**INTRODUCTION**

Platelets are essential not only for primary hemostasis but also for the initiation and propagation of thrombin generation. Neonatal platelets are known to be hypo-reactive compared to adult platelets (1). Cardiopulmonary bypass (CPB) further impairs these already compromised platelets (2). The goal of this study was to characterize the degree of neonatal platelet dysfunction at baseline, immediately after CPB and after the transfusion of adult platelets using a thromboelastography platelet mapping (TEG-PM) assay.

**METHODS**

After IRB approval and written informed parental consent, ten neonates scheduled for elective cardiac surgery requiring CPB were enrolled in this prospective observational study. Three whole blood samples were obtained: baseline before CPB, immediately after CPB and heparin reversal, and after our standard transfusion protocol of one quarter unit of apheresis platelets and three units of cryoprecipitate. TEG -PM assays (PlateletMapping®, Haemoscope, Niles, IL) were utilized to assess platelet function in response to stimulation with both arachidonic acid (AA) and adenosine diphosphate (ADP).

Due to lack of normality, Kruskal-Wallis tests were performed on each variable at all three time points. Time points for each variable were compared utilizing pairwise Wilcoxon signed-rank tests. The Holm-Bonferroni method was used to adjust for multiple comparisons. Statistical significance was set at p < 0.05.

**RESULTS**

Post-CPB MA was significantly decreased compared to baseline for all conditions. Post-CPB percent inhibition with AA (Inh-AA) and Inh-ADP were significantly increased compared to baseline but in the context of markedly reduced platelet counts. Post-transfusion MA-AA and MA-ADP showed a statistically significant increase compared to the post-CPB values but did not return to baseline. Post-transfusion Inh-AA was significantly improved post-CPB but remained elevated above the baseline value. Post-transfusion Inh-ADP remained significantly elevated and was comparable to the post-CPB level despite an increase in the overall platelet count.

**DISCUSSION**

Our data demonstrate significant inhibition of platelet function in neonates post-CPB despite transfusion with adult platelets. Possible explanations include intrinsic deficiencies in banked apheresis platelets, functional deficiencies resulting from poor integration of adult platelets into the neonatal coagulation system, or other unknown mechanisms. Return of post products MA Kaolin to baseline suggests a major role for thrombin-mediated contributions to post-operative clot strength.

**REFERENCES**