determining the characteristics and prognosis of patients treated by hypothermia in a Cardiac Intensive Care Unit of a French University Hospital for an OHCA.

**Methods:** We analyzed data of patients admitted during the 3 last years (2008-10) for management of an OHCA related to a ventricular arrhythmia, by therapeutic hypothermia. Main characteristics were compared according to the intra hospital outcome.

**Results:** During the inclusion time, 53 patients (mean age: 58±15; male: 48 (90%)) were enrolled, included 18 STEMI (34%). Mean resuscitation delays were: “no flow” 4,4±6, “low flow” 23.2±16 minutes. Comparison of main data according to the intra hospital outcome (“Good outcome” group are patient discharged with good cerebral performance or moderate cerebral disability) is presented in Table 1.

**Conclusion:** prognosis of OHCA is still poor. Factors associated with good prognosis are: male gender, short delays of resuscitation (“no flow” and “low flow”), low lactate blood, coronary artery disease on the admission angiography and angioplasty.

**Table – Comparison of main data according to the intra hospital outcome.**

<table>
<thead>
<tr>
<th></th>
<th>Good outcome Group n=22</th>
<th>Poor outcome Group n=31</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>22 (100)</td>
<td>26 (83)</td>
<td>0.04</td>
</tr>
<tr>
<td>Age</td>
<td>55 ±13</td>
<td>60x16</td>
<td>0.27</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>3 (13)</td>
<td>4 (13)</td>
<td>0.93</td>
</tr>
<tr>
<td>Current smoker</td>
<td>6 (27)</td>
<td>9 (29)</td>
<td>0.88</td>
</tr>
<tr>
<td>Known cardiopathy</td>
<td>9 (41)</td>
<td>21 (67)</td>
<td>0.052</td>
</tr>
<tr>
<td><strong>Anamnesis of the cardiac arrest and management</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time of occurring (6 am-6 pm)</td>
<td>16 (72)</td>
<td>22 (71)</td>
<td>0.28</td>
</tr>
<tr>
<td>“No flow” delay (minutes)</td>
<td>2 ±3</td>
<td>5 ±6</td>
<td>0.039</td>
</tr>
<tr>
<td>“Low flow” delay (minutes)</td>
<td>15 ±14</td>
<td>29 ±16</td>
<td>0.003</td>
</tr>
<tr>
<td>STEMI</td>
<td>10 (45)</td>
<td>9 (29)</td>
<td>0.21</td>
</tr>
<tr>
<td><strong>Coronary artery disease on the admission angiography</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angioplasty</td>
<td>18 (81)</td>
<td>16 (51)</td>
<td>0.02</td>
</tr>
<tr>
<td>Delay between OHCA and beginning of hypothermia (minutes)</td>
<td>181 ±78</td>
<td>233 ±113</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Biomarkers at the admission</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Troponin (ng/ml)</td>
<td>12.7±30</td>
<td>3.1 ±4</td>
<td>0.09</td>
</tr>
<tr>
<td>Lactate blood (mmol/l)</td>
<td>2.4 ±1</td>
<td>4.0 ±2</td>
<td>0.009</td>
</tr>
<tr>
<td>pH</td>
<td>7.33 ±0.1</td>
<td>7.29 ±0.1</td>
<td>0.18</td>
</tr>
</tbody>
</table>

**371**

**Pre-hospital treatment with prasugrel versus clopidogrel in STEMI: first year experience**

Joseph Abdo [Orateur] (1), Jean Dominique Luporsi (1), M Yassine (2), M Boursier (3), Khalifé Khalifé (1)

(1) CHR Metz, Cardiologie, Metz, France – (2) CHR Metz, Cardiologie, Metz, France – (3) CHR, Metz, France

Prasugrel a new thienopyridine inhibits adenosine diphosphate induced platelet aggregation more rapidly, more consistently, and to a greater extent than do standard and higher doses of clopidogrel.

The aim of this study was to compare the effect of pre-hospital treatment with prasugrel and clopidogrel on the TIMI flow in patients admitted in cath lab with acute STEMI in 2010. The primary efficacy endpoint was the superiority of TIMI flow in patients pretreated with prasugrel and reduction of use of Glycoprotein Iib/IIa inhibitors in primary PCI. The key safety endpoint was tolerance of prasugrel and bleeding.

The primary efficacy endpoint occurred in 41% of patients receiving prasugrel and 33% of patients receiving clopidogrel (p=0.251). The subgroup (ST elevation within 6h ) had the maximal profit of TIMI FLOW with 44.1% in the prasugrel arm compared with 27,4% in the clopidogrel arm (p=0.088). No difference in the term of reduction of Glycoprotein Iib/IIa inhibitors use in primary PCI (p=0.352). The patients receiving prasugrel had a very good tolerance without elevation in rates of major bleeding.

In conclusion, there was no significant difference in term of TIMI Flow and use of Inhibitors of Glycoprotein Iib/IIa in primary PCI between the prasugrel and clopidogrel groups. The patients presenting within 6h of ST segment elevation had a better TIMI flow if pretreated with prasugrel (P=0.088) with 16,67% gain in good TIMI flow in favor of prasugrel. More studies are needed.

**372**

**Single high sensitive troponin I test followed by coronary computed tomography for low risk patients admitted in chest pain unit**

Edouard Cheneau [Orateur] Bruno Valdat, Annamaria Molon, Dimitri Panagides

Clinique Bouchard, Marseille, France

**Background:** New troponin assays achieve early high sensitivity. A normal single high sensitive (HS) troponin at time of admission might rule out acute coronary syndrome.

**Methods:** Patients with acute chest pain and low to intermediate likelihood of coronary artery disease were referred to a dedicated protocol in our chest pain unit. Patients with normal initial EKG and no noticeable alternative cause than ACS were evaluated by a single Troponin I test (Vitros I ES assay, threshold 0.012 ng/mL, 99th percentile 0.034 ng/mL) at time of admission. All patients with normal HS troponin were referred to coronary computed tomography (CCT) angiography within 3 hours (General Electric 64-detectors Scan). Patients with suspected coronary obstruction by CCT were admitted and HS troponin additionnally tested between 6 and 24 hours.

**Results:** Sixty eight patients (54+/–13 years) referred to our chest pain unit showed normal initial Troponin and EKG. Pre test- likelihood for coronary artery disease was 58%/–24%. CCT was performed and coronary angiography reconstruction was evaluable in all patients. CCT showed significant obstruction (>50% stenosis) in 13 patients (19%). Invasive coronary angiography was performed and confirmed significant obstruction in 8 patients (12%). Multivessel disease was observed in 6 and TIMI flow was <3 in 3. Serial troponin sampling showed HS troponin elevation in only one patient.

**Conclusions:** In low risk patients with acute chest pain, a normal single HS troponin sample does not rule out acute coronary syndrome. Increasing patient detection is more efficiently performed by Coronary CT at time of admission than HS troponin serial sampling.

January 14th, Saturday 2012

**373**

**Rapid non ST elevation myocardial infarction rule out with combination of copeptin and troponin in emergency department**

Sandrine Charpentier [Orateur] (1), Françoise Maupas Schwalm (2), Maxime Cournot (3), Meyer Elbaz (4), Jean Louis Ducasse (5), Dominique Lauque (1)

(1) CHU Purpan, Urgences, Toulouse, France – (2) CHU Rangueil, Biologie, Toulouse, France – (3) CH du Val d’Ariège, Cardiologie, Foix, France – (4) CHU Rangueil, Cardiologie, Toulouse, France – (5) CHU Purpan, SAMU 31, Toulouse, France

Two large studies recently related that the combination of copeptin and troponin had a remarkable negative predictive value to rule out myocardial infarction (MI).
**Objective:** The aim of this study was to analyse the diagnostic accuracy and the clinical usefulness of combination of troponin and copeptin for rapid rule out of non ST elevation myocardial infarction (NSTEMI) diagnosis in Emergency Department (ED).

**Method:** This study was an ancillary analysis of a prospective 11 months observational study. Consecutive patients admitted to an university ED for chest pain within 12 hours of ED presentation and without ST elevation on a 12-lead ECG were eligible. Blood samples for determination of copeptin were frozen at -80°C until assayed in a blinded fashion. Patients were classified by two independent physicians (kappa=0.72) as having acute coronary syndrome (ACS) and NSTEMI if cTnI was above 0.1 μg/L on serial testing. Performance of combination of cTnI and copeptin for NSTEMI diagnosis at presentation was studied and clinical utility was assessed by multivariate analysis, area under the curve (AUC) calculation for accuracy, and reporting operating characteristics with 95% confidence intervals.

**Results:** Out of the 641 eligible patients who were recruited, non-ST elevation ACS was diagnosed in 180 patients (28%) including 95 NSTEMI if cTnI alone and with copeptin were 0.92 (95%CI 0.89-0.95) and 0.94 (95%CI 0.9-0.96) respectively, p<0.05.

**Results:** Of the 641 eligible patients who were recruited, non-ST elevation ACS was diagnosed in 180 patients (28%) including 95 NSTEMI. The negative predictive value of the combination of copeptin and cTnI measures was 97.6% (95% CI 96.4-98.7) versus 92.8% (95%CI 90.8-94.8) with cTnI alone. The patient classification was significantly improved when copeptin was added to the usual diagnostic tools used for NSTEMI management: the AUC of the model with cTnI alone and with cTnI and copeptin were 0.92 (95%CI 0.89-0.95) and 0.94 (95%CI 0.9-0.96) respectively, p<0.05.

**Conclusion:** Combination of copeptin and troponin allows a rapid rule out of NSTEMI at admission in ED and improves the early triage of patients with chest pain.

### 374 Prognostic value at 6 months of discharge residual pulmonary vascular obstruction in intermediate- to high-risk pulmonary embolism

Nicolas Meneveau [Orateur], Omar Ider, Sebastien Janin, Romain Chopard, Marie France Séronde, Vincent Descotes-Genon, Francois Schiele, Yvette Bernard

**Aim:** To evaluate the prognostic value at 6 months (m) of residual pulmonary vascular obstruction (RPVO) measured at discharge in pts with intermediate- or high-risk PE.

**Methods:** 416 consecutive pts with intermediate- or high-risk PE who survived the acute phase were prospectively included. Pts with known cardiopulmonary disease were excluded. Perfusion lung scans were performed within 6-8 days after onset of treatment. RPVO was graded as the proportion of lung not perfused. Primary objective was a combined endpoint at 6 m, including death, recurrence, PE, appearance of signs of heart failure (HF).

**Results:** At 6m, 32 patients (7.7%) had ≥1 adverse event: 15 deaths (3.6%), 12 recurrent PE (2.9%), 14 cases (3.4%) of HF. Independent predictors of combined endpoint were: cancer (odds ratio (OR) 4.51 [1.63-12.5]); presence of ≥1 risk factor for venous thrombo-embolic disease (OR 4.42 [1.53-12.8]); renal insufficiency (OR 2.91 [1.16-7.27]); persistent ECG signs of cor pulmonale (OR 3.2 [1.11-9.24]); and persistent echocographic signs of RV dysfunction (OR 4.99 [1.46-16.31]). Severity of RPVO at discharge was significantly associated with unfavorable outcome (OR 2.56 [1.69-3.87]). The incremental prognostic value of the RPVO information was confirmed by a decrease in the Akaike criterion, and increases in indices of calibration and discrimination when RPVO was added to the multivariate model. The threshold RPVO value for predicting adverse events was estimated at 35% (figure). Pts with RPVO >threshold at discharge had a significantly higher risk of death at 6m (p=0.01).

**Conclusion:** RPVO evaluated before hospital discharge in pts with intermediate- to high-risk PE is a powerful prognostic factor for outcome at 6m. RPVO ≥35% is associated with an increased risk of adverse events at 6m.

### 375 Paroxysmal supraventricular tachycardia-related adverse events


**Aim:** Paroxysmal supraventricular tachycardia (SVT) is considered as benign. The purpose of the study was to report the prevalence of SVT-related adverse events.

**Methods:** 1269 patients (pts), aged from 6 to 93 years with a normal ECG in sinus rhythm were recruited for SVT, confirmed by electrophysiological study. Pts with anterograde conduction through accessory pathway (AP) were excluded. SVT-related adverse events were collected.

**Results:** Adverse event occurs in 207 pts (16%) (group I), 10 of them had a very serious event (gr I A): resuscitated ventricular fibrillation was provoked by SVT in 1 pt with coronary heart disease (HD), 1 pt with hypertrophic cardiomyopathy; 1 pt 55 years old, with respiratory failure, died after a prolonged SVT. Six pts presented resuscitated cardiac arrest after antiarrhythmic drugs used to stop SVT (sotalol, verapamil). 197 pts presented with a poorly-tolerated SVT (gr I B), syncope (148), acute coronary syndrome (36), tachycardiomyopathy (6), stage IV of heart failure (5), inappropriate shock at ICD (1), collapse after verapamil (1). Gr I was compared to pts without adverse events (gr II). Gr I was older than gr II (55±20 vs 49±18 years; p<0.003). Male gender was more frequent in gr IB (53%) than in gr II (38%) (p<0.02). Underlying HD was more frequent in gr I (32%) than in gr II (6%) (p<0.0001). The rate in tachycardia did not differ (183±41 bpm in gr I, 186±35 in gr II). SVT mechanism was similar: reentry in a concealed AP was noted in 19 % of gr I and II. Typical and atypical AV nodal reentrant tachycardias represented other causes. In gr II, 2 ablation-related deaths in old women and one sudden death 2 months after ablation were noted among 663 patients who had SVT ablation.

**Conclusions:** SVT-related adverse events occurred in 16% of patients. Advanced age, male gender and presence of HD were predisposing factors. However, life-threatening arrhythmias were rare and represented less than 1%. Most of them were drug-related.

### 376 Levosimendan as a weaning strategy from inotropes

Didier Bresson [Orateur] F Sibellas, C Petitpe, O Bastien, J Lehot, V Cart-Regal, L Sebbag, E Bonnetoy, USIC-Hôpital Cardiologique Louis Pradel, Bron, France

**Aim:** Due to its prolonged declining clinical effect, levosimendan might help in weaning patients dependent on inotropes. We assessed the effect of a 24-hour infusion of levosimendan in a cohort of advanced heart failure patients dependent on dobutamine or milrinone.

**Methods:** In this prospective observational study, 71 patients (pts), aged 58 [IQR: 44-53] years with advanced heart failure (28 ischemic) and dependent on inotropes (define as failure of >2 consecutive trials in 7 days; dobutamine n=62, milrinone n=9) were included. They received a 24-hour intravenous infusion of levosimendan (0.1 μg/kg/min) in the ICU. 4 of them had an intra-aortic balloon pump. Success was defined as “alive and free from inotrope” at the end of ICU stay.

**Results:** The median time from hospital admission to beginning of levosimendan infusion was 16 [IQR:5.5-29.2] days. Fifty five pts (76.1%) were alive and free of dobutamine or milrinone. 9 patients (16%) had to receive norepinephrine because of hypotension. Atrial fibrillation occurred in 9 pts (12.7%). Median time to stop dobutamine or milrinone was 18 [IQR:4.5-24] hours. Reinitiation of inotropes was needed in 3 pts.