Conclusion: In patients after cardiac arrest in therapeutic hypothermia the non-responder rate is extremely high. Comparing prasugrel and ticagrelor with clopidogrel, both “new” antiplatelet drugs can ameliorate platelet inhibition significantly, but still reveal high rates of non-responders. It is suggestive that improved platelet inhibition results in better clinical outcome. Large clinical studies have to test this hypothesis also in hypothermia.

P4887 | BEDSIDE
Randomized comparison of the platelet inhibitory efficacy between low dose prasugrel and standard dose clopidogrel in patients receiving dual antiplatelet therapy after coronary stent implantation
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Background: Prasugrel inhibits ADP-induced platelet aggregation to a greater extent than do standard dose clopidogrel. However, there has been little data comparing the platelet inhibitory efficacy between low dose prasugrel (5 mg) and standard dose clopidogrel (75 mg).

Methods: Forty three patients who underwent percutaneous coronary stent implantation at least one year ago were prospectively randomized to clopidogrel 75 mg (group I, n=23) and prasugrel 5 mg (group II, n=20) with aspirin 100 mg for the following 28 days. Another 20 patients were allocated to prasugrel 10 mg (group III) as reference comparison group. All patients, who weighed >60 kg and were <75 years old, had been receiving daily aspirin 100 mg and clopidogrel 75 mg at the time of randomization. Platelet function test was performed at baseline and 28 days after randomization using VerifyNow P2Y12 point-of-care assay. The primary endpoint was P2Y12 reaction unit (PRU) at 28 days between group I and group II.

Results: There were no differences in baseline PRU values between three groups. Group II had significantly lower PRU value compared with group I (174.6±60.2 vs. 223.4±72.9, p < 0.022) at 28 days, while group III showed lower PRU value (181.7±42.5) compared with group II (p < 0.001). Group II demonstrated higher percent change of PRUs, defined as the relative difference of PRUs at baseline and 28 days, compared with group I (18.7±3.9 vs. -1.2±18.9, p<0.04). The rate of high on-treatment platelet reactivity (PRU >235) was significantly lower in group II than group I (15.0% vs. 56.5%, p=0.010).

Conclusion: Prasugrel 5 mg is more potent antiplatelet therapy than clopidogrel 75 mg in non-low body weight patients on maintenance phase of dual antiplatelet therapy after percutaneous coronary stent implantation.

P4888 | BEDSIDE
Management of cardiac surgery patients with Heparin-Induced Thrombocytopenia (HIT-II): experience with preoperative plasmaphereses plus intraoperative heparin
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Rationale: Heparin-induced thrombocytopenia (HIT) is a drug-induced, immune-mediated type of thrombocytopenia. Its incidence is continuously increasing. HIT poses a tremendous surgical challenge, specifically in cardiac surgery, where heparin is the only anticoagulant drug that can be used during cardiopulmonary bypass (CPB) with the possibility of antagonization at the end of surgery. Anticoagulants that are approved for treatment of HIT (lepirudin, argatroban, danaparoid, bivalirudin) are not approved for CPB, and may pose great bleeding risks due to their lack of antagonization.

Based on the antibody-mediated nature of the disease, we reasoned that it may be possible to eliminate HIT antibodies by plasmaphereses preoperatively, allowing heparin and protamine to be used during the main surgical procedure.

Objective: We here report our first experience with plasmaphereses in 8 HIT II-positive patients undergoing major cardiothoracic surgery using heparin/protamine.

Methods: The Patients received the following operative procedures: heart-transplantation (n=4), lung-transplantation (n=1), heart-lung-transplantation (n=1) and elective aortic valve replacement (n=2). HIT II was confirmed in all 8 patients and anticoagulant treatment was performed with argatroban until the time of surgery (or until the beginning of plasmaphereses). The transplant patients received a single run of plasmaphereses immediately after the donor organ was accepted and before transplantation. The patients requiring aortic valve replacement received two or three episodes of plasmaphereses and postprocedural verification that HIT antibodies had been fully eliminated.

The surgical procedures were then performed using standard heparin/protamine. Postoperative anticoagulation was again conducted with argatroban.

Results: All patients survived the operation and are still alive. There were no complications or side effects during the plasma exchange. The use of heparin during the transplantation or valve replacement was free of complications. No thromboembolic or bleeding complications were observed.

Conclusions: The results suggest that preoperative plasma exchange to eliminate circulating anti-heparin antibodies in HIT-II positive patients and using heparin during a major cardiothoracic procedure is safe. The technique may allow a safer and technically easier treatment of a continuously growing group of patients, specifically transplant patients. However, more experience is needed to verify this suggestion.

P4889 | BEDSIDE
Stent thrombosis after primary angioplasty - incidence, timing and long term prognostic: 5 year follow-up registry
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Objective: This study sought to assess long term incidence, timing and prognostic of stent thrombosis (ST) after drug-eluting stent (DES) or bare-metal stent (BMS) implantation in patients undergoing primary percutaneous coronary intervention (PCI).

Background: Although DES markedly reduced the incidence of stent-restenosis and the need for target lesion revascularization (TLR) compared to BMS, its widespread use has raised concerns regarding the occurrence of very late ST (~1 year). The incidence and timing of ST remain unsettled, with consequent uncertainty about risk stratification and long-term recommendations for antiplatelet medications.

Methods: From 2001 to 2007 consecutive patients undergoing PCI single tertiary-care center were included and prospectively followed up for at least 5 years. We analyzed ST occurrence as defined by the Academic Research Consortium (ARC) and also assessed the cumulative incidence of major adverse cardiac events (MACE) defined as death, reinfarction or TLR at 5-year follow up.

Results: There were 1156 STEMI patients undergoing PCI in study period. Patients not receiving a stent (92, 7.9%) were excluded from the analysis. Forty patients (3.8%) were lost to follow up. Mean follow up time was 64.3 months. DES was used in 417 (39.2%). Patients receiving DES were more likely to be younger (68 ± 10 vs 69 ± 11, p = 0.03) and have single vessel disease (56 vs 48%, p = 0.02). No other baseline characteristics were found to differ between the 2 groups (Diabetes 18.3%, Severe LV dysfunction 10.2%). Five-year definite ST was found in 6 patients (1.44%) in the DES group and in 4 (0.75%) in BMS group. Considering definite or probable ST the total events number raised to 11 (2.6%) in the DES group and to 22 (4.1%) in BMS group. There was also no difference in the timing of ST (very late ST for BMS 1.1% vs 1.4% for DES, p>ns). Interestingly, ST was associated with an increase in 5-year MACE as compared to pts with reinfection not due to ST (33 vs 19.8%, p<ns). This was not driven by a mortality difference.

Conclusion: Stent thrombosis is an infrequent event (3.38%) after PCI at 5-year follow up. DES use in this setting was not associated with an increased risk of ST. Although ST is linked with dismal prognosis, this does not seem to be worse than a reinfarction unrelated to ST.

P4890 | BEDSIDE
Mean platelet volume is associated with infarct size and microvascular obstruction estimated by cardiac magnetic resonance in ST-surgical revascularizationinfarction
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Introduction: Mean platelet volume (MPV) is an indicator of platelet activation.
High MPV has been recently considered as an independent risk factor for poor outcomes after ST-segment elevation myocardial infarction (STEMI).

**Methods:** We analyzed 128 patients diagnosed with first STEMI successfully reperfused during three consecutive years. MPV was measured on admission and a cardiac magnetic resonance (CMR) exam was performed within the first week in all patients. Myocardial necrosis size was estimated by the area of late gadolinium enhancement (LGE), identifying microvascular obstruction (MVO) if present. Clinical outcomes were recorded at one year follow-up. High MPV was defined as a value in the third tertile (≥ 9.5 IL), and a low MPV, as a value in the lower two tertiles.

**Results:** We found a slight but significant correlation between MPV and infarct size (r = 0.287, p = 0.008). Patients with high MPV had more extensive infarcted area (percentage of necrosis by LGE: 17.6 ± 12.5%, p = 0.02) and more presence of MVO (patients with MVO pattern: 44.4 ± 23.3%, p = 0.027). In a multivariable analysis, HR for MACE was 3.35 (95% CI 1.1-9.3, p = 0.03) in patients with high MPV (Table).

Multivariate Cox analysis for MACE

<table>
<thead>
<tr>
<th></th>
<th>p-value</th>
<th>HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
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<td>1.03 (0.9-1.1)</td>
</tr>
<tr>
<td>Male Sex</td>
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<td>2.56 (0.7-8.7)</td>
</tr>
<tr>
<td>Smoking</td>
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<td>1.65 (0.4-7.1)</td>
</tr>
<tr>
<td>Dyslipidemia</td>
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<td>3.07 (1.0-1.1)</td>
</tr>
<tr>
<td>Hypertension</td>
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<td>1.79 (0.3-9.9)</td>
</tr>
<tr>
<td>Diabetes</td>
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<td>1.24 (0.4-3.5)</td>
</tr>
<tr>
<td>3rd tertile MPV</td>
<td>0.03</td>
<td>3.35 (1.1-9.9)</td>
</tr>
</tbody>
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MACE, major adverse cardiac event; MPV, mean platelet volume.

**Conclusions:** High MPV in patients with first STEMI is associated with higher infarct size and more presence of MVO measured by CMR.

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**P4893 | BEDSIDE**

**Title:** Ticagrelor or prasugrel in STEMI patients: a pharmacodynamic evaluation

**Authors:** M. Kerneis, J. Abtan, J. silvain, S.A. O’Connor, O. Barthelemy.

**Methods:** Platelet reactivity was prospectively evaluated using VN-P2Y12 platelet reaction unit (PRU), light transmission aggregometry (LTA) residual platelet aggregation (RPA), and Vasodilator-Stimulated-Phosphoprotein (VSP) platelet reactivity index (PRI) 30 days after an acute STEMI. We studied 92 STEMI patients who received either prasugrel 10mg od (n=54) or ticagrelor 90 mg bid (n=38) after primary coronary stenting.

**Results:** On-treatment platelet reactivity evaluated with the VN-P2Y12 assay (PRI) was lower with ticagrelor compared to prasugrel: 20.4±1.3 vs 55±8.3 (p<0.001). However, these results were not confirmed with the other platelet function tests. The more specific test, VASP PRI (%) was 13.6±2.4 on ticagrelor vs. 18.0±2.1 on prasugrel (p<0.01). Figure, High on-treatment platelet reactivity rates were 2.6% with VASP (defined as PRI ≥50%) and 0% with VN-P2Y12 (PRU >235) and LTA (% RPA >46.2%) on ticagrelor, while it was 5% with VASP 1.8% with VN-P2Y12 and LTA on prasugrel.

**Conclusions:** VASP and LTA measurements do not confirm the pharmacological superiority of ticagrelor over prasugrel when measured with VN-P2Y12 in STEMI Patients. These results suggest that VN-P2Y12 assay could overestimate the platelet inhibitory effect of ticagrelor.