Influence of modified ultrafiltration on coagulation, fibrinolysis and blood loss in adult cardiac surgery


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Abstract

Objectives: Modified ultrafiltration (MUF) significantly reduce blood loss and transfusion requirements in pediatric cardiac surgery presumably by a reduction in inflammatory mediators which decrease the inflammatory axes and decrease the cross-activation of fibrinolysis and thrombosis. The influence of MUF on blood loss and homologous blood transfusion in adult cardiac surgery has not yet been determined. Furthermore, data about the influence on routine coagulation tests, platelet activation as well as the coagulation and fibrinolytic systems are limited.

Methods: In a prospective randomized study 48 patients scheduled for elective myocardial revascularization were randomized into a control group (n = 16), a conventional ultrafiltration (CUF) group (n = 16) and a MUF group (n = 16). Perioperatively, serial blood samples were drawn at specific intervals to evaluate coagulation, fibrinolysis, and platelet function.

Results: Neither the coagulation nor the fibrinolytic system was positively influenced by MUF or CUF. The routine clotting tests were comparable except for a significantly higher antithrombin III activity after MUF compared to the CUF control group persisting 24 h postoperatively. Platelet factor 4 activity and platelet counts showed no differences among the groups. MUF considerably reduced the postoperative blood loss (MUF, 6.4 ± 1.7 ml/kg bw per 24 h vs. CUF, 9.2 ± 2.5 ml/kg bw per 24 h (P = 0.003) vs. control, 8.9 ± 2.2 ml/kg bw per 24 h (P = 0.008)) and allogeneic blood transfusion (MUF, 2.0 ± 3.4 ml/kg bw per 24 h vs. CUF, 6.9 ± 5.1 ml/kg bw per 24 h (P = 0.034) vs. control, 7.0 ± 6.3 ml/kg bw per 24 h (P = 0.029)).

Conclusions: MUF in adult cardiac surgery significantly reduces postoperative blood loss and transfusion requirements. The mechanism for reduced blood loss could not be elucidated in this study.

Keywords: Cardiopulmonary bypass; Blood loss; Antithrombin III; Modified ultrafiltration; Adult cardiac surgery

1. Introduction

Hemostatic changes induced by cardiopulmonary bypass (CPB) are one of the major factors responsible for increased postoperative blood loss. The reason for coagulation abnormalities after CPB are multifactorial. Coagulation factors are decreased, the platelet function and number is reduced and an imbalance of the coagulation and fibrinolytic systems occurs [1–6]. Various pharmacological and non-pharmacological strategies have been introduced to reduce the sequel of CPB-induced hemostatic changes [7–12].

In pediatric cardiac surgery, modified ultrafiltration (MUF) improves hemostasis after CPB with the beneficial effect on postoperative bleeding and the need for blood transfusion [13–17]. The underlying mechanisms of these effects are not well established. To our knowledge no data are available concerning the influence of MUF on hemostasis after CPB in adult cardiac surgery.

The objective of the present study was to analyze the coagulation and fibrinolytic systems to delineate the impact of MUF and to assess the possible influence of MUF on postoperative blood loss and transfusion requirements in adult cardiac surgery.

2. Material and methods

2.1. Patients

The study protocol was approved by the local ethics committee of the Medical University of Lübeck. Written informed consent was obtained from each patient. Forty-eight patients with stable angina and preserved left ventricular function (EF > 45%) undergoing primary elective coronary artery bypass grafting were studied. Exclusion criteria were an age of >75 years, administration of acetylsalicylic acid within the last 7 days prior to surgery, a platelet count below 150 000/µl, and a documented or
reported coagulopathy assessed by routine coagulation parameters and patient history. The patients were prospectively randomized into a control group (Con.; \( n = 16 \)), a conventional ultrafiltration (CUF) group (\( n = 16 \)) and a MUF group (\( n = 16 \)). Patient characteristics are presented in Table 1.

### 2.2. CPB

Anesthesia was induced with etomidate (0.3 mg/kg bw) and sufentanil (0.8 \( \mu \)g/kg bw). Pancuronium bromide was used for neuromuscular blockade. Anesthesia was maintained with continuous administration of sufentanil and propofol during the operation. CPB materials and methods were the same in all patients: single aortic and right atrial cannulation technique, Stöckert™ roller pumps (Stöckert Instruments, Munich, Germany) and membrane oxygenators (Spiralox™, Bentley® Laboratories; Baxter Germany, Unterschleissheim, Germany) primed with 1125 ml of Ringer’s lactate, 250 ml of albumin 5% and 125 ml of manitol 20% were used. A non-pulsatile pump flow was maintained at 2.4 l/min per m\(^2\) with a perfusion pressure sustained at 20% were used. A non-pulsatile pump flow was maintained.

### 2.3. Ultrafiltration techniques

CUF was started during rewarming and continued until the end of CPB. MUF was carried out in the first 15 min after cessation of CPB. In both ultrafiltration groups a HQ 7000 ultrafilter (Baxter Bentley, Baxter Healthcare Corporation, Irvine, CA, USA) was placed with the inlet connected to the arterial line and the outlet connected to the venous line. The different ultrafiltration techniques are described in detail elsewhere [13–17].

#### 2.3.1. MUF

In brief, when the patient is weaned from CPB arterial and venous lines are kept in situ. The inlet of the ultrafilter is unclamped and arteriovenous ultrafiltration is carried out. The blood flow through the ultrafilter is about 300 ml/min, which is maintained by a Stöckert roller pump on the inlet aspect of the ultrafilter. The blood is directly returned to the right atrium. When the level in the venous reservoir is low, crystalloid is added to the venous reservoir to keep it primed. Ultrafiltration is continued until the intraoperative fluid balance is left at plus 500 ml to accommodate blood loss and compensate hypovolemia and until the bypass circuit is completely cleared of blood.

#### 2.3.2. CUF

In brief, during rewarming the filtration is started with a rate adjusted to reach the cardiotomy reservoir approaching zero at the termination of CPB. The ultrafiltered blood is directly returned to the venous reservoir. Residual blood from the CPB circuit after termination of CPB is retransfused to the patient before leaving the operation room.

### 2.4. Coagulation and fibrinolytic measurements

Venous blood samples were drawn immediately after induction of anesthesia, 20 min after administration of heparin, after termination of CPB, 20 min after administration of protamine sulfate, and 6 and 24 h after completion of CPB. Samples were drawn in 10 ml EDTA or citrate monovettes vials from the central venous catheter. For the platelet factor 4 (PF 4) assay blood was collected into vials (Diatube®, Stago, Asniere, France) which were placed on ice. Routine parameters (complete blood count, platelet count, fibrinogen, antithrombin III activity (AT III), thrombin time (TT), activated partial thromboplastin time (aPTT), prothrombin time (PT), and ACT), and parameters assessing the coagulation and fibrinolytic system (concentration of thrombin–antithrombin complex (TAT), prothrombin fragments (F1 + 2), fibrinmonomers (FM), tissue plasminogen activator (t-PA), fibrin degradation products (D-dimer), and plasmin–antiplasmin complex (PAP)) were measured. In 36 patients (12 patients in each group) routine parameters and parameters assessing the coagulation and fibrinolytic system were measured. In the remaining 12 patients (four patients in each group) only routine parameters were evaluated.

### 2.5. Assays

Complete blood count, platelet count, PT, aPTT, ACT, AT III, and fibrinogen were obtained according to standard
methods. Enzyme-linked immunosorbent assays (ELISA) for FⅠ + 2, TAT and t-PA were obtained from Dade-Behring (Marburg, Germany), ELISA kits for D-dimer and PF 4 were obtained from Boehringer (Mannheim, Germany) and the ELISA for t-PA was obtained from Chromogenix (Mölndal, Sweden). FM were assessed by a quantitative spectrophotometric assay (Behringwerke AG, Marburg, Germany). The aforementioned ELISAs and spectrophotometric analyses were performed according to the manufacturer’s instructions. All post-bypass data were corrected for hemodilution.

### 2.6. Blood loss measurements and transfusion criteria

Postoperatively, allogeneic blood was transfused if the hematocrit was less than 25% or if the hemoglobin concentration was less than 80 g/dl. Intraoperatively as well as postoperative blood loss in the first 24 h was measured. Intraoperative blood loss was determined by measuring the blood collected in the suction system during the operation, and postoperative blood loss was determined by measuring the blood collected in the suction system within the first 24 h after the operation. The total volume of transfused packed red cells, fresh frozen plasma, platelets and colloid solution were recorded.

### 2.7. Statistical methods

All values are presented as the mean ± standard deviation of the mean (SD) unless otherwise indicated. Comparisons between groups were performed by use of ANOVA for repeated measures design. Differences in blood loss and blood requirements were analyzed with a one factor (group) ANOVA. If statistically significant (P < 0.05), a post-hoc analysis with the Scheffe test was performed. A probability value of less than 0.05 was considered statistically significant. Statistical analysis was performed without alpha adjustments and therefore results are considered mainly exploratory [18]. Statistical procedures were done with SPSS for windows (SPSS Inc., Chicago, IL, USA).

### 3. Results

Demographic and operative data of the different groups were comparable, except for a significant longer aortic cross-clamp time in the MUF group compared with the control group (Table 1).

Thrombin formation during CPB took place in all patients and was not influenced by the mode of ultrafiltration (FⅠ + 2, time P < 0.001, group P > 0.1; TAT, time P < 0.001, group P > 0.1; FM, time P < 0.001, group P > 0.1) (Table 2). Table 3 illustrates the activation of the fibrinolytic system induced by CPB (t-PA, time P < 0.001, group P > 0.1; PAP, time P < 0.001, group P > 0.1; D-dimer, time P < 0.001, group P > 0.1). Neither modified ultrafiltration nor CUF had an influence on the fibrinolytic system.

The routine clotting parameters PT, aPTT, TT, fibrinogen and ACT were influenced by the time (PT, P < 0.001; aPTT, P < 0.001; TT, P < 0.001; fibrinogen, P < 0.001; ACT, P < 0.001) but not by the group (PT, P > 0.1; aPTT, P > 0.1; TT, P > 0.1; fibrinogen, P > 0.1; ACT, P > 0.1). The AT III concentration decreased significantly over time in all groups (P > 0.0001), and was significantly influenced by the group (P = 0.004). The AT III activity was significantly higher in the MUF group than in the CUF and control groups after administration of protamine sulfate and remained significantly higher throughout the observation period of 24 h (Fig. 1).

The post-bypass platelet counts decreased significantly below pre-bypass values in the three groups (P < 0.001) and the PF 4 levels increased over pre-bypass values (P < 0.001). However, no differences between the groups

### Table 2

Comparison of parameters assessing the perioperative activation of the coagulation systema

<table>
<thead>
<tr>
<th></th>
<th>Preop</th>
<th>20 min post-heparin</th>
<th>Post-CPB</th>
<th>20 min post-protamine sulfate</th>
<th>6 h post-CBP</th>
<th>24 h post-CBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>FⅠ + 2 (nmol/l) (n)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>MUF (12)</td>
<td>1.6 ± 0.9</td>
<td>1.0 ± 0.5</td>
<td>2.9 ± 2.2</td>
<td>3.7 ± 1.8</td>
<td>1.7 ± 0.5</td>
<td>1.0 ± 0.5</td>
</tr>
<tr>
<td>CUF (12)</td>
<td>1.9 ± 0.7</td>
<td>2.3 ± 2.4</td>
<td>2.8 ± 1.3</td>
<td>3.9 ± 1.5</td>
<td>2.0 ± 0.9</td>
<td>1.0 ± 0.4</td>
</tr>
<tr>
<td>Control (12)</td>
<td>1.3 ± 0.6</td>
<td>1.7 ± 2.2</td>
<td>3.2 ± 2.4</td>
<td>3.4 ± 2.1</td>
<td>2.1 ± 1.0</td>
<td>0.9 ± 0.4</td>
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<tr>
<td>TAT (μg/l) (n)</td>
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<tr>
<td>MUF (12)</td>
<td>18.1 ± 9.2</td>
<td>18.9 ± 28.5</td>
<td>64.0 ± 30.4</td>
<td>46.7 ± 20.5</td>
<td>14.3 ± 5.3</td>
<td>14.3 ± 23.2</td>
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<tr>
<td>CUF (12)</td>
<td>18.7 ± 10.2</td>
<td>13.4 ± 15.1</td>
<td>56.6 ± 35.5</td>
<td>44.9 ± 17.0</td>
<td>14.0 ± 4.4</td>
<td>8.3 ± 4.8</td>
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<td>Control (12)</td>
<td>16.9 ± 7.5</td>
<td>11.0 ± 6.6</td>
<td>67.1 ± 33.7</td>
<td>58.4 ± 34.2</td>
<td>19.4 ± 10.3</td>
<td>10.5 ± 7.2</td>
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<tr>
<td>FM (mg/l) (n)</td>
<td></td>
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<tr>
<td>MUF (12)</td>
<td>22.9 ± 6.9</td>
<td>20.0 ± 11.4</td>
<td>24.1 ± 13.2</td>
<td>22.1 ± 6.9</td>
<td>26.4 ± 10.8</td>
<td>37.7 ± 9.0</td>
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<td>CUF (12)</td>
<td>23.5 ± 5.4</td>
<td>17.2 ± 3.9</td>
<td>19.4 ± 4.6</td>
<td>17.6 ± 5.7</td>
<td>22.4 ± 7.6</td>
<td>38.9 ± 13.1</td>
</tr>
<tr>
<td>Control (12)</td>
<td>17.6 ± 6.5</td>
<td>18.8 ± 12.4</td>
<td>20.3 ± 11.8</td>
<td>17.6 ± 9.0</td>
<td>22.4 ± 11.9</td>
<td>33.4 ± 9.5</td>
</tr>
</tbody>
</table>

Data are presented as the mean ± standard deviation of the mean. There were no significant differences among groups. CPB, cardiopulmonary bypass; FⅠ + 2, prothrombin fragments; MUF, modified ultrafiltration; CUF, conventional ultrafiltration; TAT, thrombin–antithrombin complex; FM, fibrinmonomers.

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were obvious (platelet count, $P > 0.1$; PF 4, $P > 0.1$) (Table 4).

The hematocrit decreased significantly over time ($P < 0.001$) and was influenced by ultrafiltration techniques ($P = 0.045$). The hematocrit was significantly greater in the MUF group 20 min after administration of protamine sulfate compared to CUF and controls and in the CUF group 20 min after administration of protamine sulfate compared to the control group (MUF vs. CUF, $P = 0.01$; MUF vs. Con., $P < 0.001$; CUF vs. Con., $P = 0.019$). However, these differences subsided in the postoperative course (Fig. 2).

MUF resulted in a significantly reduced perioperative blood loss (MUF vs. CUF, $P = 0.008$; MUF vs. Con., $P = 0.003$) and a reduced need for transfusion of packed red cells (MUF vs. CUF, $P = 0.029$; MUF vs. Con., $P = 0.034$) (Fig. 3). Neither of the groups received platelets or fresh frozen plasma transfusion.

There were no differences among the groups regarding volume substitution with plasma and colloid solution, the urine output, the dosages of diuretics and inotropic support.

4. Discussion

The preliminary results of this study demonstrated that MUF applied in adult cardiac surgery significantly reduced the postoperative blood loss and allogeneic red blood cell transfusions. The time course of the activated coagulation and fibrinolytic parameters typical for CPB was not influenced by MUF.

In pediatric cardiac surgery, MUF reduces blood loss and blood transfusion requirements. The mechanisms responsible for the reduced blood loss and blood product requirements have to date not been clearly delineated. Naik et al. [13] showed that the bypass flow and temperature has a significant influence on blood loss but not on transfusion requirements in infants and neonates after MUF.

Concordant to MUF applied in pediatric cardiac surgery, our results demonstrate that application of this new technique in adults reduces blood loss and blood transfusion requirements. Since there were no obvious differences in the amount of postoperatively given colloids among the groups, the significantly reduced allogeneic blood transfusions may be related to the immediately higher postoperative hematocrit approximating the preoperative value in conjunction with reduced postoperative blood loss. The mechanisms for blood loss reduction are difficult to define.

**Table 3**

<table>
<thead>
<tr>
<th></th>
<th>Preop</th>
<th>20 min post-heparin</th>
<th>Post-CPB</th>
<th>20 min post-protamine sulfate</th>
<th>6 h post-CPB</th>
<th>24 h post-CPB</th>
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<tbody>
<tr>
<td>t-PA (µg/ml)</td>
<td></td>
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<tr>
<td>MUF (12)</td>
<td>10.1 ± 4.0</td>
<td>9.3 ± 4.4</td>
<td>11.8 ± 6.8</td>
<td>14.3 ± 5.4</td>
<td>15.1 ± 5.1</td>
<td>12.2 ± 4.1</td>
</tr>
<tr>
<td>CUF (12)</td>
<td>6.8 ± 3.2</td>
<td>9.4 ± 4.1</td>
<td>15.3 ± 8.2</td>
<td>14.2 ± 7.2</td>
<td>15.7 ± 5.6</td>
<td>11.7 ± 5.5</td>
</tr>
<tr>
<td>Control (12)</td>
<td>10.4 ± 4.1</td>
<td>10.1 ± 3.8</td>
<td>15.1 ± 5.3</td>
<td>16.8 ± 3.7</td>
<td>19.8 ± 4.1</td>
<td>12.7 ± 6.0</td>
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<tr>
<td>PAP (µg/ml)</td>
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<tr>
<td>MUF (12)</td>
<td>547 ± 296</td>
<td>501 ± 307</td>
<td>1174 ± 865</td>
<td>1349 ± 805</td>
<td>1161 ± 610</td>
<td>239 ± 123</td>
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<tr>
<td>CUF (12)</td>
<td>616 ± 205</td>
<td>624 ± 160</td>
<td>1640 ± 649</td>
<td>1850 ± 834</td>
<td>1532 ± 865</td>
<td>323 ± 168</td>
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<tr>
<td>Control (12)</td>
<td>539 ± 284</td>
<td>487 ± 286</td>
<td>1339 ± 635</td>
<td>1522 ± 593</td>
<td>1269 ± 567</td>
<td>320 ± 194</td>
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<tr>
<td>D-dimer (µg/ml)</td>
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<tr>
<td>MUF (12)</td>
<td>547 ± 512</td>
<td>480 ± 465</td>
<td>827 ± 549</td>
<td>867 ± 615</td>
<td>473 ± 245</td>
<td>406 ± 224</td>
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<tr>
<td>CUF (12)</td>
<td>527 ± 156</td>
<td>583 ± 301</td>
<td>1019 ± 408</td>
<td>1102 ± 563</td>
<td>650 ± 283</td>
<td>413 ± 121</td>
</tr>
<tr>
<td>Control (12)</td>
<td>393 ± 322</td>
<td>358 ± 207</td>
<td>828 ± 704</td>
<td>606 ± 506</td>
<td>557 ± 357</td>
<td>539 ± 285</td>
</tr>
</tbody>
</table>

*a* Data are presented as the mean ± standard deviation of the mean. There were no significant differences among groups. CPB, cardiopulmonary bypass; t-PA, tissue plasminogen activator; MUF, modified ultrafiltration; CUF, conventional ultrafiltration; PAP, plasmin–antiplasmin complex; D-dimer, fibrin degradation products.
Naik et al. demonstrated that MUF only reduces blood loss in pediatric patients with low flow (>25% of CPB time at ≤0.6 l/m² per min) deep hypothermic (17–22°C) CPB. In contrast, the full flow (2.4 l/m² per min) moderate hypothermic (24–28°C) bypass technique did not reduce postoperative blood loss [13]. However, we observed a significant reduction in postoperative blood loss after MUF in a setting with a full flow (2.4 l/m² per min) moderate hypothermic (28°C) bypass technique. Overall, the benefit of MUF to reduce postoperative blood loss and particularly blood requirements is more pronounced in pediatric cardiac surgery [13]. This could be causally related to the different ratio of priming volume to total body fluid volume in infants and neonates compared to adults.

Ultrafiltration has the capability to remove inflammatory mediators [19–21]. Journois et al. [22] suggested that the reduced postoperative blood loss after ultrafiltration is unlikely to be exclusively explained by hemoconcentration but could be related to removal of mediators of the inflammatory response induced by CPB. Mediators of the inflammatory response are essential for the initiation and maintenance of coagulation disturbances and increased fibrinolysis associated with CPB [23]. Thus, any pharmacologic or other method avoiding or reducing the formation of mediators may influence the coagulation and fibrinolytic systems. Elevated levels of TAT, F1 + 2 and FM indicating thrombin generation were not altered by any type of ultrafiltration. The same applied to the fibrinolytic system with increased levels of t-PA, PAP and D-dimer indicating fibrinolysis. Thus, neither changes in the coagulation nor the fibrinolytic systems can be causally related to the reduced blood loss after MUF.

Neglecting the CPB temperature and flow-dependent differences in blood loss, Naik et al. [13] speculated that coagulation factors and platelets are concentrated by ultrafiltration leading to improved clotting conditions. In this study we found no differences in platelet counts within the groups. These results are partially supported by Journois et al. [22] who found no changes in platelet count after CUF. Furthermore, the PF 4 concentration was not affected by ultrafiltration. Platelet activation was not influenced by ultrafiltration, implying that irreversible secondary platelet aggregation is responsible for 98.4% of PF 4 found in plasma [24].

Routine coagulation tests and parameters (PT, aPTT, TT, ACT, fibrinogen) as indicators of coagulation abnormalities and coagulation factor deficiencies were not affected by any type of ultrafiltration.

Significantly higher AT III levels were associated with MUF. However, whether elevated AT III levels contribute to the observed reduced postoperative blood loss has to be elucidated. From our data the higher AT III levels after MUF cannot be explained by hemoconcentration only. A reduction of AT III in association with CPB was linked to an increased serum elastase level as demonstrated by Cohen.

Table 4
Comparison of perioperative platelet counts and PF 4 levels

<table>
<thead>
<tr>
<th></th>
<th>Preop</th>
<th>20 min post-heparin</th>
<th>Post-CPB</th>
<th>20 min post-protamine sulfate</th>
<th>6 h post-CPB</th>
<th>24 h post-CPB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count (10⁹/l) (n)</td>
<td></td>
<td></td>
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<tr>
<td>MUF (16)</td>
<td>244 ± 82</td>
<td>238 ± 90</td>
<td>174 ± 48</td>
<td>163 ± 50</td>
<td>175 ± 47</td>
<td>168 ± 51</td>
</tr>
<tr>
<td>CUF (16)</td>
<td>235 ± 87</td>
<td>241 ± 82</td>
<td>170 ± 66</td>
<td>166 ± 62</td>
<td>174 ± 66</td>
<td>166 ± 59</td>
</tr>
<tr>
<td>Control (16)</td>
<td>227 ± 64</td>
<td>231 ± 66</td>
<td>167 ± 47</td>
<td>163 ± 46</td>
<td>165 ± 43</td>
<td>163 ± 51</td>
</tr>
<tr>
<td>PF 4 (μg/l) (n)</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>MUF (16)</td>
<td>17.1 ± 19.4</td>
<td>20.9 ± 12.4</td>
<td>32.8 ± 8.8</td>
<td>19.0 ± 9.3</td>
<td>21.9 ± 17</td>
<td>17.7 ± 18.9</td>
</tr>
<tr>
<td>CUF (16)</td>
<td>27.1 ± 37.7</td>
<td>38.1 ± 29.3</td>
<td>51.1 ± 16.1</td>
<td>29.1 ± 13.4</td>
<td>25.6 ± 22.2</td>
<td>12.6 ± 11.2</td>
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<tr>
<td>Control (16)</td>
<td>11.7 ± 8.4</td>
<td>23.6 ± 31.9</td>
<td>53.5 ± 53.4</td>
<td>33.7 ± 35.3</td>
<td>24.3 ± 17.4</td>
<td>14.8 ± 17.6</td>
</tr>
</tbody>
</table>

*Data are presented as the mean ± standard deviation of the mean. There were no significant differences among groups. CPB, cardiopulmonary bypass; MUF, modified ultrafiltration; CUF, conventional ultrafiltration; PF 4, platelet factor 4.
et al. [25]. Jordan et al. [26] showed that human neutrophil elastase catalyses the inactivation of AT III. We did not determine the influence of MUF on postoperative neutrophil elastase levels. Therefore, it remains speculative whether MUF in adults causes changes in neutrophil elastase which might subsequently influence AT III levels.

Several limitations of this study should be noted. First, the influence of MUF on platelet function was not determined and remains to be established. Second, individual coagulation factors were not analyzed, except for fibrinogen. Since the global coagulation parameters (PT, aPTT, TT, ACT) were comparable among the groups a decrease of a single coagulation factor sufficient enough to explain the difference in blood loss remains questionable. Third, a general handicap was that the surgeon was not blinded to the mode of management. However, blinding was practically impossible since the mode and timing of intervention was dependent on the active cooperation of the surgeon. Finally, the small number of patients in each group make it difficult to draw definite conclusions about the clinical importance of MUF in adult cardiac surgery.

In conclusion, our preliminary data provide some evidence that MUF applied in adult cardiac surgery reduces the postoperative blood loss with a subsequent reduction of allogeneic red blood cell transfusions. Whether the increased AT III levels after MUF are a factor partly responsible for the reduced postoperative blood loss remains to be investigated. Further randomized prospective assessments of the technique of MUF in adults with a larger numbers of patients are desirable to delineate the clinical importance of this new technique.

References