**Aims:** To investigate the difference in corrected count increments 1 hour after transfusion (1h-CCI) of either Amicus double or triple dose apheresis PLT collected in either plasma or PAS III (InterSol).

**Methods:** Prospective study with 41 regular PLT apheresis donors donating 4 times in a randomized sequence 4 products: double dose PLT in plasma (2PL), double dose PLT in InterSol (2IS), triple dose PLT in plasma (3PL), and triple dose PLT in InterSol (3IS). Preliminary available 1h-CCI in thrombocytopenic hematopoietic patients are compared between the 4 products of the donors completing the 4 donations (paired, Wilcoxon signed rank test, p adjusted (adj) for multiple comparisons) and between all currently transfused products (unpaired 2-sided t-test). A linear model considering product and other factors influencing CCI is calculated on all performed transfusions (F-test).

**Results:** Paired 1h-CCI comparison in 14 donors completing all 4 donations until today shows no differences between 2IS and 3IS (p unadj 0.049, adj >0.1), 2PL and 3PL (p unadj, and adj >0.1), 2IS and 2PL (p unadj and adj >0.1), and a trend toward difference between 3IS and 3PL (p unadj 0.017, adj 0.066). The mean 1h-CCI in these 4x14 transfusions are 15.2 (2IS), 11.9 (2PL), 12.4 (3PL), and 6.0 (3IS). For the total of 98 already transfused products the mean 1h-CCI are 14.2 (2IS), 12.6 (2PL), 13.7 (3PL), 8.5 (3IS), and their comparison shows no differences (however for 2IS vs. 3IS p unadj = 0.048, adj >0.1). The linear model shows an influence of the patient diagnosis on CCI (p=0.021, however the distribution is balanced between groups), a trend toward significance for the transfused product (p=0.074), and no influence of other patient characteristics potentially influencing 1h-CCI or blood group compatibility or storage time of the transfused PLT (all p>0.1).

**Conclusions:** This preliminary evaluation shows no differences between 2 PL, 2IS, and 3PL, and a trend toward a lower CCI after transfusion of 3IS, particularly in comparison with 3PL, which may also explain the observed effect in the linear model. A higher number of transfusions are needed to confirm the significance of these results.

**P476**

**Thrombocytopenia induced by granulocyte-colony-stimulating factor in a healthy peripheral blood stem cell donor**

*M. Bernimoulin, J. Rosochova, V. Kindler, E. Levrat, Y. Tirefort, B. Mohty, S. Paiano, E. Rigal, Y. Chalandon*  
*HUG (Geneva)*

**Background:** Recombinant human G-CSF is routinely used for hematopoietic stem cell (HSC) mobilization before peripheral blood stem cell (PBSC) collection. A decrease in platelet count after G-CSF stimulation is usually minor. Serious thrombocytopenia secondary to G-CSF mobilization in a healthy donor without hematologic abnormalities has so far only been described once.

**Case:** A 58 year-old sister of a woman with MDS was selected for allogenic HSCT with PBSC. The donor had arterial hypertension (candesartan, atenolol), depressive state (fluoxetine), reflux disease (omeprazole) and overweight. Her blood values were normal. Virus serologies were consistent with previous exposure to CMV, HHV-6, Toxoplasmosis, HSV and EBV. On day 5 of G-CSF stimulation with lenograstim at 10 ug/kg/d, complicated by bone pain requiring paracetamol administration, PBSC collection by cytapheresis was planned. Unexpectedly her blood testing revealed a severe thrombocytopenia of 74 G/l. A diagnostic procedure showed: thrombocytopenia using citrated blood, absence of platelet clumping or fragmentocytes on a blood smear and no splenomegaly by ultrasound. Virus testing excluded Parvovirus B19, HIV, HBV, HCV or CMV infection. Antiphospholipid antibodies were negative. The PBSC collection resulted in nadir of 30 G/l of platelets. Donor follow-up showed full platelet recovery after 1 week.

**Discussion:** Complications of G-CSF stimulation associated with thrombocytopenia have very rarely been reported: splenic rupture after harvest, Microangiopathic thrombocytopenia in a patient with an autoimmune disorder and thrombocytopenia in patients with preexisting hematologic disorders (Felty’s syndrome or ischemic heart disease). Only one patient without hematologic abnormalities developed thrombocytopenia after G-CSF stimulation and extensive investigation remained blunt. Our patient confirms this exceptional side effect.

G-CSF stimulation leads to thrombin activation and endothelial cell activation, but not to clinically significant thromboembolic complications. In mice G-CSF stimulation resulted in thrombocytopenia unrelated to presence of spleen seemingly reflecting decreased megakaryopoiesis.

**Conclusion:** We describe the second case of G-CSF induced severe thrombocytopenia in a donor without hematologic abnormalities. Diagnostic procedure remained blank and hypotheses regarding its origin remain unanswered.