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Comparison of Hematologic Effects of Coronary Artery Bypass Grafting Surgery
Performed With and Without Use of Cardiopulmonary Bypass

A Thesis Submitted to the
Yale University School of Medicine
in Partial Fulfillment of the Requirements for the
Degree of Doctor of Medicine

By
Simon B. T. Kigwana
2018

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Abstract: Coronary artery graft surgery (CABG) using a cardiopulmonary bypass (CPB) pump to allow for stopping the heart, commonly designated as “on pump CABG” or ONCAB, requires complete anticoagulation and is associated with significant postoperative anemia. In addition, a reduction in postoperative platelet counts is relatively common in large part due to heightened activation of hemostatic pathways and platelet consumption secondary to blood passing through the CPB circuit. It has been demonstrated that both anemia and nadir platelet counts after ONCAB are associated with the incidence and severity of postoperative acute kidney injury (AKI). Over the past several years, techniques have been refined for performing CABG without CPB, commonly designated as “off pump CABG” or simply OPCAB. This approach removes the need for anticoagulation thus potentially reducing postoperative bleeding and anemia, and negates the effect of CPB on platelet consumption. Whether OPCAB surgery mitigates the severity of postoperative anemia and thrombocytopenia relative to ONCAB, however, remains unclear. Furthermore, it remains unknown if the association between nadir platelet counts and AKI evident in ONCAB patients is present following OPCAB. The present study was designed to test the hypotheses that: a) nadir platelet counts and hemoglobin values, as well as bleeding and transfusion requirements differ between ONCAB and OPCAB surgeries; b) the postoperative recovery of platelet counts and hemoglobin values is more protracted in ONCAB vs. OPCAB; and c) that hemostatic derangements are more closely associated with postoperative AKI in ONCAB as

compared to OPCAB surgeries.

Methods: With IRB approval, a retrospective study from a single institution was conducted involving 634 adult patients undergoing elective OPCAB (n=255) or ONCAB (n=379). Data were captured for demographics, medical history, surgical characteristics, postoperative blood loss (defined as chest tube output in the first 48 hours), hemoglobin levels and platelet counts, and blood product transfusions. Between groups, daily median, postoperative nadir, and discharge values for hemoglobin and platelets were compared with nadir counts defined as the median lowest in-hospital value measured over the first 5 postoperative days and at discharge. In addition, the incidence of frank thrombocytopenia, defined as platelet values of $< 74 \times 10^9/\text{dL}$, was compared along with the administration of packed red blood cells (RBC), fresh frozen plasma (FFP) and platelet suspensions. AKI was defined according to KDIGO criteria, whereby postoperative serum creatinine rise $>50\%$ or 0.3 mg/dL was indicative of injury. The incidence of AKI was then determined for the OPCAB and ONCAB groups, both as a whole and when subdivided into the segment of each group that was thrombocytopenic.

Results: The ONCAB and OPCAB cohorts were similar in regard to age (67 ± 10 vs 67 ± 10), and male/female distribution (80/20 vs 72/28). For both groups, the median nadir platelet values were observed on the second postoperative day and were not different (145K vs 142K, $p=0.44$). Similarly, the incidence of thrombocytopenia was the same following both OPCAB and ONCAB (5.88% vs. 5.54%) surgeries. Median nadir postoperative hemoglobin concentration in OPCAB patients was 10.10 mg/dl and occurred on postoperative day 2. In ONCAB patients, the median nadir postoperative hemoglobin concentration was not different (9.90 mg/dl, $p = 0.95$) but occurred on

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postoperative 4. There was no difference in postoperative bleeding measured by chest tube output between ONCAB vs. OPCAB (892 ± 421 mL vs. 850 ± 441 mL, $p = 0.24$). The incidence of red blood cell (RBC) transfusion was comparable between groups. In contrast, both rates of FFP (ONCAB 20% vs. OPCAB 8%; $p < 0.001$) as well as platelet transfusion (ONCAB 35% vs. OPCAB 10%; $p < 0.001$) were different. Overall the pattern of postoperative platelet recovery was comparable, with both cohorts recovering beyond baseline values by postoperative day 5. The overall incidence of postoperative AKI was comparable between ONCAB vs. OPCAB [33.3% ($n=126$) and 34.5% ($n = 88$)]. Patients (combined ONCAB and OPCAB) who developed severe thrombocytopenia ($n=36$) had a higher rate of AKI as compared to those with normal platelet counts (55.6% vs. 32.4%; $p=0.004$). Further, intragroup analysis (ONCAB only) demonstrated a higher incidence of AKI in those with severe postoperative thrombocytopenia as compared to patients with “normal” platelet counts [62% ($n=13$) vs. 32% ($n = 113$); $p = < 0.004$]. However, the same analysis of OPCAB patients showed no difference in the incidence of AKI [47% ($n = 7$) vs. 34% ($n=81$); $p = 0.31$] for severe thrombocytopenia vs. normal platelet counts.

Conclusion: Our findings demonstrate that contrary to the study hypotheses, ONCAB and OPCAB surgeries are actually similar in regard to postoperative thrombocytopenia, anemia, bleeding, red blood cell transfusion rates, and recovery patterns for hemoglobin and platelets. Despite these similarities, there were higher rates of FFP and platelet transfusions in the ONCAB group. Overall, the incidence of AKI was the same when comparing the entirety of both groups. However, in the subgroup of patients with postoperative thrombocytopenia, patients who underwent ONCAB had a markedly higher rate of AKI.

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Introduction

In the United States, nearly 200,000 patients undergo coronary artery bypass graft (CABG) procedures yearly for medically refractory coronary artery disease.¹ Such coronary revascularization has traditionally been performed with use of an extracorporeal apparatus named cardiopulmonary bypass (CPB)- allowing the heart to be arrested pharmacologically with cardioplegia. This approach has been further characterized as on-pump CABG or “ONCAB”; whereas, CABG performed without CPB (off-pump) is termed “OPCAB”.

Most complex cardiac surgical procedures require use of CPB. Employing this approach offers many advantages such as providing a bloodless field via diversion of blood to the CPB circuit, i.e., bypassing the heart. On the other hand, it also introduces several physiologic, metabolic, and hematologic derangements that influence morbidity and mortality.^{2,3} For example, hemodilution, consumption of coagulation factors and fibrinogen, and platelet abnormalities can potentiate bleeding and anemia that, in turn, would necessitate transfusion of blood products.^{4,5} Contact activation between blood and the extracorporeal material also triggers significant cellular and inflammatory responses and ischemia-reperfusion injuries, which have systemic effects including acute end-organ failure such as acute kidney injury (AKI).⁶ Moreover, cardiac surgery including OPCAB can generate microemboli arising from intracardiac sources and from aortic atheromatous plaques.⁷ Microaggregates formed from activated leukocytes and activated platelets may be pivotal in the pathogenesis of postoperative stroke, acute respiratory failure, and AKI.⁸ The coagulation factors, platelets, fibrinogen, and leukocytes are not only exposed to the non-biologic surface of the machine, but must endure the sheering forces of the CBP

machine.^{7,9} This may lead to release of cytokines that promote further microthrombi, exacerbating thrombocytopenia.¹⁰

In order to mitigate CPB associated complications, cardiac surgeons may opt to perform OPCAB. Development of cardiac stabilizers has made it possible to conduct CABG surgery on a beating heart and thereby avoid cardiac arrest and CPB. Ascione et al. found that OPCAB is associated with a significant reduction in inflammatory response and postoperative infection when compared with conventional revascularization with ONCAB.¹¹ OPCAB, however, is technically more challenging. Nevertheless, in a retrospective review of the STS National Adult Cardiac Database, Polomsky et al. analyzed 876,081 patients who underwent CABG between January 1, 2005 and December 31, 2010.¹² They concluded that OPCAB was associated with reduced risk of death, stroke, acute renal failure, mortality or morbidity, and prolonged length of stay compared with ONCAB.¹² In contrast, Schopka et al. conducted a matched-pair analysis of 1428 patients undergoing CABG.¹³ The patients were further stratified according to their preoperative renal function, as ascertained from glomerular filtration rate (GFR) and to risk of postoperative AKI. They concluded that use of CPB or its duration did not increase risk or severity of postoperative AKI.¹³ Thus, there remains on going controversies and inconclusive information regarding incremental benefits of OPCAB over ONCAB. It is also uncertain whether OPCAB offers advantage over ONCAB with regards to hemostatic derangements (thrombocytopenia, anemia, bleeding, and transfusion requirements). At our Institution, OPCAB surgery is performed by one surgeon, whereas, several surgeons perform ONCAB. This unique environment, allowed

us to design a retrospective study investigating aforementioned hematologic changes in ONCAB vs. OPCAB.

Primary Aim: To compare hemostatic derangements (thrombocytopenia, anemia, bleeding, transfusions) between ONCAB vs. OPCAB surgeries

Secondary Aim: (1) To compare and characterize postoperative patterns of recovery of both thrombocytopenia and anemia in ONCAB vs. OPCAB surgeries (2) and to investigate whether hemostatic derangements in ONCAB and in OPCAB surgeries relate to postoperative AKI.

- We hypothesize that hemostatic responses (nadir platelet counts, nadir hemoglobin values, bleeding, and transfusion requirements) will differ in ONCAB vs. OPCAB surgeries.
- We also hypothesize that the postoperative pattern of recovery of nadir platelet counts and of nadir hemoglobin values will be significantly more protracted in ONCAB vs. OPCAB.
- Lastly, we hypothesize that hemostatic derangements will be significantly associated with postoperative AKI in ONCAB as compared to OPCAB surgeries.

Methods

Approval by the Institutional Review Board for Clinical Investigations, Yale University School of Medicine, New Haven, CT was obtained.

Data Collection and Measurements

The following data were retrospectively collected on 634 adult patients who underwent non-emergent ONCAB or OPCAB surgeries at Yale New Haven Hospital, New Haven, CT: age, gender, race and ethnicity, height, weight, blood pressure, history of COPD, CVA, recent MI, CPB time, aortic cross clamp time, surgical course, laboratory values (platelets, hemoglobin, chemistry profile), perioperative medications, and clinical outcomes. Hemoglobin values, platelet counts, and serum creatinine data were collected from time of patient admission to hospital discharge. Three independent investigators (STK*¹, MMM*, AN*) verified the data quality by performing regular crosschecks for completeness and consistency between the data set assembled and the information available from the electronic medical records. STK created a java program in order to format data collection into an excel file.

Definitions

The primary aim involved comparison of “hemostatic derangements” that included: baseline platelets, daily nadir platelet counts; baseline hemoglobin, nadir hemoglobin values, postoperative bleeding (postoperative 48 hour chest tube outputs), transfusions (red blood cells, fresh frozen plasma, cryoprecipitate, cell saved blood, and

¹ MMM – Miriam Monteiro-Mascarenhas AN –Adambeke Nwozuzu

platelets). Baseline laboratory values indicate preoperative measurements closest to the date of surgery. Subsequent measurements were characterized as day of surgery (DOS), postoperative day (POD) 1, 2, 3, etc., and day of discharge from the hospital (Discharge). Nadir platelet counts was defined as the median of the lowest in-hospital values measured for the first 7 postoperative days and at discharge. Also nadir hemoglobin value was defined as the median of the lowest in-hospital values measure for the first 7 days postoperatively and at discharge. Likewise, we also recorded perioperative creatinine values and defined baseline creatinine as the preoperative closest to the date of surgery. An algorithm was constructed to ensure consistency for recording baseline creatinine values.

Acute kidney injury was defined according with the Kidney Disease Improving Outcomes (KIDGO) guidelines. Briefly, KIDGO separates AKI into 3 stages. Stage 1*a*. Serum creatinine increase of (≥ 0.3 mg/dl) within a 48 hr. interval; 1*b*- an increase of creatinine levels of 1.5-1.9 times baseline recordings; stage 2 AKI as a serum creatinine increase of >2.0 – 3.0 -times baseline recordings; stage 3*a* AKI serum creatinine increase of >3.0 -times baseline recordings; and stage 3*b as* serum creatinine of (≥ 4.0 mg/dl) with an acute increase of at least (0.5 mg/dl).

Lastly, severe thrombocytopenia was defined as platelet values $< 74 \times 10^9$ /dL based on the lowest tertile median value from our study cohorts.

Statistics

The Wilcoxon rank sum test was used for statistical analysis of hemoglobin and platelet values. Hemoglobin, platelets, creatinine percent change was calculated by dividing the patients baseline lab value into the patient's daily lab value minus baseline lab value:

$$\text{Percent Change} = \frac{\text{Daily lab value} - \text{Baseline lab value}}{\text{Baseline lab value}}$$

Absolute change was calculated by subtracting baseline lab value, from Daily lab value,

$$\text{Absolute change} = \text{Daily lab value} - \text{Baseline lab value}$$

The means for these values for each group + 95% confidence upper and lower limits were plotted against, postoperative day. T-test was used for the following comparisons between ONCAB and OPCAB: demographics, clinical and surgical characteristics, hemostatic responses (transfusion, bleeding), intraoperative, and postoperative pharmacological therapy categories. Chi-square test was used to calculate rates of AKI.

Results

In total, 634 patients were included in our retrospective study. The ONCAB cohort consisted of 369 patients, while the OPCAB cohort consisted of 255 patients. Demographic, medical, surgical characteristics are presented on table 1.

Perioperative Platelets Trajectory and Recovery

The median baseline, nadir, and discharge values for platelet counts were similar in ONCAB vs. OPCAB and are shown in table 2. The median nadir platelet values were observed on POD2 for both groups (145K vs. 142K, $p=0.44$). The pattern of daily decline and recovery of platelet counts was also similar in ONCAB vs. OPCAB; albeit, non-clinically significant differences were observed between the two cohorts. For example, on POD3 and POD4, the median platelet counts were (156K vs. 163K, $p < 0.04$ and 191K vs. 202K, $p=0.035$) for ONCAB vs. OPCAB, respectively. Figure 1 depicts postoperative platelet counts according to percent change and absolute change from baseline platelet values. Overall, the incidence of thrombocytopenia was similar in OPCAB vs. ONCAB (5.88% vs. 5.54%) surgeries.

Perioperative Anemia and Recovery

Median baseline, nadir, and discharge hemoglobin values are presented in table 3. Overall, nadir postoperative hemoglobin concentration was 10.10 mg/dl and occurred on POD2 in OPCAB patients; whereas, in ONCAB patients, the minimum median postoperative hemoglobin concentration was 9.90 mg/dl- occurring on POD4. Using the Wilcoxon rank sum test, there was a significant difference in median hemoglobin values on POD4, POD5, and on day of discharge between groups (table 3). Figure 2a and 2b

summarize perioperative patterns of hemoglobin- depicting its percent and absolute changes from baseline. Such patterns of hemoglobin decline was similar in ONCAB vs. OPCAB. Further, our results show markedly protracted recovery of in-hospital anemia for both groups.

Transfusion of Blood Products

Table 4 contains results on transfusion of blood component therapies- comparing ONCAB and OPCAB surgeries. There was no significant difference in *total volume* of blood products transfused between the two groups. That is, among those patients transfused in ONCAB and in OPCAB, average total volumes of RBCs, FFP, platelets, cryoprecipitate, and cell saved blood were similar. Of interest, the incidence of RBC transfusion was also comparable. In contrast, both rates of FFP (20% vs. 8%; $p<0.001$) and platelets transfusion (35% vs. 10%; $p<0.001$) were significantly greater in ONCAB vs. OPCAB.

Postoperative Acute Kidney Injury

Using the KDIGO criteria for AKI, the overall incidence of postoperative AKI was comparable between ONCAB vs. OPCAB [33.3% (n=126) and 34.5% (n = 88)]. In both groups, the majority of patients experienced stage I AKI [ONCAB (91%) vs OPCAB (83.3%)]; stages II (0% vs. 7%), and stage III (9% vs. 11%).

Consistent with our prior work, we investigated the influence of platelets on postoperative AKI. Whereas the incidence of severe thrombocytopenia was similar in ONCAB vs OPCAB, patients (combined ONCAB and OPCAB) who developed severe thrombocytopenia (n=36) had a higher rate of AKI as compared to those with normal platelet counts (55.6% vs. 32.4%; $p=0.004$). Further, intragroup analysis (ONCAB only)

demonstrated a significantly higher incidence of AKI in those with severe postoperative thrombocytopenia [62% (n=13) vs. 32% (n = 113); $p < 0.004$] as compared to patients with “normal” platelet counts. The same analysis was conducted in OPCAB patient showing a non-significant incidence of AKI (47% vs. 34%; $p = 0.31$) for severe thrombocytopenia vs. normal platelet counts.

Postoperative Bleeding

Postoperative bleeding was evaluated by total chest tube outputs and by mediastinal re-exploration for bleeding. Overall chest tube outputs were unremarkable between ONCAB and OPCAB (832 ± 364 mls vs. 940 ± 537 mls, $p=0.05$) and no difference in re-exploration was observed.

Discussion

The hematologic effects of either ONCAB or OPCAB remain poorly studied. As such, based on available and relevant information, we surmised that compared to OPCAB, CPB associated hematologic derangements in ONCAB surgery would have a more profound effect on postoperative platelet counts, anemia, bleeding, and transfusion requirements. Also, based on our previous work linking thrombocytopenia to postoperative AKI in ONCAB surgery, we designed the current study to investigate the relation between postoperative platelets and AKI in ONCAB and in OPCAB.

Our findings are intriguing as both ONCAB and OPCAB surgeries resulted in similar and significant postoperative reduction from baseline values for platelets and for hemoglobin. Further, rates of severe thrombocytopenia, volume of chest tube outputs, incidence of AKI, and transfusion of RBCs were comparable in ONCAB vs. OPCAB. We did observe a near two-fold increase in transfusion rate for FFP and a near three-fold higher rate for platelets transfusion in the ONCAB vs. OPCAB procedure.

Perioperative Platelets and Clinical Implications in CABG Surgery

Platelet activation during cardiac surgery is well recognized, and several studies have shown its role in perioperative stroke and myocardial infarction.¹⁴ During and after CPB, complex pathophysiologic processes in both humoral and cellular processes occur that involve coagulation factors, platelets, fibrinogen, vascular endothelium, and leukocytes, which may promote a shift in risks from bleeding toward microthrombosis.^{15,16} In certain perioperative conditions, a transition zone between bleeding-related and thrombosis-promoting coagulopathy is likely present; while in other

settings, bleeding and microthrombosis can occur concurrently, as observed in disseminated intravascular coagulation.¹⁷

Nevertheless, CPB has also been shown to promote formation of microemboli consisting of fibrin, activated leukocytes, and aggregated platelets. In addition, recent evidence supports the role of platelets^{15,16} in defining endothelial responses and neutrophil recruitment in ischemia/reperfusion injury including distant organ injury¹⁸ and postischemic renal failure.¹⁹ It is noteworthy that thrombocytopenia is one of the most common laboratory abnormalities in critically ill patients, and has been attributed in this setting to increased platelet destruction (immune and non-immune), hemodilution, platelet sequestration, or decreased production.^{20,21} Importantly, such thrombocytopenia is closely associated with prolonged ICU length of stay, reduced survival and increased incidence of AKI.^{22,23}

We recently reported a significant association between postoperative nadir platelet counts and postoperative AKI.⁸ The magnitude of decrease in platelet counts correlated significantly with the severity of kidney injury, as well as short-term mortality.⁸ While a significant drop in platelet counts is prevalent after CPB, the implication of this finding has not been comprehensively examined in a cardiac surgical patients undergoing OPCAB surgery. Our findings are novel as the pattern of reduction in platelet counts and its postoperative recovery in ONCAB was very similar OPCAB- suggesting that CABG surgery itself- irrespective of CPB- causes profound effects on platelet activation and consumption. These findings were unexpected as conventional teaching and thinking would support a greater CPB-associated quantitative and qualitative platelet derangement as compared to OPCAB. Our findings also challenge

conventional views on bleeding risks in ONCAP vs. OPCAB. It is opined that together with platelet abnormalities, hemodilution, and marked reduction of coagulation factors and fibrinogen would increase bleeding in ONCAP vs. OPCAB. Based on chest tube outputs, we could not demonstrate a difference in bleeding between the two cohorts. We found similar transfusion rate of RBCs- also raising an important question regarding pre-existing clinical beliefs about hematologic effects of CPB in relation to major vascular surgery without use of CPB. Nevertheless, we acknowledge that effects of CPB on platelet counts cannot be overlooked as our study found thrice the rate of platelets transfusion in ONCAP vs OPCAB. Giving the retrospective design of our study; lack of a protocol-guided transfusion practice; and qualitative assessment of platelet function, it remains uncertain whether transfusion of platelets and FFP were driven by subjective vs. objective factors.

Consistent with our previous report, postoperative thrombocytopenia was associated with AKI. Overall, compared to patients with normal platelet counts, the presence of postoperative severe thrombocytopenia significantly increased the rate of AKI (56% vs. 34%). Importantly, when we analyze severe thrombocytopenia (platelet counts <75K) as a predictor of AKI, we also found a significant association in ONCAP patients but not in OPCAB (47% vs. 34%, $p=0.31$). This is likely because of the small sample size of patients with severe thrombocytopenia ($n= 15$) in the OPCAB cohort. Our rationale for exploring AKI in patients in the lowest tertile is because this level of thrombocytopenia (platelet counts <75) reflects a clinically important threshold of platelet counts. Further, this approach should be a less arbitrary threshold for defining thrombocytopenia (i.e. platelet counts < 100K) as is commonly chosen in some studies.

In the OPCAB cohort, the incidence of AKI in thrombocytopenic patients was also much higher than in patients with normal platelet counts

Postoperative Anemia and Recovery in ONCAB vs. OPCAB

We demonstrated that the magnitude of anemia after surgery and its recovery was similar in OPCAB as observed in ONCAB. This finding also differs from our hypothesis as we surmised that CPB-associated consumptive processes, hemodilution, and bleeding would result in a more profound anemia, greater RBC transfusion and a slower recovery of anemia as compared to the OPCAB approach. Thus, these findings raise many questions regarding hemostatic effects of CABG surgeries and their management. Are anesthesiologists, cardiac surgeons, and intensivists less restrictive with fluid administration in OPCAB vs. ONCAB? Does OPCAB surgery have similar consumptive and damaging effects on RBCs as seen in ONCAB? Should there be similar transfusion triggers in OPCAB as in ONCAB?

Limitations

Because data was not acquired prospectively, there are two instances of potential human errors. First, when clinical information was first documented into medical records. Second, when the information was retrieved from medical records. Thirdly, all of the information was acquired from electronic medical records; therefore, several missing data points could not be retrieved.

Our study was conducted at a single tertiary medical center. This fact limits the applicability of our results to study populations at other centers. Nevertheless, we had

sufficient data on perioperative hematologic factors (platelets, hemoglobin, creatinine, chest tube outputs, and specific transfusion therapies)- allowing for relevant and adequate comparisons of predictor and outcome variables between ONCAB and OPCAB patients.

Lastly, we are not able to provide causes of postoperative anemia and thrombocytopenia or a more precise mechanism of platelet associated AKI.

Conclusion

Our study reveals that ONCAB and OPCAB surgeries are associated with similar hematologic effects such as postoperative thrombocytopenia, anemia, transfusion rates of RBCs, chest tube outputs, and incidence of AKI. We also observe higher rates of FFP and platelets transfusion in the ONCAB group. Lastly, patterns of postoperative recovery of both platelet counts and anemia were similarly protracted in both ONCAB and OPCAB. Future studies on mechanisms of hematologic derangements in OPCAB surgery are warranted.

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Table 1: Demographics, Medical History, Laboratory Values, and Acute Kidney Injury

	OPCAB (N = 253)	ONCAB (N =376)	P Values
Age	67.38 (9.97)	67.29 (9.53)	0.94
Male	182 (71.94%)	298 (79.26%)	
Female	71 (28.06%)	78 (20.74%)	
Medical History			
COPD	31 (12%)	62 (16.5%)	0.14
PVD	42 (17%)	40 (11%)	0.029
CVA	14 (5.5%)	28 (7.5%)	0.35
Diabetes	103 (40.7%)	153(40.7%)	1.00
Preoperative laboratory Values			
Creatinine Baseline ^β	1.15 ± (0.78)	1.15 ± (0.56)	0.99
Platelets Baseline	209.00± (80)	218.50± (73)	0.97
Hemoglobin Baseline	13.60± (1.8)	13.25± (1.9)	0.1505
Post operative clinical events			
Acute Kidney Injury	88 (34.51%)	126 (33.25%)	0.74

Table 2. Perioperative Platelet Counts in ONCAB vs. OPCAB

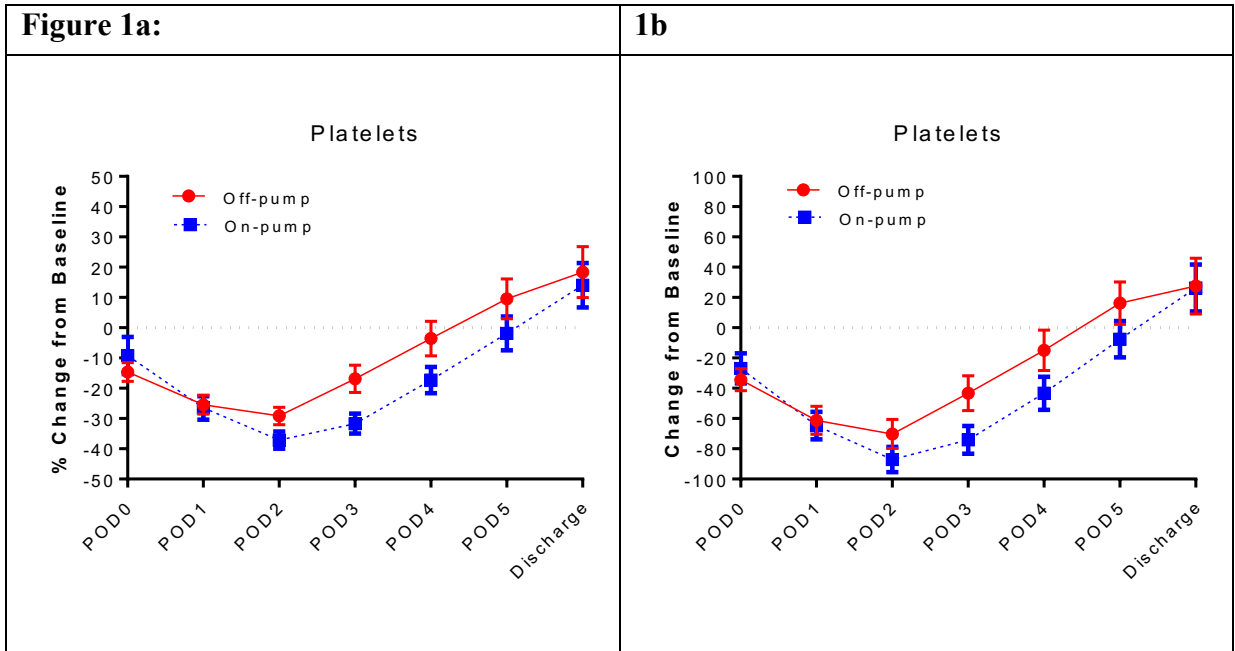
	OPCAB			ONCAB			
	Median	min	max	median	min	max	P value
Baseline	209	121	609	219	120	790	0.05
Nadir	139	83	380	132	81	357	0.14
Discharge	233	92	662	215	104	616	0.055

Table 3. Perioperative Hemoglobin Values in ONCAB vs. OPCAB

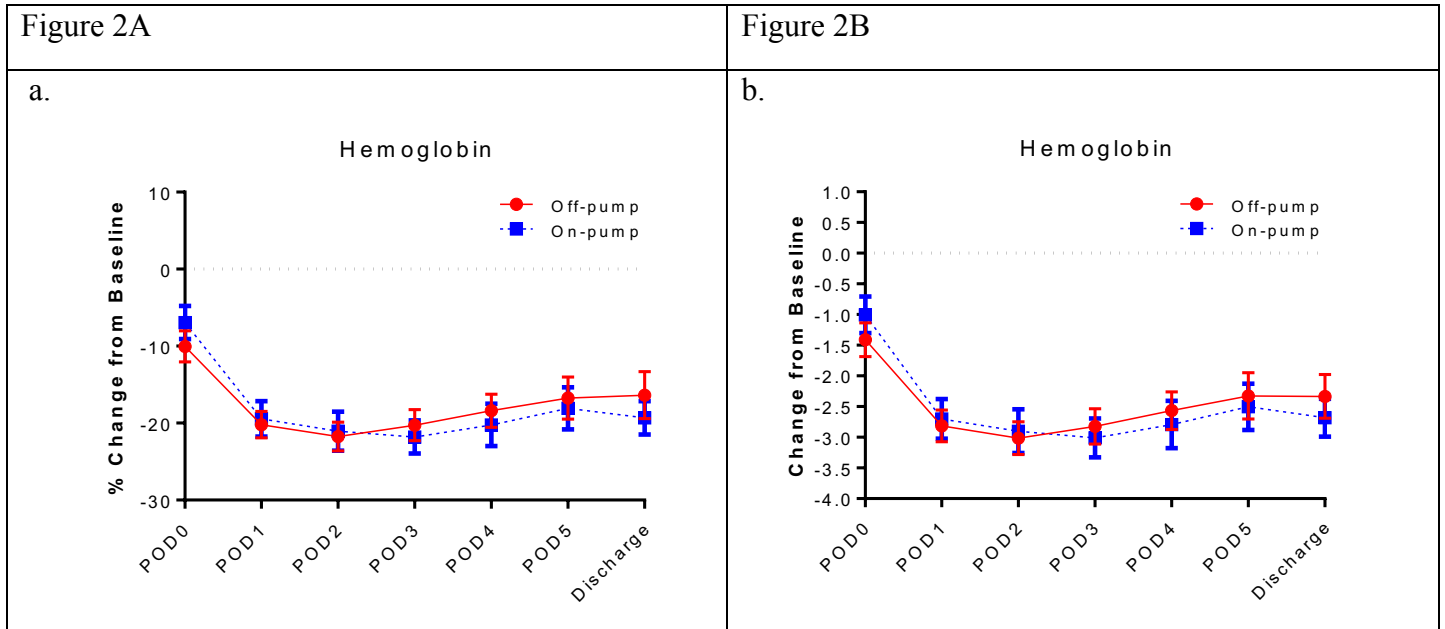
	OPCAB			ONCAB			
	median	min	max	median	min	max	P value
Baseline	13.6	7.9	17.5	13.3	7.2	18.2	0.15
Nadir	9.40	7.9	14.6	9.50	7.2	17.2	0.95
Discharge	10.60	7.9	15.4	10.30	7.2	19.3	.002

Table 4: Transfusion Patterns in ONCAB and OPCAB Surgeries

	OPCAB (n=255)	ONCAB (n = 379)	P-value
Red Blood Cells	79 (31%)	129 (34%)	0.45
Fresh frozen plasma	20 (8%)	69 (18.6%)	<0.001
Platelets	25 (10%)	131 (35%)	<0.001
Cryoprecipitate †	5 (2%)	8 (2%)	0.91
Cell Saver	204 (81%)	318 (85%)	0.22



Perioperative platelet counts profile and recovery. 1a (left) depict % change in platelet counts and figure 1b representing absolute changes in platelets from baseline. Note, similar trajectories in decline and postoperative recovery of platelet counts were observed in both groups. Further, nadir platelet counts was noted on postoperative day 2 in ONCAB and in OPCAB surgeries.



Perioperative hemoglobin values and recovery. 1a (left) depicts % change in hemoglobin and figure 1b representing absolute changes in hemoglobin from baseline. Note, similar trajectories in both decline in hemoglobin and postoperative recovery of anemia were observed in both groups. Further, nadir hemoglobin values were noted on postoperative day 2 for OPCAB and on POD4 for ONCAB surgery. Importantly, recovery from anemia was protracted and markedly incomplete at time of discharge.