RAPID RECOVERY OF PLATELET FUNCTION AFTER CARDIOPULMONARY BYPASS

To the Editor:

The complex mechanism of altered platelet function during cardiopulmonary bypass (CPB) is a subject under investigation because it might result in understanding of blood-biomaterial interaction and, subsequently, optimal intervention. However, the common biochemical and functional parameters of platelets do not clarify the mechanism. Recently, new apparatuses were constructed to mimic the hemostatic mechanism by perfusing whole blood through a filter or a small hole at a specific shear. In the present study, we introduce another new technique for the in vitro bleeding test (Thrombostat 4000R; VDG-von der Goltz, Seeon, Germany, and Baxter Diagnostics AG, Dudingen, Switzerland) to investigate the change of platelet function during clinical CPB. From six patients, blood samples were collected in 0.3% sodium citrate at the time of anesthesia, 5 minutes before and 5 and 30 minutes after the start of CPB, 10 minutes before and 5 and 20 minutes after the end of CPB, and 30 minutes after the infusion of protamine. To investigate the effect of dilution to the measures, control experiments were performed with blood samples from four healthy volunteers. Blood used was undiluted or diluted similarly to CPB blood. Bleeding volume and time until capillary occlusion was measured in the apparatus, perfusing citrated whole blood through a capillary under constant pressure (40 mm Hg), into a cellulose acetate filter (ID 150 μm) covered with collagen type I, soaked with adenosine diphosphate solution (10 mmol/L).

According to the in vitro bleeding test, bleeding volume increased slightly \( (P < .05) \) after systemic heparinization, and increased abruptly thereafter \( (P < .01) \) during the first 5 minutes after the start of CPB, and remained high during CPB (Fig 1). This increase appeared to be mainly independent of hemodilution (Table 1). After the end of CPB, bleeding volume quickly decreased \( (P < .01) \) within 5 minutes and decreased further \( (P < .05) \) until 20 minutes. The administration of protamine did not influence the bleeding volume (Fig 1).

The superiority of the in vitro bleeding test to detect the abnormality of platelet function has been shown, although the results are somewhat affected by hemodilution. However, the rapid increase of bleeding volume at the start of CPB mainly represents the impairment of platelet function, because the separate experiment showed that hemodilution during CPB increases the bleeding volume only up to 40%. The rapid recovery of bleeding volume after CPB seems to reflect totally the recovery of platelet function, because the hematocrit did not significantly improve during this short period.

We previously reported the rapid decrease of platelet membrane receptor antigen in just 5 minutes after the start of CPB. Several in vitro experiments showed that the degradation and relocation of platelet membrane receptors take place due to plasmin and throm-

![Figure 1. Platelet function assessed by the in vitro bleeding test.](Image)
Interestingly, these receptors are rapidly replenished by the internal regulation after the cessation of plasmin attack. Therefore, the observation of the quick change of platelet function in the present study supports the hypothesis that temporary loss of functional platelet receptors is a main cause of platelet dysfunction during CPB.

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REFERENCES
Rapid recovery of platelet function after cardiopulmonary bypass [letter]

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