to the ED. The primary end-point was 30-day mortality.
Methods: From January to June 2016 the Triage III trial included all patients

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