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PLATELET ACTIVATION ACROSS THE CEREBRAL CIRCULATION IS ASSOCIATED WITH COGNITIVE DECLINE AFTER CABG SURGERY

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Introduction: Cardiac surgery with cardiopulmonary bypass (CPB) leads to cerebral embolization of particulate matter. In patients with atherothrombotic stroke, marked platelet activation characterized by surface expression of CD62P is a common finding. We therefore postulated that intraoperative platelet and leukocyte activation within the cerebral circulation would result in cognitive decline after coronary bypass (CABG) surgery.

Methods: Following IRB approval, 150 patients undergoing CABG surgery were enrolled. Patients were excluded if they had a history of symptomatic cerebrovascular disease, psychiatric illness, renal failure, active liver disease, bleeding disorders, less than a 7th grade education or were undergoing combined surgical procedures. Prior to induction of anesthesia, a retrograde right internal jugular venous catheter was successfully inserted into the jugular bulb of 125 of these patients. Arterial and jugular venous blood samples were simultaneously drawn prior to surgery, before cross-clamp release (XCR), 10 minutes after XCR, end of CPB, and end of surgery. Mean CD11b fluorescence and percentage of platelets expressing CD62P were determined on a flow cytometer as respective markers of leukocyte and platelet activation. Cognitive function was assessed preoperatively and 6 weeks after surgery with a battery of 6 tests. Cognitive deficit was defined by factor analysis as a decline of one standard deviation or more in at least 1 of 4 cognitive domains (dichotomous outcome). A change score (continuous outcome) was also calculated by subtracting the baseline from the follow-up sum of the 4 domain scores. Statistical analysis was conducted using the Spearman Rank Correlation and the Wilcoxon rank sum test; a P-value <0.05 was considered significant.

Results: In awake patients, platelets were activated to a greater degree in jugular venous blood samples than in the arterial circulation, suggesting an increase in platelet activation during passage through the cerebral circulation. At all other intraoperative time points, there was a net loss of activated platelets during transit through cerebral tissues was detected shortly after XCR ($p < 0.05$, Figure 1). Similarly, a significant correlation ($p = 0.005$) was seen between the continuous cognitive score and herein the patients demonstrating the largest cognitive decline experienced the greatest platelet activation after XCR.

Conclusions: An increase in platelet activation across the cerebral circulation following cross-clamp release is associated with cognitive decline after cardiac surgery. The appearance of activated platelets shortly after clamp release (when cerebral emboli are thought to be maximal) suggests the participation of these platelets in microvascular obstruction leading to cognitive dysfunction.

