

Single Atrio caval Cannulation is Associated with Increased Incidence of Hypercirculatory Failure after Cardiopulmonary Bypass

Thomas Busch, MD, PhD,¹ Horia Sirbu, MD, FETCS,¹ Stephan Kazmaier, MD,² Ivan Aleksic, MD, FETCS,¹ Martin Friedrich, MD,¹ Harald Dalichau, MD, PhD,¹ and Wolfgang Buhre, MD²

Cardiopulmonary bypass (CPB) can lead to hypercirculatory cardiac failure (HCF). Despite the activation of inflammatory mediators, the infusion of cardioplegic solution into the systemic circulation may result in decreased systemic vascular resistance and thus may cause HCF. The present prospective study was conducted to investigate in cardiac surgical patients, the effects of single atrial versus bi-caval venous drainage and intraoperative hemofiltration on the incidence of HCF.

Methods and Results: 120 patients undergoing coronary artery bypass surgery (CABG) were randomized in 3 groups: A- single atrial cannulation; B- single atrial cannulation and intraoperative zero fluid balance hemofiltration; C- bi-caval cannulation. Myocardial protection was performed using cold crystalloid cardioplegia (Bretschneider's HTK) administered into the aortic root and moderate hypothermia (32°C). Hemodynamics, fluid balance, vasoactive drugs, body temperature, and hemoglobin/hematocrit ratio were recorded during and up to 12 hours after surgery. We noted a significantly increased incidence of HCF in-group A (32%, n=13) and B (40%, n=16) when compared to group C (10%, n=4, p<0.05), with significantly increased requirements for vasoactive medication in patients developing HCF.

Conclusion: The present study results demonstrate that single atrial cannulation is associated with a significantly higher incidence of HCF. This is presumably caused by infusion of cardioplegic solution into the systemic circulation. (*Ann Thorac Cardiovasc Surg* 2001; 7: 210–5)

Key words: hyperdynamic failure, systemic inflammatory response syndrome, extracorporeal circulation, venous cannulation

Introduction

Hypercirculatory failure (HCF) is a severe complication after cardiac surgery with the use of cardiopulmonary bypass (CPB).¹⁻³⁾ The reported incidence of HCF after

CPB ranges between 8 to 10%.²⁾ There is a growing evidence that hypercirculatory response after cardiac surgery is caused by the activation of different mediator systems like cytokines, complement, tumor necrosis factor, bradykinin/kallikrein system and is similar to the systemic inflammatory response syndrome (SIRS).¹⁻³⁾ The question if a SIRS-like reaction is induced per se during cardiac operation remains unsolved.¹⁾ In most studies the cytokine levels were found to be increased in patients with no clinical signs of HCF.¹⁾ Different cardioplegic strategies and the use of individual venous drainage techniques during CPB may additionally be the cause of peripheral vasodilatation, potentially leading to HCF.^{2,4-7)}

From ¹Klinik für Thorax-, Herz- und Gefäßchirurgie, and ²Zentrum Anaesthesiologie, Rettungs- und Intensivmedizin, Georg-August Universität, Göttingen, Germany

Received December 4, 2000; accepted for publication February 14, 2001.

Address reprint requests to Thomas Busch MD: Klinik für Thorax-, Herz- und Gefäßchirurgie, Georg-August-University Göttingen, Robert Koch Strasse 40, D-37075 Göttingen, Germany.

In a retrospective analysis performed at our institution we observed an 8% incidence of HCF in the 1200 patients investigated. In the majority of these patients, venous drainage was performed using single-atrial venous cannulation, a technique which is inevitably associated with the infusion of significant amounts of cardioplegic solution into the systemic circulation.⁴⁾

The present prospective study was undertaken to investigate if the different venous drainage techniques during CPB and the systemic infusion of cardioplegic solution are related with a higher incidence of HCF in patients undergoing coronary artery bypass grafting (CABG). In addition we studied the role of the intraoperative hemofiltration on the incidence of HCF.

Material and Methods

After approval of the local institutional human research committee and written informed consent, 120 patients undergoing elective CABG surgery were randomized. Exclusion criteria were left ventricular ejection fraction (EF) <40%, concomitant cardiac valve disease, diabetes, peripheral arterial vascular disease, and severe pulmonary, renal and/or hepatic failure. The patients were divided into three groups according to the type of venous cannulation and the use of hemofiltration. Group A: mono-atrial venous cannulation (DLP, 46C, Medtronic 91246 C) and systemic circulation of the cardioplegic solution after coronary administration, group B: mono-atrial venous cannulation and intraoperative zero-fluid balanced hemofiltration (Jostra BCA 140) of the cardioplegic solution, and group C: bi-caval venous cannulation (Stöckert, 36F, V122-36) with complete removal of the cardioplegic solution from the right atrium.

Prior to anaesthesia induction, hemodynamic monitoring was established including electrocardiography (ECG), arterial, central venous and pulmonary arterial catheterisation. Anaesthesia was performed using a modified opioid-benzodiazepine technique. The following hemodynamic parameters were continuously monitored (Sirecust 1281, Siredoc 220, Siemens, Germany): heart rate (HR), mean arterial pressure (MAP), central venous pressure (CVP) and pulmonary arterial pressure (PAP).

Thermodilution measurements of cardiac output (CO), the calculation of systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) were performed intermittently before induction of anesthesia (I), pre- and post bypass (II, III), after sternal closure (IV), after arrival at the intensive care unit (ICU) (V) and hourly the

first twelve postoperative hours (VI-XIV). In addition, mixed venous oxygen saturation, serum sodium and potassium, hemoglobin and hematocrit were monitored during the entire study period. The cumulative fluid balance, the use and dosage of the vasoactive medication (norepinephrine, epinephrine, nitroglycerine) were additionally recorded online. The postoperative therapy was performed by physicians not involved in this study and blinded to the type of venous drainage performed.

The CPB-priming was standardized and consisted of 1000 ml Ringer's-lactate-solution, 500 ml hydroxyethylstarch 6% and 7500 I.U. heparin. Surgery was performed under moderate hypothermia (32°C), using roller pumps (Polystan) and membrane oxygenators (Jostra, Quadrox). During the CPB a non-pulsatile pump flow rate of 2.4 L*min⁻¹ *m⁻² was maintained in all patients. A hematocrit of 20-24% was constantly monitored during CPB. The CPB-acid base status was performed according to the pH-stat mode.

Combined myocardial protection was performed by infusion of 2000 ml Bretschneider-HTK solution (Custodiol®) at 4°C via a needle-vent (Research Medical, Inc., ATC-011-MV) in the aortic root and the use of saline topical cooling. In groups A and B, the cardioplegic solution entered the systemic circulation. In group C the cardioplegic solution was removed from the right atrium as complete as possible. In group B hemofiltration was performed once CPB was initiated and a total of 2000 ml ultrafiltrate was filtrated during the extracorporeal circulation.

HCF failure was defined as the simultaneous presence of CO >6L*min⁻¹, SVR <800 dyn*sec*m⁻⁵ and a MAP <60 mmHg.

All table and figure data are given as mean ± standard deviation, unless stated otherwise. The effects of the different venous drainage techniques were tested by multiple analyses of variance for repeated measurements (MANOVA) using the statistical package Statistica 3.1. (Statsoft Inc.). Post-hoc testing within each group and between groups was performed when a significant interaction between the repeated measures factor and the type of venous drainage was observed. Student's t-test was used for post-hoc comparison. A level of p<0.05 was considered significant.

Results

Each group consisted of 40 patients. The groups were comparable with respect to biometric data, time of is-

Table. Biometrical, surgery related data and the length of ICU stay

Group	Group	A	B	C
Age	(years)	63 ± 2	65 ± 2	64 ± 1
Body weight	(kg)	75 ± 8	76 ± 6	75 ± 7
Body height	(cm)	176 ± 5	179 ± 3	177 ± 6
Gender	men	18	21	17
	women	22	19	23
No. of grafts	(n)	4	5	4
Ischemia	(min)	67 ± 4	68 ± 3	70 ± 3
Perfusion	(min)	47 ± 7	49 ± 2	47 ± 7
ICU stay	(hours)	20 ± 2	21 ± 2	28 ± 3

chemia, time of perfusion and the number of performed bypass grafts (Table 1). Neither myocardial infarction nor bleeding complications leading to re-thoracotomy were observed. The need for transfusion of packed red blood cells (RBC) and fresh frozen plasma (FFP) was slightly elevated in group A when compared to group B and C, however, this finding did not reach statistical significance. The crystalloid fluid balance did not differ sig-

nificantly between the groups, nor did the amount of colloid solution until 12 hours postoperatively. Moreover, hemoglobin and hematocrit values were comparable between the groups throughout the entire study period.

The incidence of HCF was significantly increased in groups A and B. In groups A and B, 32.5% and 40 % of the patients fulfilled the criteria of HCF, whereas only 10% of the patient's in group C developed HCF ($p < 0.05$). The time course of CO and SVR are shown in Figs. 1 and 2. When compared to group C, the CO after CPB was significantly increased in-group A and B. Concomitant we noticed a decrease in the SVR (Figs. 1 and 2). In contrast, the MAP was not significantly different in all groups (Fig. 3). However, in order to stabilize the MAP after CPB in groups A and B the patients received significantly higher amounts of norepinephrine and epinephrine when compared to group C (Fig. 4). The HR, CVP, MAP, and the pulmonary capillary wedge pressure (PCWP) showed no significant differences between the groups. Only the PVR showed an increasing tendency in group C (Fig. 5). The mixed venous oxygen saturation

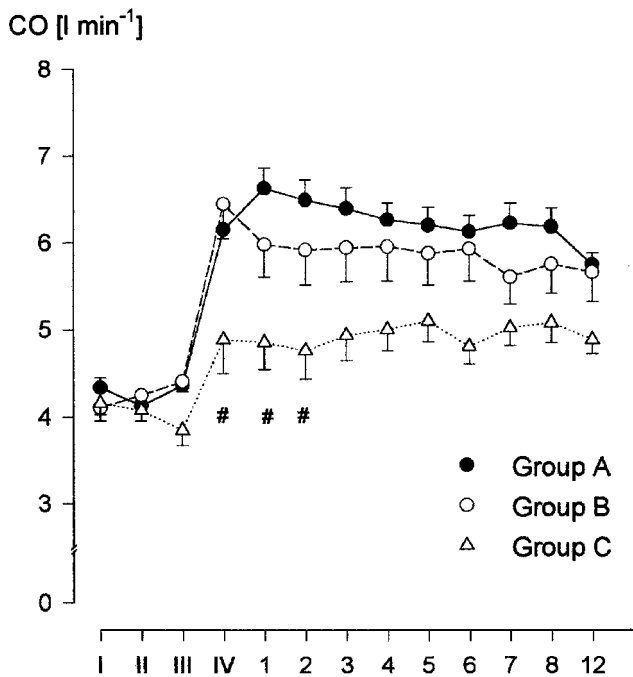


Fig. 1. Perioperative time course of cardiac output (CO). Data are given as mean ± standard error of mean (SEM). Measurements were performed before (I), directly after cardiopulmonary bypass (II), after sternal closure (III), at the end of surgery (IV) and 1, 2, 3, 4, 5, 6, 7, 8, and 12 hours after surgery. A significant difference between the patients in group C compared to groups A and B was found at the end of surgery and during the initial ICU period.

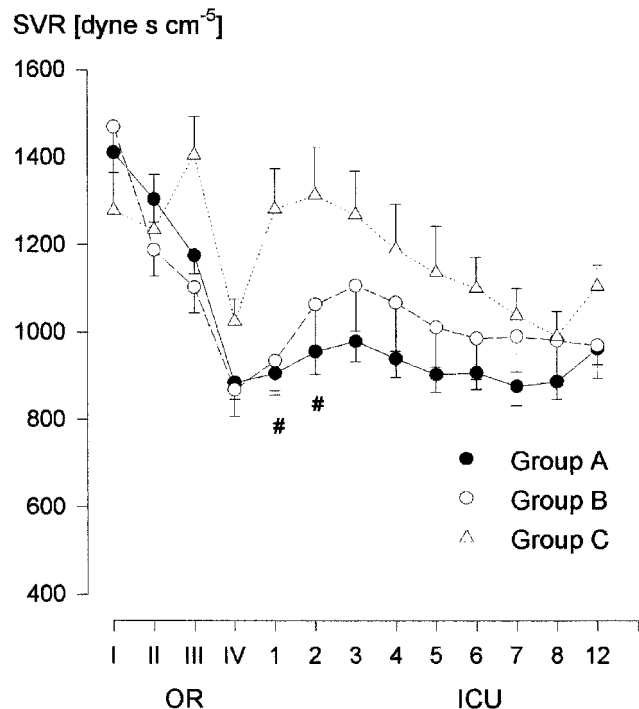


Fig. 2. Perioperative time course of systemic vascular resistance (SVR). Data are given as mean ± SEM. Measurements were performed before (I), directly after cardiopulmonary bypass (II), after sternal closure (III), at the end of surgery (IV) and 1, 2, 3, 4, 5, 6, 7, 8, and 12 hours after surgery. SVR was significantly decreased in patients with single-atrial cannulation and 1 and 2 hours after at the ICU.

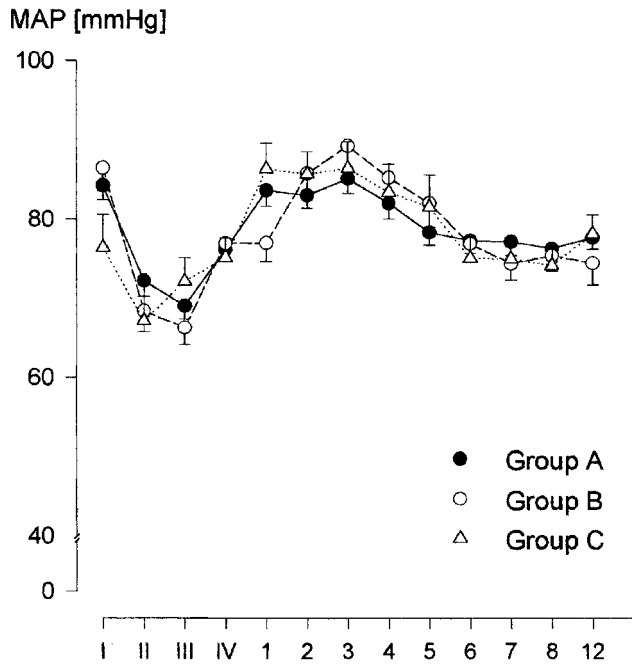


Fig. 3. Perioperative time course of mean arterial pressure (MAP). Data are given as mean \pm SEM. Measurements were performed before (I), directly after cardiopulmonary bypass (II), after sternal closure (III), at the end of surgery (IV) and 1, 2, 3, 4, 5, 6, 7, 8, and 12 hours after surgery. No significant differences in MAP were observed during the entire study period.

was significantly increased in the HCF patients (86 0.6%, n=33) when compared with those with stable hemodynamics (62 0.3%, n=87, $p < 0.05$).

Discussion

The present study results demonstrate that single-atrial cannulation and the inevitable infusion of Bretschneider’s cardioplegic solution into the systemic circulation per se are associated with a significant increase in HCF in patients undergoing CABG. In our patients the intraoperative zero-fluid balanced hemofiltration was not beneficial with respect to the incidence and severity of HCF. Bicaval cannulation and the subsequent complete removal of cardioplegic solution were associated with a lower incidence of HCF.

HCF after CPB is one of the major complications in cardiac surgery causing an increased need for catecholamine support and potentially leading to an increased morbidity and mortality rate.¹⁾ Cremer et al.²⁾ observed an HCF incidence of approximately 10% in their patient group, comparable with our retrospective data on 1200 patients. While it remains unclear why the HCF is oc-

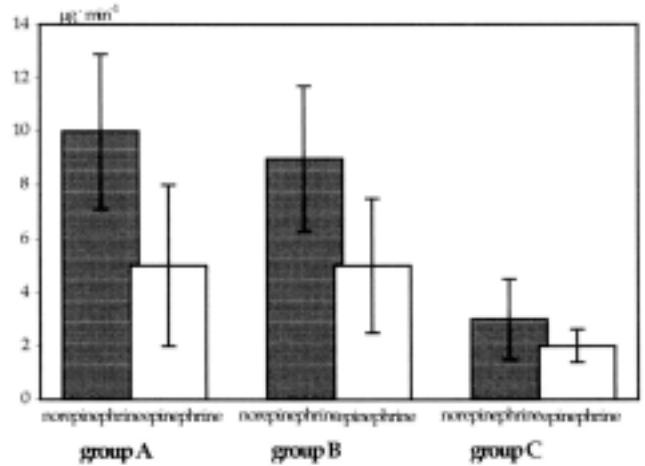


Fig. 4. Cumulative doses of norepinephrine and epinephrine during the entire study period. A significant difference in both norepinephrine and epinephrine doses were observed between patients in group C and patients in groups A and B.

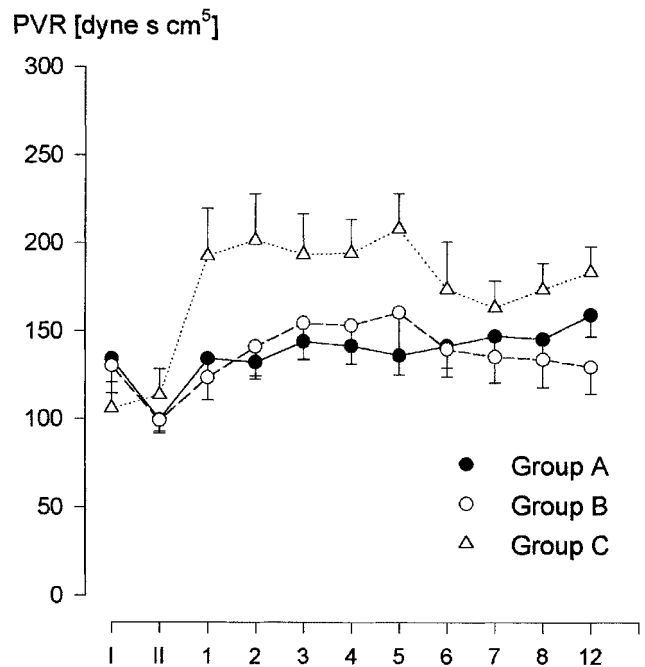


Fig. 5. Perioperative time course of pulmonary vascular resistance (PVR). Data are given as mean \pm SEM. Measurements were performed before (I), directly after cardiopulmonary bypass (II) and 1, 2, 3, 4, 5, 6, 7, 8, and 12 hours after surgery. No significant differences in PVR were observed during the entire study period. However, PVR was elevated in group C.

curing in one particular individual,^{1,3)} there is growing evidence that the reasons for HCF are multifactorial. It has been suggested that HCF is similar to the “systemic inflammatory response syndrome” (SIRS), which can be

seen during the early phase of sepsis or endotoxemia.^{1,8)} Thus, it can be hypothesized that CPB leads by itself to a SIRS-like reaction and only a subgroup of predisposed patients may develop HCF.^{1,2,7,8)} However, it is less clear which factors are responsible for the frequency of HCF after cardiac surgery.^{1,3)} During the past years, the activation of different mediator systems has been extensively studied in a variety of experimental and clinical settings.^{2,8-12)} Recently, Cremer et al. observed that interleukin 6 (IL-6) levels were significantly increased in patients developing hemodynamic instability and concluded that the circulating IL-6 plays a major role in the inflammatory cascade initiated by the CPB.⁸⁾ In contrast, Taylor et al. showed that IL-6 levels were also considerably increased in patients who did not develop HCF.¹⁾ Another cause of HCF may be the choice of cardioplegic solution (blood, crystalloid and/or colloid solution) and its application technique, i.e. single dose versus multi-dose cardioplegia.^{4-7,10,11)} So far, it remained unclear if the infusion of cardioplegic solution into the systemic circulation, which occurs inevitably when single-atrial cannulation is performed, increases the incidence of HCF.^{10,11)}

The present study was designed as a prospective, controlled, randomized trial to clarify whether the type of venous drainage, the infusion of the cardioplegic solution and/or intraoperative zero fluid balanced hemofiltration influences the incidence and severity of HCF. Our results demonstrate that the incidence of HCF was significantly increased in patients with single-atrial cannulation in which cardioplegic solution was not removed from the systemic circulation. In contrast, Louagie et al. reported no differences in their study between single atriocaval and bi-caval cannulation with respect to the systemic hemodynamics.⁸⁾ However, the cardioplegic application technique was different in both studies. Louagie et al. used multidose cardioplegia, whereas in our study one single dose of cardioplegic solution was infused immediately after cross-clamping.⁸⁾ The different cardioplegic application modalities may explain in part the different results thereafter. Beyersdorf et al. compared the hemodynamic effects of hypothermic ventricular fibrillation, multi-dose blood cardioplegia and single dose Bretschneider cardioplegia.⁴⁾ In their study no differences regarding the SVR and/or the CO have been observed.⁴⁾ In Beyersdorf's study the bi-caval cannulation technique was used in the single dose Bretschneider cardioplegic group. The hemodynamic

results of these patients were comparable with our patients group C.

The available literature and the present study results are suggesting that single dose infusion of one dose of crystalloid cardioplegic solution is a main factor for the increased incidence of HCF seen in group A and B.¹⁰⁻¹³⁾ Gallandet-Huet et al. studied also the hemodynamic effects of different crystalloid cardioplegic solutions.⁶⁾ They could demonstrate that the vasodilative properties of Bretschneider's cardioplegia are more pronounced when compared with the modified St. Thomas solution.⁶⁾ Using 20 ml kg⁻¹ of Bretschneider's cardioplegic solution and single-atrial cannulation a 30% SVR decrease was observed.⁶⁾ Patients receiving Bretschneider's solution demonstrated significantly increased CO values immediately after CPB when compared with those receiving St. Thomas solution.⁶⁾ Furthermore, 81.8 % of the patients in the Bretschneider group received vasoconstrictors, compared to 60.7 % in the St. Thomas group. The results of the present study support the Gallandet-Huet findings.

In our study, the amount of norepinephrine necessary to reach adequate arterial perfusion pressure was increased after single-atrial venous cannulation. The intraoperative zero flow-balanced hemofiltration was performed only in one group of patients. However, we observed no significant benefit with respect to hemodynamics, blood transfusion and/or incidence of HCF. These results are in line with Tassani's study,⁹⁾ who demonstrated that hemofiltration during rewarming did not result in hemodynamic improvement. Hemofiltration performed immediately after CPB leads to a decrease in the pulmonary shunt fraction and an increase in the MAP.⁸⁾ In addition, Babka et al. compared the systemic hemodynamics, the transfusion requirements, the postoperative blood loss and the postoperative ICU stay in patients with and without hemofiltration during CPB.¹⁵⁾ As in our study, no significant benefits of the hemofiltration could be detected in their controlled clinical study.

In summary, we conclude that single-atrial venous cannulation is a risk factor for HCF after CABG surgery. The increased incidence of HCF is probably caused by the inevitable infusion of cardioplegic solution into the systemic circulation required during CPB with the use of single-atrial venous drainage technique. Zero-fluid balanced hemofiltration during CPB offers no benefit with respect to systemic hemodynamics and in particular the incidence of HCF.

References

1. Taylor KM. SIRS—the systemic inflammatory response syndrome after cardiac operations. *Ann Thorac Surg* 1996; **61**: 1607–8.
2. Cremer J, Martin M, Redl H. Systemic inflammatory response syndrome after cardiac operations. *Ann Thorac Surg* 1996; **61**: 1714–20.
3. Royston D. The inflammatory response and extracorporeal circulation. *J Cardiothorac Vasc Anesth* 1997; **11**: 341–54.
4. Beyersdorf F, Krause E, Sarai K. Clinical evaluation of hypothermic ventricular fibrillation, multi-dose blood cardioplegia, and single-dose Bretschneider cardioplegia in coronary surgery. *Thorac Cardiovasc Surg* 1997; **38**: 20–9.
5. Buckberg GD. Strategies and logic of cardioplegic delivery to prevent, avoid and reverse ischemic and reperfusion damage. *J Thorac Cardiovasc Surg* 1987; **93**: 127–39.
6. Gallandat-Huet RCG, Karliczek GF, Homan van der Heide JN. Clinical effect of Bretschneider-HTK and St. Thomas cardioplegia on hemodynamic performance after bypass measured using an automatic datalogging database system. *Thorac Cardiovasc Surg* 1988; **36**: 151–6.
7. Menasché P, Fleury JP, Veyssié L. Limitation of vasodilatation associated with warm heart operation by a “mini-cardioplegia” delivery technique. *Ann Thorac Surg* 1993; **56**: 1148–53.
8. Louagie Y, Gonzalez M, Collard E. Assessment of two venous drainage techniques in coronary artery bypass graft surgery. *Thorac Cardiovasc Surg* 1989; **37**: 169–73.
9. Tassani P, Richter JA, Eising GP. Influence of combined zero-balanced and modified ultrafiltration on the systemic inflammatory response during coronary artery bypass grafting. *J Cardiothorac Vasc Anesth* 1999; **13**: 285–91.
10. Miller BE, Levy JH. The inflammatory response to cardiopulmonary bypass. *J Cardiothorac Vasc Anesth* 1997; **11**: 355–66.
11. Wan S, Yim AP, Arifi AA. Can cardioplegia management influence cytokine responses during clinical cardiopulmonary bypass? *Ann Thorac Cardiovasc Surg* 1999; **5**: 81–5.
12. Hall RI, Smith MS, Rocker G. The systemic inflammatory response to cardiopulmonary bypass: pathophysiological, therapeutic, and pharmacological considerations. *Anesth Analg* 1997; **85**: 766–82.
13. Pilz G, Kaab S, Kreuzer E, Werdan K. Evaluation of definitions and parameters for sepsis assessment in patients after cardiac surgery. *Infection* 1994; **22**: 8–17.
14. Gomes WJ, Carvalho AC, Palma JH. Vasoplegic syndrome after open-heart surgery. *J Cardiovasc Surg (Torino)* 1998; **39**: 619–23.
15. Babka RM, Petress J, Briggs R, Helsal R, Mack J. Conventional hemofiltration during routine coronary bypass surgery. *Perfusion* 1997; **12**: 187–92.