myocardial bypass unit consisting of an artificial lung and a centrifugal pump. We ex-
amined in-hospital mortality with comparison of characteristics between patients who were weaned from the respirator and those who were not weaned and the long-term prognosis.

Results: In 14 patients, intraaortic balloon pumping (IABP) was simulta-
neously used. Since FS improved from 11.6 ± 6.3 to 30.8 ± 8% (P < 0.01) following me-
dian 8 days support, 13 patients were successfully weaned from percutaneous ECMO and survived. Therefore, the in-hospital survival rate was 92.3%. Patients who were not weaned from ECMO including one patient bridged to ventricu-
lar assist device showed older age (61 ± 7 vs 40 ± 5 years), higher prevalence of gastrointestinal bleeding (71% vs 17%) and higher creatinine phosphokinase MB isoform levels (674 ± 143 vs 88.8 ± 60 U/L) than those who were weaned from ECMO (P < 0.05). During median 62 (3–154) month follow-up period, none of the 13 in-hospital survivor died or received cardiac transplantation. Only one patient was re-hospitalized due to heart failure after 14 months.

Conclusions: These findings suggested that percutaneous ECMO might be a highly effective form of hemodynamic support for the acute survival of patients with fulminating myocarditis. Once a patient recovers from inflammatory myocardial damage, the long-term prognosis is quite favorable.

3510 | BENCH
Adiponectin attenuates adverse cardiac remodeling in inflammatory heart disease by induction of matrix metalloproteinase-9 expression
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Purpose: Adiponectin (APN) is an immunomodulatory adipocytokine that inhibits left ventricular hypertrophy induced by pressure overload as well as hyperten-
sion and attenuates fibrosis after myocardial infarction. Coxackievirus B3 (CVB3) causes severe myocarditis associated with increased extracellular matrix (ECM) remodeling, which might progress to dilated cardiomyopathy. Here, we investi-
gated whether APN inhibits adverse ECM remodeling in CVB3 myocarditis by affecting matrix metalloproteinase (MMP) expression and possible underlying mechanisms.

Methods: Myocarditis was induced by CVB3 infection of APN-KO and wild-type (WT) C57BL/6 mice. mRNA and protein expression/activity of MMPs was quanti-
fied by qRT-PCR and zymography, respectively. Activation status of protein ki-
nases was determined by immunoblot. Results: In cultured cardiac myocytes and fibroblasts APN induced MMP-9 mRNA and protein expression/activity of MMPs was quanti-
ried as assessed by left ventricular relaxation (dP/dtmin), left ventricular relaxation time (Tau) and end left ventricular diastolic volume compared to WT littermates.

Conclusions: Our observations indicate that APN inhibits adverse cardiac re-
modeling by inducing inflammatory heart disease by inducing MMP-9 expression in resi-
dent cardiac and infiltrating immune cells. Persistently enhanced cardiac MMP-9 activity results in increased cleavage of accumulating collagens and augmented ECM turnover thereby attenuating development of fibrosis and cardiac dysfunc-
tion.

RAPID FIRE – UNDERSTANDING THE DIVERSITY OF ACUTE CORONARY SYNDROMES

3511 | BEDSIDE
The diagnostic value of clinical symptoms in women and men presenting with chest pain at the emergency department
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Background: Evidence about differences in the diagnostic value of clinical symp-
toms for the diagnosis of coronary artery disease (CAD) between women and men presenting with chest pain at the emergency department (ED) is lacking.

Methods: Data from a prospective validation of the HEART score for chest pain patients at the emergency department was used. Patients admitted to the ED with chest pain were eligible. The endpoint was a composite of clinically diag-
noses CAD or death within six weeks after ED presentation. CAD was defined as a myocardial infarction, PCI, CABG or a significant coronary artery stenosis at coro-
}

Results: A total of 2433 patients were included. We excluded 84 patients (3%) with an incomplete follow up. Of the remaining 2349 patients 43% (1008) were women. Therefore, 116 women (12%) and 293 men (22%), devel-
op the endpoint. The association between each clinical symptom or baseline characteristic and the presence or absence of the endpoint was quantified first. Candidate predictors with a p-value ≤ 0.15 were included in a multivariate logistic regression model. The diagnostic value of clinical symptoms and baseline char-
acteristics for predicting the endpoint as expressed by the area under the receiver operating characteristics curve, was better in women than in men, namely 0.81 (95% CI: 0.77-0.85) versus 0.74 (95% CI: 0.71-0.77), p-value < 0.01. (see added figure: red line are women, black line are men)

Conclusion: Clinical symptoms were strong predictors of CAD or death within 6 weeks in both women and men presenting with chest pain at the ED. In contrast to general belief, the diagnostic value of clinical symptoms was even better in women than in men.

3512 | BEDSIDE
Gender-specific diagnosis of acute myocardial infarction using high-sensitivity assayed cardiac troponin I
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Objective: To date, algorithms involving troponin to diagnose acute myocardial infarction (AMI) have not been differentiated by gender. Employing a recently CE-
marked high-sensitivity cardiac troponin I (cTnI) assay (Abbott), we assessed the gender-specific performance of a previously established diagnostic algorithm.

Methods: We studied 1446 pts (61±14 years, 951 men [65%]) with acute chest pain; AMI prevalence was 19%. Different 99th percentile (P99) concentrations were used for men (26pg/ml) and women (14pg/ml). cTnI was measured on ad-
mission (cTnI[0h]) and after 3 hours (cTnI[3h]). AMI rule-out criterion was cTnI[3h]
≤ P99. To rule in AMI, pts were stratified by gender and range of cTnI[0h] split at P99, with cTnI[0h] > P99 divided into terciles. AMI rule-in criteria were cTnI[3h] > P99 with a relative 3h change (ΔcTnI > predefined cutoff in pts with cTnI[0h] > P99, and exclusively ΔcTnI > predefined cutoff in pts with cTnI[0h] ≤ P99. The ΔcTnI cutoff that optimized sensitivity (S) and positive predictive value (PPV) was considered best.

Results: AMI rule-out was achieved in both genders with a negative predictive value >99%. For AMI rule-in (Table 1) in pts with cTnI[0h] > P99, ΔcTnI >200% yielded sensitivities of 92% (men) and 100% (women, p < 0.09) and PPVs of 79% in men, yet only 39% in women (p = 0.015). In pts with cTnI[0h] ≤ P99, ΔcTnI decreased with increasing cTnI[0h], with higher ΔcTnI cutoffs required for optimized S and PPV in women; sensitivities and PPVs for AMI rule-in were statistically not different between genders.

Table 1

<table>
<thead>
<tr>
<th>cTnI[0h] (pg/ml)</th>
<th>ΔcTnI</th>
<th>ΔcTnI &gt;200%</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ P99</td>
<td>2676</td>
<td>&gt;300%</td>
</tr>
<tr>
<td>P99</td>
<td>2614</td>
<td>&gt;200%</td>
</tr>
</tbody>
</table>

Conclusions: Gender-specific cTnI diagnosis ruled out AMI at 3h with high con-
fidence in men and women alike. Except for women with cTnI[0h] ≤ P99, AMI rule-in according to cTnI[0h] range – and implementing gender-specific ΔcTnI cutoff values – was achieved with comparably high sensitivities and PPVs in both genders.