# LETTERS TO THE EDITOR

## The benefit of heparin-bound circuits

*To the Editor:*

We read with interest the recently published paper by Gorman and associates[1] on surface-bound heparin in extracorporeal circuits. The study was well conceived and conducted. Several questions remain, however. The first question concerns the appropriateness of including several of the patients in the study. Four of the 20 patients, two in each group, underwent pulmonary arterial thrombectomy. Circulatory arrest of relatively long duration was applied in all, and at low body temperatures, varying from 12° to 14.9° C. Extreme hypothermia can result in severe disturbances in coagulation.[2] The prolonged circulatory arrest in these patients suggests the presence of great quantities of thrombotic material. If these thrombi had been recently formed—and this was not stated in the paper—they may have resulted in significant consumption of coagulation factors. In our opinion these three patients should not have been included in the study.

Second, the authors state that a Cell Saver System (Haemonetics, Braintree, Mass.), was used in all patients, implying extensive contact between blood and uncoated synthetic material. Washing blood with normal saline solution results in removal of plasma, which would also greatly affect coagulation. In addition, no mention was made of how much blood per patient was so treated.

Third, experimental and clinical studies have shown that heparin-coated circuits yield some attenuation of inevitable damage to blood components, but that the difference with uncoated circuits is slight. We were therefore surprised that the study's conclusions correspond entirely with those of our animal experimental research,[3, 4] despite the possible shortcomings of the methods. Our study used a venovenous coated or uncoated circuit with low flow in unheparinized dogs. We, too, found some protection of circulating blood by coated circuits, and we also found no reduction in blood coagulability. We therefore agree with the authors' conclusion that reducing the dose of heparin when a heparin-coated system is used, as is advocated by some,[5] is unwise and even dangerous.

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**12/8/74049**

*Reply to the Editor:*

We appreciate Drs. Moulijn and Amsel's interest in our study and are happy to clarify the points raised that may also have concerned others. The patients were consecutive to reduce any possibility of bias. The patients with pulmonary thromboendarterectomy were included primarily to permit the heparin-coated circuits to demonstrate their benefits, if any, by increasing the duration of perfusion and therefore blood trauma. The prothrombin fragment F1.2 measurements at the end of cardiopulmonary bypass for the four patients having thromboendarterectomy were 6.22, 5.63, 5.19, and 5.51 mmol/L. Data from these four patients did not exceed the range of the other patients at any time for any variable studied. In addition, statistical analysis of the data after exclusion of these patients did not change our results or conclusions. Concern that these patients' pulmonary arteries contained large quantities of fresh thrombus that "resulted in significant consumption of coagulation factors" is unfounded. All these patients had the syndrome of chronic pulmonary thromboembolism.

The lack of heparin coating on the Cell Saver System (Haemonetics, Braintree, Mass.) is irrelevant to the hypothesis tested. The same system was used in both groups and only packed red blood cells were returned to the patients. Any coagulation factors activated by contact with the Cell Saver System would have been discarded before reinfusion.

We are sorry that Drs. Moulijn and Amsel find shortcomings in our methodology. In defense, we point out that a large number of quantitative measurements of platelet, neutrophil, and plasma protein activation were assessed. Although we found that heparin coatings increase adsorbtion of antithrombin III, affect complement activation, and increase platelet and monocyte adhesion, we did not demonstrate an anticoagulant effect.

The results of our study, Wagner's study, Moulijn and Amsel's study, and an increasing number of others concur that heparin-coated perfusion circuits do not reduce thrombin formation or activity. The use of these circuits without full systemic heparization subjects patients to the increased risk of thromboembolic complications associ-