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 cTnI and HEART-Score in ACS vs. non-ACS patients.

Figure 1

Conclusion: The diagnostical 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.

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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-TnI) 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-TnI 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™ cTnl Assay, with a limit of detection of 0.08ng/L. All measurements were performed directly on admission and after one hour. Us-TnI was measured using admission us-TnI concentrations and the 0–1h change. The diagnostic 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.

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 directly 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 ≤2ng/L on admission or a 0–1h delta ≤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.

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 UltraSens 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).

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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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 TTT 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 effects of 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 then increasing frequencies (up to 1.5 Hz) with hypothermia. The effects of hypothermia on myocardial contractile function were measured at 37–33–29°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.

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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 pain, palpitations or dypsnea) 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, BNP, troponin, potassium, creatinine, 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