

observed at a median of 9 days (range: 0-99). The kinetics profile of platelets recovery is shown in the figure below. In the whole study population, the nadir was observed around day +7 after allo-SCT, and a plateau was reached about day +35. Filtered and irradiated donor apheresis platelets were used and patients needed a median of 1 unit (range: 0-53). In this series, 83 pts (50%) did not require any platelets transfusion during the follow-up period (median follow-up: 442 days) and 83 patients (50%) received at least one transfusion of platelets (54 were not transfused beyond day +100 after allo-SCT). Platelets count prior to RIC allo-SCT (median count 144 G/L; HR 0.44 (0.28-0.7) $p=0.002$), conditioning regimen (use of ATG; HR 1.86 (1.08-3.2) $p=0.025$) and the occurrence of acute (HR 1.54 (1.17-2.01); $p=0.001$) and severe GVHD (HR 2.36 (1.38-3.05) $p=0.0006$; 82% of patients with grade 3-4 acute GVHD were transfused) were the parameters significantly associated with platelets transfusion needs in Multivariate analysis. In this cohort, 145 pts could be assessed for platelets recovery at day +100: among them, 99 (68%) had a platelet count >99 G/L. Univariate analysis found a significant impact of AGVHD ($p<0.0001$) and Platelet count prior conditioning ($p=0.012$) but only acute GVHD (HR 5.52 (2.48-12.25); $p=0.001$) was associated with a delayed platelet recovery in a multivariate model. No impacts of pathology, GVHD prophylaxis regimen or CD34+ cell dose were demonstrated. Overall, these observations show a significantly lower rate of platelets transfusions and a quicker kinetic of platelets recovery after RIC allo-SCT and point out the effect of acute GVHD. In addition, considering the low level of myeloablation observed, RIC could be an appropriated field of investigation for the testing of megakaryocytic stimulating agents, towards further improving the safety and outcome of RIC allo-SCT.

Platelets recovery after PBSC RIC allo-SCT

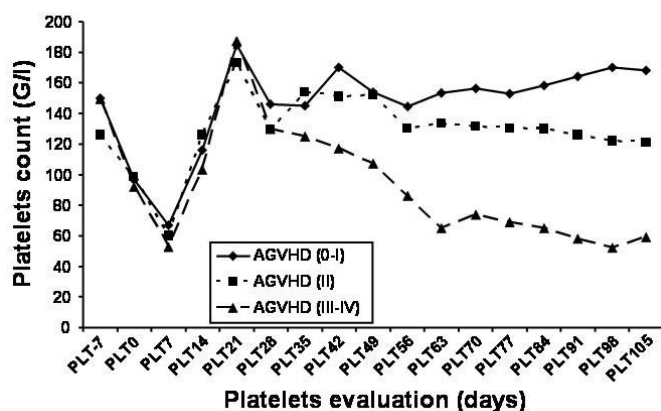


Figure 1. Analysis of Platelets recovery.

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CHANGES OF HAEMOSTASIS INDUCED BY LDL-APHERESIS

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Background. Familial hypercholesterolaemia and familial combined hyperlipidaemia are genetic disorders, which are associated with high incidence of severe cardiovascular complications. Extracorporeal elimination is used for selective removal of LDL-cholesterol in severe hypercholesterolaemias as combined strength of conservative and invasive lipid-lowering therapy may reduce progression of atherosclerosis in these high-risk patients. Activity of haemostasis plays an important role in the development of atherosclerotic complications. **Aims.** We hypothesize that LDL-apheresis reduces total plasma cholesterol and partially improves haemostasis too. The aim of this work was to verify this hypothesis. **Methods.** Repeated LDL-apheresis procedure (treatment interval 17.5±1.6 days) based on immunoadsorption has been used to treat nine patients with primary hypercholesterolaemia. Primary device Cobe-Spectra (USA); secondary device ADA (Adsorption-desorption automat, Medicap, Germany) with adsorbers Lipopak (Pocard, Russia). To assess the changes of lipid metabolism and haemostasis we analysed many markers - in this branch of our study we measured plasma concentration of thrombomodulin (Asserachrom Thrombomodulin), von Willebrand factor (STA LIAtest vWF), t-PA (Asserachrom t-PA), PAI-1

(Asserachrom PAI-1) and fibrinogen (Fibri-Prest automate 5). We compared plasma concentration of all the above items before and after LDL-apheresis. In long-term monitoring we compared plasma concentrations before LDL-apheresis. All results were evaluated as proportional differences with software Statistica 6.0 (StatSoft Inc., Tulsa, USA). **Results.** LDL-apheresis procedure induced a significant interrelated decrease of total plasma cholesterol, thrombomodulin (-29.1%), von Willebrand factor (-15.1%) and fibrinogen (-21.7%). We have found no significant changes of all the above-mentioned markers in long-term monitoring (the levels of markers were compared before procedures during the period of 300 days). **Summary/Conclusions.** Therapeutic LDL-apheresis is an invasive and effective method, which not only reduces total plasma cholesterol but also partially improves impaired haemostasis too.

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0546

METABOLIC MARKERS AND FUNCTIONAL PARAMETERS OF PLATELET CONCENTRATES COLLECTED BY MULTICOMPONENT APHERESIS WITH TWO DIFFERENT CELL SEPARATORS

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Background. In the recent years the demand for blood components is constantly increasing, while exclusion criteria for donors are strengthened. With multicomponent collection (MCC) we are able to produce several standardized components during one blood donation session. **Aims.** In the present study we investigated platelet (plt) function and metabolic parameters in double plt concentrates (PC) collected by MCC additionally to a packed red blood cell (PRBC) concentrate by two devices with different collection modes. **Methods.** 15 donors were randomly allocated to either the TRIMA Access (Gambro BCT) or the AMICUS (Baxter) device and vice versa in the second procedure following a time interval of at least two months. The separators were programmed to collect 6×10^{11} plts (2 units) and one unit of PRBC. Sample collections and analyses were done on day (d) 0 (donation day), d2 and d7. We determined blood cell counts (Sysmex SE-9500, Müller), metabolic markers (Omni, Roche), LDH (Dimension Xpand, Dade Behring) and visual plt quality (swirling effect). Activation of coagulation was performed by INTEG Assay on the ROTEM Coagulation Analyzer (Pentapharm GmbH). To assess specific function of plts in clot formation the assays were repeated by addition of abciximab (Reopro, Centocor B.V.) and cytochalasin D (Sigma Aldrich). Maximum clot firmness (MCF) and difference of maximum clot elasticity (ΔMCE) were calculated. Assays of hypotonic shock response (HSR) were performed on the SPA 2000 (Chrono-log). **Results.** Plt yields on d0 were 2.79 ± 0.23 and $2.70 \pm 0.54 \times 10^{11}$ /unit (Trima-PC and Amicus-PC). Metabolic markers are shown in the Table.

	Day 0	Day 2	Day 7
pH			
Amicus-PC	7.05 ± 0.06	7.15 ± 0.13	6.92 ± 0.15
Trima-PC	7.17 ± 0.11*	7.30 ± 0.07*	7.06 ± 0.12*
Glucose (mmol/L)			
Amicus-PC	288.00 ± 22.06	245.75 ± 20.03	144.13 ± 22.62
Trima-PC	309.29 ± 29.78*	284.64 ± 27.41**	183.00 ± 36.85*
Bicarbonate (mmol/L)			
Amicus-PC	19.02 ± 3.17	13.49 ± 2.19	5.89 ± 2.79
Trima-PC	19.07 ± 1.88	14.21 ± 1.33	6.65 ± 1.87
Lactate (mmol/L)			
Amicus-PC	3.99 ± 1.87	8.73 ± 1.64	18.03 ± 3.15
Trima-PC	2.17 ± 0.62*	5.80 ± 0.93**	15.73 ± 4.82*
Potassium (mmol/L)			
Amicus-PC	3.36 ± 0.35	3.41 ± 0.28	3.60 ± 3.79
Trima-PC	3.17 ± 0.23	3.21 ± 0.16*	3.40 ± 0.19
LDH (U/L)			
Amicus-PC	406.63 ± 71.44	492.63 ± 124.43	572.81 ± 147.67
Trima-PC	298.50 ± 51.75**	359.21 ± 46.26**	394.86 ± 51.34**

Values as mean ± SD, * $p<0.05$, ** $p<0.001$

Results of thrombelastography showed a statistically significant difference ($p<0.05$) only on d2: MCF 72.64 ± 2.34 (T-PC) and 69.88 ± 2.6 (A-PC); MCE 243.21 ± 26.41 (T-PC) and 209.88 ± 26.43 (A-PC); during storage the results of thrombelastography did not change significantly. Results of HSR showed a significant difference ($p<0.05$) between the two