To the Editor:

The report by Kestin et al.1 made interesting and informative reading. The investigators' conclusion that the platelet function defect related to cardiopulmonary bypass (CPB) represents an extrinsic rather than an intrinsic platelet disorder is of considerable significance. In this regard, I would like to draw your attention to some additional data relevant to the subject.

Wenger et al.2 examining the effects of CPB on platelets in 10 patients, found a reduction in the number of fibrinogen binding sites from 31,730 ± 12,802 per platelet to 18,590 ± 9,644 per platelet. Bypass was also found to reduce the amount of platelet mem-
brane glycoprotein IIIa (GPIIIa), a part of the fibrinogen receptor complex, from 17.1 ± 33.6 ng/10 platelets to 12.9 ± 4.7 ng/10 platelets. This is in contrast to the investigators’ finding of unchanged surface expression of GPIIb-IIIa complex and fibrinogen binding sites during CPB.

Another recent study found an increase in monocyte-platelet conjugates from 18% ± 1.5% to 44% ± 4.5% during CPB. The role of this leukocyte-platelet adhesion induced by CPB on platelet function remains to be elucidated. Recent studies have also shown that the administration of aprotinin during extracorporeal circulation preserves platelets by protecting the platelet aggregating capacity. An improved understanding of the mechanisms for abnormal platelet function in CPB will no doubt translate into improved diagnostic and therapeutic maneuvers to reduce the hemorrhagic complications of this procedure in the future.

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REFERENCES
Platelet function defects related to cardiopulmonary bypass [letter; comment]

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