

## EVALUATION OF THE EFFECT OF TWO DIFFERENT DOSES OF ASPIRIN IN PATIENTS UNDERGOING CORONARY ARTERY BYPASS GRAFT (CABG)

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**Introduction.** Aspirin is the most widely used antiplatelet agent that prevents the formation of TXA<sub>2</sub> by inhibition of COX-1. Despite clear benefit from aspirin (ASA) in patients with cardiovascular disease, evidence of heterogeneity in the individual response has given rise to the concept of aspirin failure to prevent a thrombotic event. In this study we investigate platelet function and the antiplatelet effect of ASA in 44 patients undergoing Coronary Artery Bypass Graft (CABG) and randomly assigned to 100 or 325 mg/d ASA treatment. **Methods.** Venous blood was collected before, 3 and 5 days after surgical intervention. Collagen induced aggregation of platelet rich plasma (PRP) and thromboxane B<sub>2</sub> levels in PRP and in serum were evaluated. **Results.** Before surgery the two groups of patients were comparable in terms of platelet aggregation and TXB<sub>2</sub> levels. After 2 days of ASA treatment, collagen-induced aggregation was inhibited by 38% in both groups of patients and a similar degree of inhibition was recorded 5 days after surgery. 3 days after surgery serum TXB<sub>2</sub> levels were reduced by 95% and 90% in patients receiving 325 or 100 mg/d ASA with residual TXB<sub>2</sub> of 10.8±2.2 and 24.6±3.7 ng/ml (p<0.02), respectively. No significant differences in the levels of TXB<sub>2</sub> were observed after 5 days. Measurement of TXB<sub>2</sub> in collagen stimulated PRP showed a 80% reduction in patients treated with 100 mg ASA and 95% in those treated with 325 mg/d. **Conclusions.** In patients undergoing CABG ASA 325mg/d led to an almost complete inhibition of TXB<sub>2</sub> production, whereas ASA 100mg/d showed a residual amount of this metabolite. Inhibition of platelet aggregation was comparable with the two treatments.