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## Perioperative monitoring of circulating and central blood volume in cardiac surgery by pulse dye densitometry

Received: 29 September 2003  
Accepted: 25 August 2004  
Published online: 18 September 2004  
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**Abstract** *Objective:* To determine perioperative changes in circulating (BVI) and central blood volume (CBVI) by a new dye dilution technique using pulse dye densitometry. *Design and setting:* Prospective observational study in the cardiac anesthesia and intensive care unit of a university hospital. *Patients:* Sixty-six patients undergoing coronary artery bypass surgery. *Measurements and results:* Hemodynamic measurements by the dye dilution method using pulse dye densitometry were performed prior to skin incision and  $3.3 \pm 1.4$  h and  $17 \pm 2.7$  h after surgery. Based on conventional monitoring the therapeutic goals of hemodynamic therapy were achieved in all of the patients of this study. Despite a marked positive fluid balance which developed during surgery mean BVI decreased significantly after surgery while CBVI remained unchanged.

Postoperative BVI deficits vs. preoperative values were observed in 78% of patients; these BVI deficits were profound in 29% of the cases. In contrast, 65% of the individual patients showed no or only minor postoperative changes in CBVI vs. preoperative values. *Conclusions:* Changes in the intravascular volume compartments affected BVI to a greater extent than CBVI. Therefore measuring circulating in addition to central blood volume may be useful to ensure a normal circulating blood volume that can compensate for any change in the central vascular compartment.

**Keywords** Blood volume · Central blood volume · Dye dilution technique · Pulse dye densitometry · Perioperative · Cardiopulmonary bypass

### Introduction

Changes in intravascular blood volume after cardiac surgery using cardiopulmonary bypass are considered a major cause of hemodynamic instability. Since both hypovolemia and hypervolemia have been shown to contribute significantly to perioperative morbidity and mortality [1, 2, 3], methods to determine intravascular volume load are crucial for guiding fluid therapy. Central venous and pulmonary artery occlusion pressure are generally for this. However, in ventilated patients this practice has been under debate recently since no correlation has been established between these preload parameters and cardiac

output [4, 5, 6, 7, 8, 9]. In contrast, central blood volume (CBV), which refers to the blood volume of the intrathoracic vascular compartment, has recently been shown to be a better indicator of cardiac preload [4, 5, 6, 7, 8, 9]. Another volume compartment which can be quantified at the bedside is circulating blood volume (BV), which refers to entire circulation. Several studies report that BV is significantly reduced after surgery [1, 10, 11, 12, 13, 14]. Furthermore, Tschaikowsky and coworkers [15] suggest the goal of maintaining BV in the perioperative period to achieve hemodynamic stability after cardiac surgery. However, there are no studies in patients undergoing

cardiac surgery addressing the perioperative monitoring of both CBV and BV.

Various indicator dilution techniques are available for measuring intravascular volume compartments. Recently a method for bedside measurement of CBV and BV using a dye dilution technique with indocyanine green (ICG) as indicator has been introduced to the market. Unlike other commercially available devices requiring a specific arterial catheter in the femoral artery [16], the device used in our work detects the ICG blood concentration by pulse dye densitometry (PDD) transcutaneously [17]. In this observational study we report the pattern of perioperative changes in CBV and of BV after coronary bypass surgery using a novel, less invasive monitoring device.

## Material and methods

After institutional approval, 66 patients scheduled for coronary bypass surgery gave their written informed consent and were included in the study. Exclusion criteria were: allergy to ICG or contrast media, ejection fraction below 40%, application of an aortic counterpulsation device, renal failure, atrial fibrillation and detection of intracardiac shunting by echocardiography. Five patients in whom the pulsation amplitude of the PDD device was less than 1% were excluded from analysis. The mean characteristics of the remaining 61 patients were: age  $66.8 \pm 7.8$  years, height  $170 \pm 7.9$  cm, weight  $81 \pm 11.3$  kg, body surface area BSA  $1.9 \pm 0.2$  m<sup>2</sup>, body mass index  $28.1 \pm 3.2$ .

On the day of surgery the patients were given 40 mg chloralhydrate. General anesthesia was induced with 30 µg/kg alfentanil and a target controlled infusion of propofol (target 2.5 µg/ml). Pancuronium bromide (0.1 mg/kg) was used to facilitate tracheal intubation. Anesthesia was maintained by continuous infusion of propofol (target 2–3 µg/ml) and alfentanil (50–100 µg/kg per hour) until the patient was transferred to the intensive care unit (ICU). Patients were ventilated (Kion, Siemens, Erlangen, Germany) using volume-controlled mode. The ventilator was set up to a tidal volume of 10 ml/kg, positive end-expiratory pressure 5 cmH<sub>2</sub>O and mandatory breath rate to achieve normocapnia. FIO<sub>2</sub> was adjusted to maintain a PaO<sub>2</sub> of 10 kPa or higher. Analgesia was continued postoperatively using incremental doses of piritramide. Until extubation criteria were met, patients were sedated using propofol infusion, and they were ventilated (Siemens Servo 300, Erlangen, Germany) by synchronized intermittent mandatory ventilation mode. Mean duration of surgery was  $53.4 \pm 24.8$  min.

### Measurement of intravascular volume compartments

Intravascular volume compartments and cardiac output were determined by a dye dilution technique using indocyanine green (Pulsion, Munich, Germany). After injection of ICG via the distal port of the central venous catheter the blood concentration of ICG in the systemic circulation was determined by pulse dye densitometry (DDG-Analyser 2001, Nihon-Kohden, Tokyo, Japan). The detector cell is a clip attached to the patient's nasal wing. PDD works similarly to pulse oximetry. It uses three instead of the usual two wavelengths to detect the maximal absorption of ICG in addition to oxyhemoglobin and reduced hemoglobin. As the PDD device detects only the relative ICG blood concentration, separate determination of the actual hemoglobin concentration by blood gas analysis (ABL 625, Radiometer, Copenhagen, Denmark) is needed to calculate absolute ICG blood concentrations. To obtain reliable

measurements the pulsatile component of light absorption in pulse oximetry should account for 1–5% of the total signal. If the PDD monitor indicated a pulsation amplitude below 1%, measurements were not performed.

After ICG injection the dye densitometer calculates cardiac output (CO) and mean transit time (MTT) from the dye dilution curve obtained over a period of 90 s. MTT is the time between the first detection of the indicator at the injection-detector attached to the central venous catheter and the mean time of indicator persistence at the detector cell attached to the nasal wing. The product of MTT and CO is the distribution volume of ICG between the injection site (right atrium) and the detection site (arterial vascular system), which is called CBV. In this study CO and MTT were calculated from mean values out of three consecutive dye dilution measurements. For each measurement 5 mg ICG diluted in 1 ml sterile water was filled into an injection line, which was attached to the distal port of the central venous catheter. ICG was injected into the circulation by flushing this line with 9 ml 5% glucose. Since total elimination of ICG would last about 3 half-lives (approx. 12 min), remaining plasma ICG would cause error when measurements were repeated every 2 min during CO determination. Therefore the device automatically calibrates to zero ICG plasma concentration at the time a new measurement starts. This baseline gradually drops during the measurement as a result of the elimination of the remaining ICG. Hence the area under the ICG concentration curve is underestimated, but, as previously reported, the resulting error is negligible [18].

To measure BV the ICG blood concentration was detected over a total period of 15 min following injection, and the resulting concentration vs. time data was fitted to a one compartment model using the following equation:  $C = A \times \exp(-Kt)$ , where C=ICG concentration, A=constant factor, K=elimination rate constant. To determine BV the ICG concentration calculated at MTT was applied to:  $BV = D/C_{MTT}$ , where D=ICG dose,  $C_{MTT}$ =ICG concentration at MTT. A more detailed description of CBV and BV determinations by the PDD method is given elsewhere [19]. In this study BV was measured using a single ICG-bolus of 0.4 mg/kg which was injected via the distal port of the central venous catheter. To avoid indicator loss the central venous line was flushed by 10 ml glucose 5%. Accumulation of ICG was not observed in our patients since in every case ICG plasma concentrations dropped rapidly and reached values below 1.5 mg/l at the end of each CO and BV measurement.

After induction of anesthesia both BV and CBV were measured before skin incision ( $t_0$ ). These values served as control. After surgery two subsequent measurements were performed, one after admission to the intensive care unit ( $t_1$ ) and a second on the first postoperative day ( $t_2$ ). All measurements were performed by a single investigator.

### Hemodynamic management

Starting with anesthesia induction 10 ml/kg ringer solution and 6 ml/kg hydroxyethylstarch (Voluven, Fresenius Kabi, Bad Homburg, Germany) were infused in each patient prior to skin incision. During surgery and the postoperative period Ringer's solution was constantly infused at a rate of 1 ml/kg per hour. When needed, additional amounts of Ringer's solution were given to compensate for excessive urine output. Intraoperative blood losses and postoperative drainage losses were treated by infusion of equal amounts of hydroxyethylstarch or packed red blood cells. Hemodynamic therapy was guided by criteria such as heart rate, mean arterial blood pressure, central venous pressure, and urine output (intervention triggers: >100 beats/min, <60 mmHg, <8 mmHg, <1 ml/kg). Fluids were substituted to maintain the parameters as specified above. In the case of failure to the clinical standard therapy ( $n=10$ ), a femoral arterial catheter (PulsioCath 4F PV2014L16 and PiCCO, Pulsion Medical Systems, Munich, Germany) was introduced to improve

hemodynamic therapy according to the results of an extended monitoring [intervention triggers: cardiac index  $<2 \text{ l min}^{-1} \text{ m}^{-2}$ , systemic vascular resistance (SVRI)  $<1200 \text{ dyne s}^{-5} \text{ m}^{-2}$ ). Dobutamine was used for standard inotropic support; dobutamine was replaced by epinephrine in the case of severe cardiac dysfunction. Norepinephrine was the drug of choice in patients with relevant hypotension due to decreased SVRI. Nitroglycerin was used for treatment of hypertension (mean arterial blood pressure  $>90 \text{ mmHg}$ ). Physicians involved in patient care were unaware of the study results of determination of the volume compartments.

#### Fluid balances

Net fluid balances were obtained at each measurement and at the end of surgery. Crystalloid infusions, cardiopulmonary bypass priming solutions, urine output, gastric tube losses, and perspiration insensibilis (35 ml/h) referred to the crystalloid balance. The colloid balance was calculated from the amount of colloid infusions, blood and fresh frozen plasma transfusions, shed blood, drainage losses, and the remaining volume in the cardiopulmonary bypass circuit at the end of surgery.

#### Statistics

Unless otherwise stated, continuous variables are expressed as mean  $\pm$ SD. The Shapiro-Wilk test was used to test for normal distributions. The mean of physiological and volumetric parameters was analyzed by analysis of variance for repeated measurements. If significant differences were found, post hoc comparisons were performed using Tukey's honest significant difference test. Statistical significance was set at  $p \leq 0.05$ .

## Results

Data were collected  $3.3 \pm 1.4 \text{ h}$  ( $t_1$ ) and  $17 \pm 2.7 \text{ h}$  ( $t_2$ ) after skin closure. At  $t_0$  and  $t_1$  all patients were artificially ventilated, and at  $t_2$  all but four patients (6.1%) were extubated. At  $t_0$  no vasoactive drugs were necessary, but infusions of epinephrine, norepinephrine, dobutamine, and nitroglycerin were used in 1, 10, 14, and 12 patients, respectively, after surgery (Table 1). Patients developed significant positive fluid balances intraoperatively, but fluid in- and output was balanced postoperatively (Table 1). During the study period 29% of the patients were transfused by a mean of  $1.6 \pm 0.6$  packed red cells, which were mainly given during surgery. Most of the physiological parameters obtained (Table 2) showed significant differences between pre- and postoperative values. In general both cardiac index and heart rate were significantly increased after surgery while SVRI showed a significant reduction. Only ten patients with decreased SVRI developed hypotension requiring norepinephrine infusions. Mean CBVI remained unchanged throughout the study period, whereas mean BVI was significantly decreased after surgery (Fig. 1).

Figure 2 presents a more detailed view on the postoperative BVI changes, analyzing the incidence of various degrees of differences between pre- and postoperative BVI. At  $t_1$  49% of the patients had a BVI deficit of up to

**Table 1** Fluid balances, urine output, and major drug therapy differences during different time intervals ( $t_0$  after induction of anesthesia,  $t_1$  after admission to the intensive care unit,  $t_2$  first postoperative day, EOS end of surgery,  $Balance_{Cryst}$  cumulative amount of crystalloid infusions minus urine output and gastric tube losses,  $Balance_{Coll}$  cumulative amount of colloid solutions, blood, and fresh frozen plasma transfusions minus shed blood and drainage losses)

	$t_0$ -EOS	EOS- $t_1$	$t_1$ - $t_2$
Interval duration (h)	$3.4 \pm 1.1$	$3.3 \pm 1.4$	$17 \pm 2.7$
Balance <sub>Cryst</sub> (ml/kg)	$17.8 \pm 13.3$	$1.8 \pm 1.6$	$0.2 \pm 1.1$
Balance <sub>Coll</sub> (ml/kg)	$5.1 \pm 3.8$	$1.6 \pm 2.1$	$0.9 \pm 1.9$
Diuresis (ml/h)	$208 \pm 137$	$233 \pm 96$	$120 \pm 29$
Furosemide (mg)	$15 \pm 15$	$3 \pm 4$	$33 \pm 29$
Dobutamine			
<i>n</i>	14 <sup>a</sup>	14	8
Mean ( $\mu\text{g kg}^{-1} \text{ min}^{-1}$ )	$4.7 \pm 1.8$	$5.1 \pm 2$	$2.1 \pm 1.3$
Epinephrine			
<i>n</i>	1 <sup>a</sup>	1	1
Mean ( $\mu\text{g kg}^{-1} \text{ min}^{-1}$ )	0.08	0.09	0.07
Norepinephrine			
<i>n</i>	7 <sup>a</sup>	10	3
Mean ( $\mu\text{g kg}^{-1} \text{ min}^{-1}$ )	$0.05 \pm 0.02$	$0.06 \pm 0.03$	$0.03 \pm 0.01$
Nitroglycerin			
<i>n</i>	5 <sup>a</sup>	12	12
Mean ( $\mu\text{g kg}^{-1} \text{ min}^{-1}$ )	$0.36 \pm 0.13$	$0.57 \pm 0.22$	$0.63 \pm 0.21$

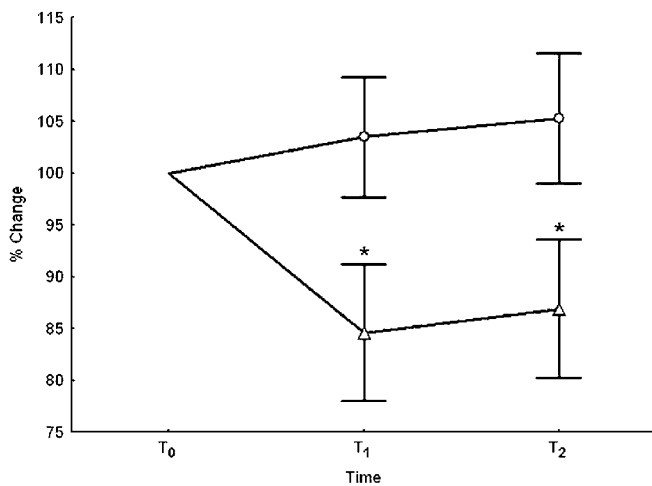
<sup>a</sup> Drug therapy started at the end of cardiopulmonary bypass

**Table 2** Physiological parameters before ( $t_0$ ) and after ( $t_1$ ,  $t_2$ ) coronary artery bypass operation (MAP mean arterial pressure, CVP central venous pressure, SVI stroke volume index, SVRI system vascular resistance index, CBVI central blood volume index, BVI blood volume index)

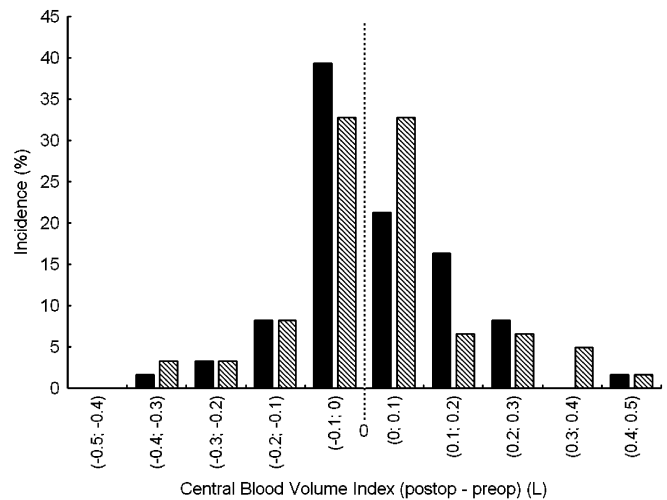
	$t_0$	$t_1$	$t_2$
Hematocrit (%)	$38.5 \pm 4.3$	$37 \pm 4.4^*$	$35.7 \pm 3.7^*$
Temperature ( $^{\circ}\text{C}$ )	$36.9 \pm 0.2$	$36 \pm 0.7^*$	$37.5 \pm 0.5^{***}$
Heart rate ( $\text{min}^{-1}$ )	$57 \pm 9$	$82 \pm 14^*$	$83 \pm 13^*$
MAP (mmHg)	$92 \pm 16$	$80 \pm 14^*$	$84 \pm 9^*$
CVP (mmHg)	$10 \pm 4$	$11 \pm 3$	$12 \pm 3^*$
Cardiac index ( $\text{l min}^{-1} \text{ m}^{-2}$ )	$1.9 \pm 0.4$	$2.4 \pm 0.9^*$	$2.6 \pm 0.6^*$
SVI ( $\text{ml m}^{-2}$ )	$34.3 \pm 7.5$	$29.3 \pm 9.4^*$	$31.7 \pm 7.2$
SVRI ( $\text{dynes s}^{-1} \text{ cm}^{-5} \text{ m}^{-2}$ )	$3593 \pm 952$	$2594 \pm 976^*$	$2396 \pm 762^*$
Lactate ( $\text{mmol l}^{-1}$ )	$1.0 \pm 0.3$	$2.2 \pm 1.9^*$	$1.9 \pm 1.2^*$
Base excess ( $\text{mmol l}^{-1}$ )	$1.5 \pm 0.3$	$0.1 \pm 0.6^*$	$4.8 \pm 0.6^{***}$
CBVI ( $\text{ml m}^{-2}$ )	$672 \pm 128$	$681 \pm 145$	$679 \pm 136$
BVI ( $\text{ml m}^{-2}$ )	$2775 \pm 658$	$2227 \pm 495^*$	$2319 \pm 556^*$

\* $p < 0.05$  vs.  $t_0$ , \*\* $p < 0.05$  vs.  $t_1$

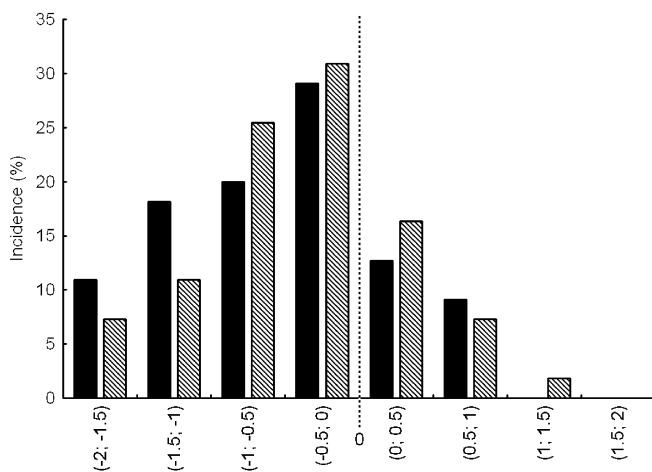
1000 ml, and another 29% had one greater than 1000 ml. Although there was a shift from large ( $>1000 \text{ ml}$ ) to less pronounced BVI deficits ( $<1000 \text{ ml}$ ), there was a similar pattern of the differences between pre- and postoperative BVI at  $t_2$ . Higher postoperative BVI (22% at  $t_1$  and 25% at  $t_2$ ) was observed less frequently than BVI deficits (Fig. 2). In contrast, nearly 65% of the patients had an unchanged CBVI or showed differences between pre- and postoperative CBVI less than 100 ml at both  $t_1$  and  $t_2$  (Fig. 3). Larger postoperative CBVI changes were ob-



**Fig. 1** Postoperative time course of mean indexed central blood volume (open circles) and mean indexed circulating blood volume (open triangles) following admission to the intensive care unit ( $t_1$ ) and on the first postoperative day ( $t_2$ ), presented as percentage change compared to preoperative ( $t_0$ ) values. \* $p < 0.05$



**Fig. 3** Frequency distribution of differences between central blood volume (CBVI) before and after coronary bypass surgery (postop-preop) within distinct volume ranges following admission to the intensive care unit ( $t_1$ ; solid bars) and on the first postoperative day ( $t_2$ ; hatched bars)



**Fig. 2** Frequency distribution of differences between circulating blood volume (BVI) before and after coronary bypass surgery (postop-preop) within distinct volume ranges following admission to the intensive care unit ( $t_1$ ; solid bars) and on the first postoperative day ( $t_2$ ; hatched bars)

served less frequently, and this pattern did not differ between  $t_1$  and  $t_2$ .

## Discussion

Measurement of CBV and BV commonly performed using ICG injection via central venous catheter and detection by a reflection densitometer in the descending aorta are regarded as accurate and less sensitive to artifacts [6, 9]. A recent study comparing this techniques with the less

invasive one (one catheter, PDD method) also used in this study reported a good correlation between the two methods with respect to the ICG plasma disappearance [20]. The same investigators later found moderate agreement between the methods in comparing corresponding values of CO and CBV in patients with sepsis and acute respiratory distress syndrome [21]. The authors concluded that the PDD method is accurate in measuring relative changes in ICG plasma concentration but less precise in detecting absolute ICG concentrations, mainly due to large intraindividual variability. In contrast, the intraindividual variability was not that large in our study (6.3%). A possible explanation for these different results is that in contrast to our work Sakka and coworkers [21] did not use the mean of a triplet of ICG injections to determine CO and CBV.

BV determination using ICG is based on the dilution principle and depends on the correct measurement of absolute ICG plasma concentrations [22]. Good agreement was reported recently in comparing the PDD method with classical techniques of BV measurement using blood samples to detect the concentration of ICG or  $^{51}\text{Cr}$ -labeled red blood cells in cardiac surgical patients [23, 24]. In contrast, Ishihara and coworkers [25] reported BV determination by PDD to be inaccurate early after cardiac surgery. However, in that study all patients but one required vasoactive drugs after surgery, and the authors themselves suggested that poor peripheral circulation, a known problem with the PDD method, related to hypovolemia and vasopressor therapy, was most probably the reason for their results.

We observed profound changes in the hemodynamic profile of our patients after cardiopulmonary bypass sur-

gery, which is in accord with previous reports [5, 6, 26, 27]. The preoperative period was characterized by cardiocirculatory depression due mainly to  $\beta$ -blocking agents and tranquilizers, while decreased SVRI and increased cardiac index and heart rate were observed postoperatively. Reduction in vascular resistance after cardiopulmonary bypass is a well-known phenomenon and most likely the result of a temporary systemic inflammatory response syndrome [28, 29, 30].

Guided by conventional monitoring, the therapeutic goals of hemodynamic therapy as stated above were reached in all of our patients. However, compared to preoperative values we found the average BV to be reduced by about 15% after surgery despite marked positive fluid balances which is well in agreement with other reports [11, 12, 13, 14, 15]. Interestingly, liberal administration of fluids during surgery did not compensate for postoperative BV reduction. Two-thirds of our patients developed moderate to profound BV deficits after surgery compared to preoperative values, a finding which is well in agreement with previous findings by Tschaikowsky and coworkers [15]. These authors found increased plasma lactate concentrations corresponding to the postoperative time course of BV deficits and considered reduced organ perfusion due to hypovolemia to be a likely cause.

In contrast to BV, the average CBV throughout the study period remained unchanged. Major perioperative changes in CBV were both less frequent and less pronounced than the perioperative BV changes. The finding of perioperatively unchanged CBV is interesting because the three time points in our study referred to different pathophysiological conditions, i.e., pre- vs. postoperative status, with and without mechanical ventilation. Schmidt and coworkers [31] studied the effect of mechanical ventilation on CBV and reported that CBV was significantly increased by changing mechanical ventilation to T-piece breathing, and that CBV returned to the values obtained during mechanical ventilation 10 min after extubation. Since we used a different timing that omitted CBV measurement during T-piece breathing, we cannot compare our findings to those of Schmidt et al. However, an unchanged CBV after extubation was found in both studies.

CBV has been reported to be an important indicator of cardiac preload [4, 5, 6, 7, 8, 9, 32]. Hence an unchanged CBV, as found in the majority of our patients, may indicate adequate perioperative volume management. However, Hinder and coworkers [6] recently reported that CBV had to be greater after cardiac surgery than before the incision to maintain a constant left ventricular end diastolic area measured by echocardiography. The authors hypothesized that the cause was an increased pulmonary vascular capacity due to vasodilation related to systemic inflammatory response syndrome in cardiac surgery patients. Strey and coworkers [33] studied pressure-volume relationships and examined compliance of the central and the peripheral low-pressure vascular systems due to

hemorrhage in healthy adults. They reported a large distensibility of the central vascular compartment compared to the extrathoracic vasculature. In view of the variable capacitance of the central vascular compartment suggested by the findings of Strey et al. [33] and Hinder et al. [6], a normal CBV may not inevitably allow an adequate preload in cardiac surgery patients. However, normal or even increased BV after surgery could possibly compensate for any change in the central compartment capacitance. Measuring BV in addition to CBV may thereby improve perioperative hemodynamic management.

The pattern of perioperative changes in CBV and BV, especially the relationship between the two parameters, may be affected by changes in the intrathoracic pressure, i.e., initiation or cessation of positive pressure ventilation. In our study the ratio between CBV and BV changed from 25% before skin incision to more than 30% after surgery while the patients were still artificially ventilated. On the first day after surgery, when most of our patients were extubated, we still found CBV to BV ratios of about 30%. Hence a larger fraction of blood volume may have referred to CBV after extubation, suggesting volume shifts from extra- to intrathoracic compartments. In a recent study von Spiegel and coworkers [34] measured the effect of positive pressure ventilation on both CBV and BV. The authors reported a significant blood volume shift from intra- to extrathoracic compartments after onset of positive pressure ventilation during anesthesia induction. Although our findings cannot be compared directly to those of von Spiegel and coworkers, the underlying pathophysiology of volume shifts between intra- to extrathoracic compartments may be similar.

In summary, the less invasive and easy-to-use method of PDD seems to be a useful tool for determining volume compartments in cardiac surgery patients at the bedside. However, PDD is sometimes difficult to perform due to motion artifacts and poor tissue perfusion after surgery. Readings of ICG blood concentrations should therefore always be checked for plausibility and interpreted carefully. Since CBV is considered to be a measure of cardiac preload, achieving hemodynamic stability accompanied by an overall unchanged CBV after surgery, as seen in our patients, supports the adequacy of fluid therapy. However, CBV may after cardiac surgery vary due to systemic inflammatory response syndrome related vasodilation of the central vasculature. Preservation of a normal BV could possibly compensate for any change in the central vascular capacitance. Thus monitoring BV in addition to CBV may be worthwhile. However, further studies are required to define the therapeutic goals that a hemodynamic therapy based on monitoring of CBV and BV should aim at to maintain organ perfusion perioperatively.

**Acknowledgement** The authors thank Dr. S. Ensminger for critically reading the manuscript.

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