were classified as having transudative effusion and the other 5 patients were classified as having exudative effusion [fulfilling only one (n=4) or both (n=1) of the LDH criteria]. The effusion-serum electrolyte gradient (Figure) was significantly higher in chloride (7.4±2.6 mEq/L; range 4–14 mEq/L) than in sodium (0.87±1.36 mEq/L; range -1 to 4 mEq/L) and potassium (-0.12±0.254 mEq/L; range -0.8 to 0.2 mEq/L; P<0.001 for each).

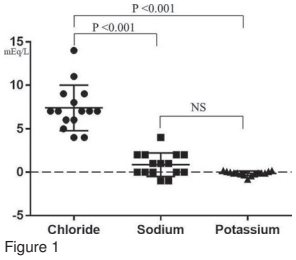

Figure 1

Conclusion: In acutely worsening HF patients with associated pleural effusion, concentration of chloride in the pleural effusion is greater than that in the serum, indicating that chloride may have an important and active role in the formation and retention of body fluid in the pleural space, and possibly in the interstitial space.

P918
Prognostic value of the combination of plasma volume status and acute kidney injury in acute decompensated heart failure patients with preserved left ventricular ejection fraction

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Background: Plasma volume (PV) expansion plays an essential role in heart failure (HF). Recently, it has been reported that PV is estimated by a simple formula based on hematocrit and body weight, not using radioisotope assays, and PV status provides prognostic information in HF patients. Acute kidney injury (AKI) during heart failure treatment is associated with poor outcome in patients admitted for ADHF. However, there is no information available on the prognostic significance of the combination of PV status and AKI in patients with acute decompensated heart failure (ADHF) with preserved left ventricular ejection fraction (LVEF).
Methods and results: We studied 184 patients admitted for ADHF with LVEF≥40%. At the admission, we calculated PV status as the following: Actual PV=(1-hematocrit) x [a + (b x body weight)] (a=1530 in males and a=864 in females, b=41 in males and b=47.9 in females), Ideal PV = c x body weight (c=39 in males and c=40 in females), and PV status=[(actual PV-ideal PV)/ideal PV] x 100 (%). AKI during ADHF treatment was defined according to AKI Network criteria (stage 1: ≥0.3mg/dl absolute or 1.5- to 2.0-fold relative increase in Cr, stage 2: >2- to 3-fold increase in Cr, stage 3: >3-fold increase in Cr or Cr≥4.0mg/dl with an acute rise of ≥0.5mg/dl). During a follow-up period of 4.6±4.0 yrs, 43 patients had cardiovascular death (CVD). At multivariate Cox analysis, PV status and AKI were significantly associated with CVD after adjustment with systolic blood pressure, hemoglobin, serum sodium and creatinine levels. CVD was significantly frequently observed in patients with both greater PV status (≥8.9% by ROC analysis; AUC 0.619 [0.519–0.721]) and stage 2/3 AKI than those with either or none of them (54% vs 29% vs 14%, p<0.0001, respectively). Hazard ratio for CVD in patients with greater PV status and stage 2/3 AKI was 6.7 [2.7–17.7], which was double of that in patients with either of them (3.4 [1.7–6.8]).
Conclusion: The combination of PV status and moderate to severe AKI might be useful for stratifying patients at risk for CVD in patients with ADHF and preserved LVEF.

P919
Clinical characteristics of patients with cancer and takotsubo cardiomyopathy - Observations from the international takotsubo registry

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Background: Takotsubo syndrome (TTS) is an acute heart failure syndrome with significant morbidity and mortality. Interestingly, we have recently reported that the prevalence of malignancies in TTS is substantially higher than in an age- and sex-matched cohort of patients with acute coronary syndrome (ACS).
Purpose: To date, clinical characteristics of TTS patients with malignancies and long-term outcomes have not been fully elucidated. As such, we analyzed the prevalence of distinct types of cancer in patients with TTS and compared their outcomes to those without malignancy.
Methods: Data from 1604 TTS patients contained in the International Takotsubo Registry (InterTAK Registry) were used to analyze the differences. InterTak Registry is a collaboration between 26 centers in 9 countries. Diagnosis of TTS was made according to the modified Mayo Clinic Diagnostic Criteria. In a sub-analysis, 411 TTS patients from the InterTAK Registry and 441 ACS patients from the Zurich Acute Coronary Syndrome Registry, were included to evaluate data on long-term mortality.
Results: Of the 1604 TTS patients, history of malignancy was observed in 16.6% of patients. TTS patients with malignancies were significantly older (69.5±11.2 vs. 65.8±13.1 years, P<0.001) and more often suffered from physical triggering factors (47.9% vs. 34.2%, P<0.001) vs. emotional stress (18.0% vs. 30.3%, P<0.001). TTS patients with malignancies had a more complicated in-hospital course, more often requiring acute cardiac care treatment (26.7% vs. 19.4%, P=0.007) and had a higher in-hospital mortality (6.7% vs. 3.4%, P=0.010). While 30-day mortality was similar between groups (P=0.17), TTS patients with malignancies had a less favourable long-term outcomes. A sub-analysis of TTS and ACS patients revealed the highest mortality rate in ACS patients with malignancies and the second highest mortality in TTS patients with malignancies. Results of a multivariable analysis demonstrated that age >70 years, atrial fibrillation, peak troponin >10x ULN, peak creatinine kinase >10x ULN, LVEF <45%, malignancy, acute or chronic neurologic and psychiatric disorders are independent predictors of long-term mortality.
Conclusions: The overall prevalence of malignancies in TTS is high and patients with TTS and malignancies have poor in-hospital and long-term outcomes. Therefore, our study suggests that there might be a close relationship between presence of malignancies and TTS. As such, it is reasonable to consider malignancy in the differential diagnosis of potential triggering and exacerbating factors in TTS presentation and course in those patients with previous history of, current known, or clinically suspected malignancy.

P920
The influence of iron status on the impaired functioning of human cardiofibroblasts and cardiomyocytes in the course of acute phase of myocarditis

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Background: Iron homeostasis is important for the maintenance of optimal cellular functioning and stands at the crossroads of the cellular processes such as inflammation and energy metabolism. Cardiofibroblasts and cardiomyocytes are the main cells involved in the pathophysiology of myocarditis and they are especially sensitive to changes in iron homeostasis. Thus, iron status could be of a particular importance for the functioning of these cells in the context of myocarditis.
Purpose: We evaluated the expression of genes involved in intracellular iron metabolism and also malfunctioning of cultured human cardiomyocytes (HCMs) and cardiofibroblasts (HCFs) exposed to sera from patients with acute phase of myocarditis and after 6 weeks follow-up and compared with healthy controls. The expression of genes under study was related to the clinical profile of patients.
Methods: In HCMs and HCFs cultured with sera from patients for 48 hours, we analyzed expression of genes involved in myocardium malfunctioning, i.e. matrix metalloprotease 1 [MMP1], transforming growth factor beta-1 [TGFB1], galectin-3 [LGALS3] and also genes of iron metabolism: ferritin heavy chain [FTH], ferritin light chain [FTL] and transferrin receptor 1 [TfR1]) at the mRNA level using qPCR. Iron status of patients was defined by serum iron and serum ferritin.
Results: HCMs cultured with sera from patients in an acute phase of myocarditis exhibited increased expression of TfR1 (p<0.05) comparing to the cells treated with sera from healthy controls, suggesting intracellular iron depletion. Cells also displayed augmented expression of TGFB1 (p<0.05) and LGALS3 (p<0.05), suggesting cardiomyocyte hypertrophy and cell malfunctioning. Further, an upregulation TfR1 of in HCMs was also strongly associated with LGALS3 upregulation (R= 0,89; p<0.0001). HCFs exposed to acute phase sera also displayed an increased expression of TfR1 (p<0.01). HCFs exhibited augmented expression of FTH (p<0.01), FTL (p<0.05) as well as upregulated expression of MMP1 (p<0.05), suggesting increased proremodeling potential. Moreover, higher expression of TfR1 was associated with increased expression of MMP1 (R=0.74, p<0.01).
Considering iron status of patients, augmented expression of both of LGALS3 and TfR1 in HCMs, was strongly correlated with serum ferritin level (R=0.89 and R=0.96, respectively; p<0.05). In HCFs, serum iron level was related to higher expression of TfR1 (R= -0.86; p<0,05) and FTL (R=-0.83; p<0,05).
Both cell lines cultured with sera collected after 6 weeks displayed a downregulation of TfR1 expression (p<0.01 for HCFs, p<0.001 for HCMs) when compared to those cultured in the presence of acute-phase sera. Similar pattern was observed in MMP1 (p<0.05), FTH (p<0.01) and FTL (p<0.01) expression in HCFs, which was also decreased.
Conclusions: The results suggest that iron homeostasis may be related to mal-

functioning of cardiomyocytes and cardiofibroblasts in the acute phase of myocarditis.
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P921
Incidence of reversible left ventricular dysfunction in post-cardiac arrest syndrome

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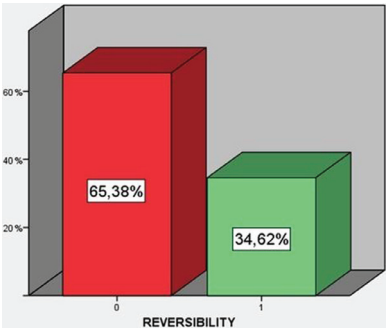
Background: Post-cardiac arrest syndrome (PCAS) includes cardiovascular and ventricular dysfunction (VD). The incidence of VD and its reversibility has not been sufficiently studied It is intended to quantify the magnitude of VD and its reversibility, and the clinical factors that may be related to.
Methods: Retrospective cohort study, which included patients admitted to our CCU with the diagnosis of sudden cardiac death (SCD), in which two echocardiogram were available.
Ejection fraction (EF) greater than 52% is classified as preserved; the EF between 46–52% as mild depressed; the EF between 40–45% as mild-moderately depressed; EF between 35–39% as moderately depressed; EF between 30–34% as moderately-severely depressed; EF less than 30% is classified as severely depressed. We classified the patients into non-reversibility (NR group) or reversibility (R group). Reversibility was defined in FE as the difference of 1 or more degrees.
Results: Data were collected from 228 patients diagnosed with SCD between February 2013 and July 2017, finally we could analyze 75. The mean ROSC of our data was 25.2 minutes.
A 34.62% reversibility in FE was observed. Reversibility was not associated with mortality (p=0.203).
The ROSC was 17'11min (group NR) vs 29'5 min (group R) (p=0'008). The 48'7% presented CPC-1 at discharge.

	Reversibility	Non reversibility	p value
Age (y)	57 (SD 15)	59 (SD 15)	0.622
Sex (w)	29.6%	28%	0.880
Prior MI	11.1%	20%	0.320
Asystole	3.7%	12%	0.520
n DF	4 (SD 4)	4 (SD 6)	0.964
ROSC (min)	29 (SD 18)	18 (SD 17)	0.015
Device (n)	3.7% (1)	4% (2)	0.949
STEMI	56%	40%	0.344
Hypothermia	56%	52%	0.765
pH	7.24 (SD 0.14)	7.27 (SD 0.16)	0.463
CPC 1	85.2%	91.3%	0.455
Exitus (n)	0% (0)	6% (3)	0.199



Conclusions: We observed reversibility of EF in the 34.6% of cases. Among the variables studied, ROSC has a statistically significant association with reversibility (p=0.008). The rest of the variables showed no association, but asystole was 4 times more frequent in the NR group.

P922
Model of end-stage liver disease excluding INR score provides additional prognostic information to the get with the guidelines-heart failure risk score in acute decompensated heart failure patients

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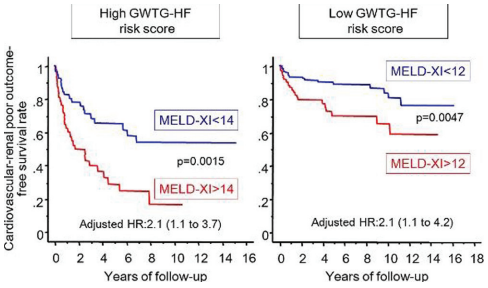
Background: The Get with The Guidelines (GWTG) heart failure (HF) risk score was developed in the GWTG inpatient HF registry to predict in-hospital mortality and has been recently reported to be associated with post-discharge long-term outcomes. On the other hand, cardiohepatic interactions have been a focus of attention in heart failure, and the model of end-stage liver disease excluding INR (MELD-XI), a robust scoring system of liver dysfunction, has been shown to be useful for prediction of poor outcome in patients with acute decompensated heart failure (ADHF). However, there is no information available on the prognostic value of MELD-XI score in ADHF patients, relating to GWTG-HF risk score.
Methods and results: We studied 303 patients admitted for ADHF and discharged with survival. Variables required for the GWTG-HF risk score were race, age, systolic blood pressure, heart rate, serum levels of blood urea nitrogen and sodium, and the presence of chronic obstructive pulmonary disease. MELD-XI score was calculated by the formula: 5.11 × ln(bilirubin) + 11.79 × ln(creatinine) + 9.44. During a follow-up period of 4.8±4.2 yrs, 85 patients had cardiovascular-renal poor outcome (CVR), defined as cardiovascular death and the development of end-stage renal disease requiring renal replacement therapy. At multivariate Cox analysis, GTWG-HF risk and MELD-XI scores were significantly independently associated with CVR, independently of prior heart failure hospitalization, after the adjustment with BMI, hemoglobin and serum albumin levels, left ventricular and atrial dimension indexes. In group with high GTWG-HF risk score (≥39: AUC 0.694 [0.626–0.763]), patients with greater MELD-XI score (≥14: AUC 0.714 [0.617–0.817]) had a significantly increased risk of CVR than those with MELD<14 (63% vs 32%, p=0.0015). Furthermore, in group with low GTWG-HF risk score (<39), patients with greater MELD-XI score (≥12: AUC 0.687 [0.588–0.786]) also had a significantly increased risk of CVR than those with MELD <12 (27% vs 13%, p=0.0015).



Conclusion: MELD-XI score would provide the additional long-term prognostic information to GWTG-HF score in patients with ADHF.

P923
Association of intra-abdominal pressure changes with early diuretic response and improvement of dyspnea in patients with acute heart failure

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Background: Increased intra-abdominal pressure (IAP) in congested patients with acute heart failure (AHF) contributes to renal dysfunction possibly through reduction of renal perfusion and increase of renal venous pressure. Appropriate IAP reduction through aggressive diuresis is associated with less worsening renal function during decongestion.
Purpose: We sought to investigate whether IAP and its changes during decongestive therapy is associated with better early diuretic response and symptomatic improvement in patients with AHF.
Methods: Forty patients hospitalized for AHF were included in the study. IAP was measured with the transvesical method via a standard Foley catheter, connected with a pressure transducer placed in-line with the iliac crest at the mid-axillary line. Measurements were performed upon admission before initiation of therapy and 48 hours after. Early diuretic response was determined as net urine output/40mg furosemide received and weight loss/40mg furosemide at 24 and 48 hours after commencement of therapy, respectively. Dyspnea was assessed by a 100-point visual analogue scale at baseline and 48 hours after. Absolute values and changes of IAP were correlated with indices of early diuretic response and change of dyspnea score at 48 hours after treatment initiation.
Results: Study population consisted of 78% males, mean age 75±11years, LVEF 37±14%, admission systolic blood pressure 132±30mmHg, and creatinine 1.34±0.61mg/dl. Mean IAP at admission was 13.1±2.6mmHg and was positively correlated with jugular venous distention (r=0.590, p<0.0001). IAP was significantly reduced at 48 hours after commencement of decongestive therapy (9.7±1.8mmHg, p<0.0001). Relative change of IAP at 48 hours compared to baseline was correlated with net urine output/40mg furosemide at 24 and 48 hours (r=0.331, p=0.037; r=0.309, p=0.052, respectively) and weight loss/40mg furosemide at 24 and 48 hours (r=0.344, p=0.03; r=0.315, p=0.048, respectively). Patients who achieved poor vs better relative reduction of IAP (< vs ≥ median value) did not differ as to baseline creatinine (p=0.311) and SBP (p=0.207); total intravenous loop diuretic dose received (p=0.988) and in-hospital use of thiazide diuretics (p=0.548); and intravenous therapy with inotropes/vasopressors (p=0.151) and vasodilators (p=0.256). A significant inverse correlation was observed between IAP at 48 hours and relative reduction in dyspnea visual analogue scale at 48 hours (r= -0.582, p<0.0001).
Conclusion: In patients with AHF, reduction of IAP by decongestive therapy is associated with better early diuretic response and improvement of dyspnea.
Funding Acknowledgements: Disclosures: EA is currently a Novartis associate and declares that this scientific research is unrelated to Novartis.