

Continuous right ventricular end-diastolic volume in comparison with left ventricular end-diastolic area

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Background and objective Intraoperative management of patients with end-stage liver disease undergoing liver transplantation requires fluid administration to increase cardiac output and oxygen delivery to the tissues. Filling pressures have been widely shown to correlate poorly with changes in cardiac output in the critically ill patient. Continuous right ventricular end-diastolic volume index (cRVEDVI) and left ventricular end-diastolic area index (LVEDAI) monitoring have been increasingly used for preload assessment. The aim of this study was to compare cRVEDVI, LVEDAI, central venous pressure and pulmonary artery occlusion pressure with respect to stroke volume index (SVI) during liver transplantation.

Methods Measurements were made in 20 patients at four predefined steps during liver transplantation. Univariate and multivariate panel-data fixed effect regression models (across phases of the surgical procedure) were fitted to assess associations between SVI and cRVEDVI, pulmonary artery occlusion pressure, central venous pressure and LVEDAI after adjusting for ejection fraction (categorized as ≤ 30 , $31-40$, >40).

Results SVI was associated with continuous right ventricular ejection fraction. The model showing the best fit to the data was that including cRVEDVI: even after adjusting

for continuous right ventricular ejection fraction and phase, the regression coefficient of cRVEDVI in predicting SVI was statistically significant and indicated an increase in SVI of 0.21 ml m^{-2} for each increase of 1 ml m^{-2} . At the multivariate analysis, an increase in LVEDAI of 1 cm m^{-2} led to an increase in SVI of 1.47 ml m^{-2} ($P=0.054$).

Conclusion cRVEDVI and LVEDAI gave a better reflection of preload than filling pressure, even if only cRVEDVI reached statistical significance. *Eur J Anaesthesiol* 26:272–278

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Introduction

Assessing the optimal volume status in the perioperative course of major surgery is a challenge for the anaesthesiologist. It would be better to monitor volumes instead of the widely used cardiac filling pressures that have shown a poor correlation with cardiac output (CO) changes. Volumetric assessment of preload using a pulmonary artery catheter (PAC) has been suggested [1–4]. Continuous right ventricular end-diastolic volume (cRVEDV) assessed by modified PAC has been shown to be a better indicator of left ventricular filling than pressure preload variables [5–9]. In the late 1990s, the current generation of volumetric and continuous cardiac output (CCO) PAC was introduced. These catheters differ significantly from the original in that they allow for calculation of continuous right ventricular end-diastolic volume index (cRVEDVI) from continuous right ventricular ejection fraction (cRVEF) and stroke volume index (SVI) [8]. This volume index also

showed a better correlation with cardiac performance than cardiac filling pressures in studies performed both in critically ill patients and in trauma and surgical patients [5–13]. Transoesophageal echocardiography (TOE) should be performed during anaesthesia for liver transplantation (LTx) allowing rapid visualization of the left ventricular (LV) dimension and function [14]. The planimetry of the left ventricular end-diastolic area (LVEDAI) is easy to obtain and provides a reliable and consistent measure of LV filling that has been shown to correlate well with changes in SVI during volume therapy [15–18].

The present study was designed to compare volumetric preload indexes as measured by pulmonary arterial thermodilution (cRVEDVI), LV preload assessed by TOE (LVEDAI), pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP) with respect to the SVI during LTx.

Methods

We obtained approval from the Ethics Committee and written informed consent from 20 patients (15 men and five women) who were about to undergo orthotopic LTx. Patients with preexisting pulmonary or cardiac diseases or both, other than the common symptoms of end-stage liver disease (ESLD) [19], valve disease, diastolic dysfunction and patients with acute hepatic failure, hepato-pulmonary syndrome, retransplant procedure or pulmonary hypertension were excluded from the study. These features were evaluated during the preoperative clinical assessment of the liver transplant candidates.

Anaesthetic technique

Standard monitoring consisted of two-lead electrocardiography (II/V5) to measure the heart rate (HR), pulse oximetry and invasive systemic arterial pressure monitoring (PCM SpaceLabs Inc., Redmond, Washington, USA). Anaesthetic management was standardized and consisted of propofol ($0.5\text{--}1\text{ mg kg}^{-1}$) for the induction, cisatracurium besilate (0.15 mg kg^{-1}) as muscle relaxant and alfentanil ($7\text{--}10\text{ }\mu\text{g kg}^{-1}$) for analgesia. Anaesthesia was maintained with sevoflurane (end-tidal 0.8%) or desflurane (end-tidal 4%) and remifentanyl continuous infusion ($0.1\text{--}0.5\text{ }\mu\text{g kg}^{-1}\text{ min}^{-1}$). For postoperative analgesia assessment, all patients received sufentanil continuous infusion ($0.001\text{--}0.002\text{ }\mu\text{g kg}^{-1}\text{ min}^{-1}$) until the start of surgery. Intermittent positive pressure ventilation and analysis of inspired gases and end-tidal CO_2 was done with a volumetric anaesthesia ventilator (CATO, Dräger Werk HG, Lubek, Germany). All LTxs were completed without veno-venous bypass, using the piggy-back technique. Body temperature was controlled to avoid hypothermia using a warming blanket (Gaymar Meditherm, Orchard Park, New York, USA) and warm intravenous fluids (HOT LINE, SIMS Medical System, Inc., Rockland, Massachusetts, USA).

Cardiopulmonary monitoring

An 8.0 Fr PAC (Swan-Ganz Catheter CCOmbo $\text{CO/SvO}_2/\text{CEDV/VIP}$, catheter 777HF8; Edwards Lifescience, Irvine, California, USA) was placed via a 8.5 Fr introducer (AVA 3Xi Edwards Lifescience) inserted into the right internal jugular vein and attached to the Vigilance monitor (Edwards Lifescience) for intermittent CO, CVP, PAOP, mean pulmonary arterial pressure (mPAP), body temperature (T°) and cRVEDVI monitoring and to also obtain CCO, continuous mixed venous oxygen saturation (SvO_2), cRVEF and continuous SVI. Intermittent CO measurements were made by manual injection of 10 ml isotonic saline solution, at room temperature, into the superior vena cava through the atrial port. Three consecutive boluses were injected regardless of the phase of the respiratory cycle over a 2 min period. In cases in which a more than 10% discrepancy in the CO measurements was detected, measurements were repeated five times and the lowest and the highest results were discarded. All data were

obtained while patients were mechanically ventilated. The zero reference for the supine position was the mid-axillary line.

The new cRVEDVI algorithm generates a relaxation waveform resembling the bolus thermodilution washout decay curve. Decay curve data are averaged over the entire CCO on-and-off sequence. ECG is slaved in and HR is time averaged over this period. The cRVEF is calculated based on the estimation of the exponential decay time constant (τ) of this curve and HR ($\text{cRVEF} = 1 - \exp[-60/(\tau \times \text{HR})]$). Displayed volumetric values are updated every 60 s reflecting an average of the last 6–9 min. The waveform is based on the repeating on–off input signal and is generated by accumulating the temperature change for each on and off segment of the input signal. cRVEDV is calculated in the following way:

$$\text{cRVEDV} = \frac{(\text{CCO}/\text{HR})}{\text{cRVEF}}$$

where CCO is obtained from the PAC. Estimation of cRVEF is based on all the data available in the new relaxation curve and not just a few data points. Details of this method have been published elsewhere [10]. CVP and PAOP were measured using a standard transducer (Edwards Lifesciences, Irvine).

TOE was performed using a Power Vision 8000 system with a multiplane TOE probe (Toshiba, Tokyo, Japan). The probe was positioned to obtain the transgastric mid-papillary short-axis view of the LV. This position was maintained throughout the whole study period. LV end-diastolic area was measured at the peak of the electrocardiographic R wave and by manually tracing the endocardial border including the papillary muscles. Simultaneously acquired TOE images and ECG signals were recorded on a magneto-optical disk and analysed off-line by an experienced investigator blinded to the haemodynamic results. For each measurement, an average of at least four consecutive cardiac beats throughout the respiratory cycle was evaluated.

Experimental protocol

After induction of anaesthesia and achievement of stable haemodynamic conditions, baseline haemodynamic data were evaluated. Haemodynamic measurements were obtained at four stages: 30 min after induction of anaesthesia, after all lines were placed and before skin incision (T_0), 20 min after the start of anhepatic phase (T_c), at least 30 min after graft reperfusion (T_r) and finally at the end of surgery after skin suture (T_f). At each time point, the following data were recorded: HR, mean arterial pressure (mAP), mPAP, CVP, PAOP, LVEDAI, cRVEF and cRVEDVI. The CO was determined on the basis of the intermittent thermodilution technique as it is considered the clinical gold standard and SVIs were based on

the bolus CO measurements. We analysed cRVEDVI, PAOP, CVP, LVEDAI and cRVEF values with respect to SVI. If arrhythmias occurred during the measurements, the data recorded were discarded and measurements were repeated. A stable haemodynamic condition was a prerequisite for this measurement and was achieved in all patients. To ensure this, infusion of large volumes of colloids or crystalloids or the bolus administration of vasopressors or inotropes was not permitted during the measurements. To reduce the influence of changes in contractility and afterload, each set of measurements was performed in a steady-state period, that is, at least 15 min after the change in dosage of catecholamine or sedatives, infusion rate, or ventilator settings. This was carried out in this manner to avoid changes in the SVI by a factor unrelated to volume status.

Statistical methods

Descriptive statistics were produced for the general, clinical and haemodynamic characteristics of the patients. Mean and SD are presented for normally distributed variables. Minimum–maximum ranges are also presented.

Univariate panel-data fixed effect regression models (where ‘panels’ were patients) were fitted to assess associations between SVI and cRVEDVI, LVEDAI, CVP and PAOP; multivariate models were subsequently fitted. As models including cRVEF (categorized as ≤ 30 , 31–40, >40) and phase of surgical procedure (T0, anaesthesia; Tc, clamping phase; Tr, reperfusion phase and Tf, end of surgery) showed a significant improvement in goodness of fit (as tested by the likelihood ratio test), all multivariate models needed to include this variable. Interaction between the putative predictor variables and cRVEF was also tested. Goodness of fit of the models was assessed by means of r^2 , which shows the proportion of the data variability explained by the fitted model (i.e. in a model with an r^2 of 0.4, the variables included in the model explain 40% of data variability; a model with an r^2 of 0.5 would better fit the data than one with an r^2 of 0.4). The regression coefficient represents the slope of the regression line (the linear relationship between the two variables that is obtained by regression analysis) and indicates the increase in SVI (in ml m^{-2}) per each unit increase in the putative predictor.

Model differences were tested by means of likelihood ratio tests and statistical significance for inclusion of a variable in the multivariate models was considered at the 0.05 level. Statistical analysis was performed with Stata 9.2 (StataCorp LP, College Station, Texas, USA).

Results

Five women and 15 men, aged 52.3 (± 7.7) years (range 35–64 years), with mean body surface area 1.9 (± 0.2) m^2 (range 1.5–2.1 m^2) were enrolled in the study. The underlying diseases necessitating LTx were respectively

Table 1 Haemodynamic data reported as mean \pm SD (range)

Data	T0	Tc	Tr	Tf
HR (beats min^{-1})	75 \pm 15 (51–108)	95 \pm 17* (64–118)	97 \pm 16 (61–120)	91 \pm 19 [†] (54–119)
mAP (mmHg)	76 \pm 15 (54–106)	74 \pm 13 (52–98)	75 \pm 10 (51–95)	73 \pm 11 (51–92)
mPAP (mmHg)	21 \pm 7 (11–35)	18 \pm 5 [‡] (11–31)	25 \pm 7* (16–41)	24 \pm 5 (12–35)
CVP (mmHg)	11 \pm 4 (5–15)	9 \pm 2 (5–12)	11 \pm 2* (8–14)	11 \pm 3 (6–15)
PAOP (mmHg)	13 \pm 4 (6–20)	13 \pm 3 (6–17)	15 \pm 4* (9–22)	15 \pm 3 (10–22)
CI ($\text{l min}^{-1} \text{m}^{-2}$)	4.5 \pm 1.2 (2.4–6.3)	3.4 \pm 1.1 [‡] (2.4–5.7)	5.5 \pm 1.3* (3.9–8.0)	5.3 \pm 1.4 (3.5–8.7)
CCI ($\text{l min}^{-1} \text{m}^{-2}$)	5.2 \pm 1.4 (3.1–7.7)	4.1 \pm 1.3 (1.4–5.9)	6.1 \pm 1.3** (4.0–8.1)	5.5 \pm 1.4 (3.6–9.1)
SVI (ml m^{-2})	61 \pm 13 (34–85)	37 \pm 17* (15–76)	55 \pm 13 [†] (35–76)	54 \pm 11 (35–93)
cRVEDVI (ml m^{-2})	136 \pm 33 (69–183)	108 \pm 31 [‡] (63–187)	129 \pm 29* (67–183)	131 \pm 27 (98–195)
cRVEF (%)	48.9 \pm 11.54 (26–69)	36.8 \pm 11.74 [‡] (17–60)	45.4 \pm 10 [†] (29–68)	45.5 \pm 8.7 (33–68)
LVEDAI (cm m^{-2})	14 \pm 2.0 (11–17)	11.8 \pm 2.6 [†] (8–16)	15.8 \pm 2.5** (12–21)	15.1 \pm 2.5 (12–20)

T0, after anaesthesia induction (during mechanical ventilation); Tc, during anhepatic phase; Tr, at least 30 min after graft reperfusion; Tf, final at the end of surgery. Significantly different from the previous phase. CCI, continuous cardiac index; CI, cardiac index; cRVEDVI, continuous right ventricular end-diastolic volume index; cRVEF, continuous right ventricular ejection fraction; CVP, central venous pressure; HR, heart rate; LVEDAI, left ventricular end-diastolic area index; mAP, mean arterial pressure; mPAP, mean pulmonary arterial pressure; PAOP, pulmonary artery occlusion pressure; SVI, stroke volume index. * $P < 0.001$; ** $P < 0.0001$; [†] $P < 0.01$; [‡] $P < 0.05$.

10 cases of hepatitis C (HCV) cirrhosis, three of hepatitis B (HBV)-related cirrhosis, five of hepatocarcinoma and two of alcoholic cirrhosis. The liver disease classifications were as follows: two patients were categorized as Child Pugh class A; 13 patients, Child Pugh class B; and five, Child Pugh class C. Mean cold ischaemia time was 6.9 (± 0.9) h (range 5.5–8.3 h) and mean anaesthesia time was 9.7 (± 2.3) h (range 5.6–13.5 h). No inotropic drug was administered during the study period. Haemodynamic data are reported as mean \pm SD in Table 1. A total of 80 haemodynamic measurements were analysed.

SVI was strongly associated with cRVEF: in patients with cRVEF higher than 40%, SVI was approximately 16 ml m^{-2} (i.e. coefficient) higher than those with an ejection fraction of 30 or less at univariate and multivariate (Table 2) analyses. In addition, SVI significantly varied with phase of surgical procedure at univariate and multivariate (Table 2) analyses. Therefore, in all subsequent multivariate models, these two variables needed to be included.

At univariate analyses, cRVEDVI, LVEDAI and PAOP were correlated with SVI, and CVP was not (data not shown). Four multivariate models were fitted, including, together with cRVEF and phase, each of the following: cRVEDVI, LVEDAI, PAOP and CVP (Table 3). At multivariate analysis, only cRVEDVI retained statistical significance at the 0.05 level: even after adjusting for

Table 2 Multivariate regression model for stroke volume index and continuous right ventricular ejection fraction, including phases

Variable	Regression coefficient (ml m ⁻²)	P	95% CI	r ²	
cRVEF	31–40 vs. ≤30	5.88	0.29	–5.20 to 16.95	0.47
	>40 vs. ≤30	16.15	0.008	4.37 to 27.94	
Phase	Tc vs. T0	–21.04	<0.001	–28.78 to –13.31	
	Tr vs. T0	–7.09	0.045	–14.04 to –0.15	
	Tf vs. T0	–5.86	0.10	–12.86 to 1.14	

The regression coefficient is reported in ml m⁻². Phase: T0, after anaesthesia induction (during mechanical ventilation); Tc, during anhepatic phase; Tr, at least 30 min after graft reperfusion; Tf, final at the end of surgery. r², coefficient of determination; CI, confidence interval; cRVEF, continuous right ventricular ejection fraction.

cRVEF and phase, the regression coefficient of cRVEDVI in predicting SVI indicates an increase in SVI of 0.21 ml m⁻² for each increase of 1 ml m⁻² of cRVEDVI (Table 4, Fig. 1). Moreover, at multivariate analysis, an increase in LVEDAI of 1 cm m⁻² leads to an increase in SVI of 1.47 ml m⁻² ($P=0.054$) (Table 3, Fig. 2). PAOP and CVP showed a regression coefficient of 0.96 ($P=0.052$; Table 3, Fig. 3) and -0.29 ($P=0.51$), respectively (Table 3). Moreover, the model showing the best fit to the data was, again, that including cRVEDVI (Table 4, Fig. 1), showing an r^2 of 0.60. No interaction between cRVEF and phase was found for any of the variables considered; in particular, the correlation coefficient for cRVEDVI was not statistically different in the three classes of cRVEF; therefore, a single correlation coefficient is reported.

Discussion

The main result of this study is that cRVEDVI and LVEDAI gave a better reflection of preload than filling pressures even if only cRVEDVI reached the statistical significance. We observed that an increase in cRVEDVI of 1 ml m⁻² led to an increase in SVI of 0.21 ml m⁻² (Table 4). LVEDAI showed a trend with SVI but the correlation was not statistically significant. The correlations between SVI and PAOP, and CVP were less strong. We also found a correlation between SVI and cRVEF. As in this study the SVI was strongly associated with cRVEF in all multivariate models in which cRVEF was included. The correlations between SVI and cRVEDVI, LVEDAI, PAOP and CVP were not influenced by cRVEF.

Finally, there still remains the problem based on the possibility that a mathematical coupling could create the

condition of an ‘autocorrelation’ between cRVEDVI and SVI [20,21]. This study was not designed to evaluate the mathematical coupling between the SVI, cRVEF and cRVEDVI. So far we have no new data on whether the correlation we observed is the result of a mathematical coupling or not: our main objective of the study was to evaluate the clinical usefulness of cRVEDVI, CVP and PAOP as a preload indicator, as other studies did in other settings with the same technology [22]. A randomized controlled trial (RCT) designed with this aim in mind could answer this question.

In liver transplanted patients, we found higher cRVEDVI values than in other populations previously reported [23,24]. The high values of cRVEF, cardiac index and SVI observed confirm the presence of a hyperdynamic status in patients with ESLD. A slight decrease in cRVEF, cRVEDVI and LVEDAI was observed only during the anhepatic phase. As previously reported, there is evidence that estimation of right ventricular volumes is superior to conventional pressure monitoring [4–13]. In previous work, we demonstrated that, in LTx surgery, PAOP should not be considered as a preload index [25]. Also in the present study, PAOP failed to correlate with SVI.

At the end of the 1980s, a PAC that was able to calculate both RVEF and RVEDV and that had been validated against radionuclide angiography, contrast ventriculography and echocardiography of the right heart [26–29] was introduced into clinical practice. De Wolf [14], using this first generation of volumetric PAC, described a hyperdynamic haemodynamic system in patients with ESLD. He also demonstrated that, in patients undergoing LTx with normal right ventricular function, CVP is a less reliable

Table 3 Multivariate regression analysis between stroke volume index and continuous right ventricular end-diastolic volume index, pulmonary artery occlusion pressure, central venous pressure and left ventricular end-diastolic area, including covariate continuous right ventricular ejection fraction

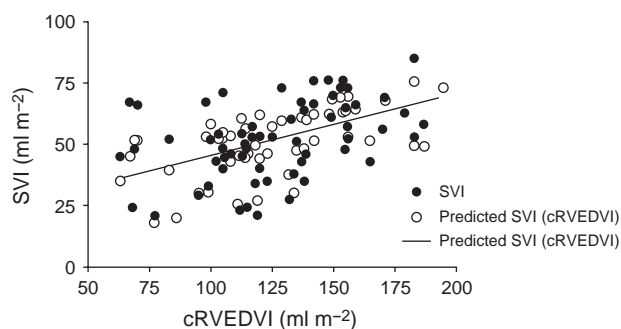
Variable	Regression coefficient (ml m ⁻²)	P	95% CI	r ²	
cRVEDVI	(per unit change)	0.21	<0.001	0.10 to 0.32	0.60
CVP	(per unit change)	–0.29	0.51	–1.45 to 0.87	0.47
PAOP	(per unit change)	0.96	0.052	–0.01 to 1.94	0.48
LVEDAI	(per unit change)	1.47	0.054	–0.03 to 2.97	0.52

The regression coefficient is reported in ml m⁻². Continuous right ventricular ejection fraction not shown. r², coefficient of determination; CI, confidence interval; cRVEDVI, continuous right ventricular end-diastolic volume index; cRVEF, continuous right ventricular ejection fraction; CVP, central venous pressure; LVEDAI, left ventricular end-diastolic area; PAOP, pulmonary artery occlusion pressure.

Table 4 Multivariate regression analysis between stroke volume index and continuous right ventricular end-diastolic volume index, including the covariate continuous right ventricular ejection fraction

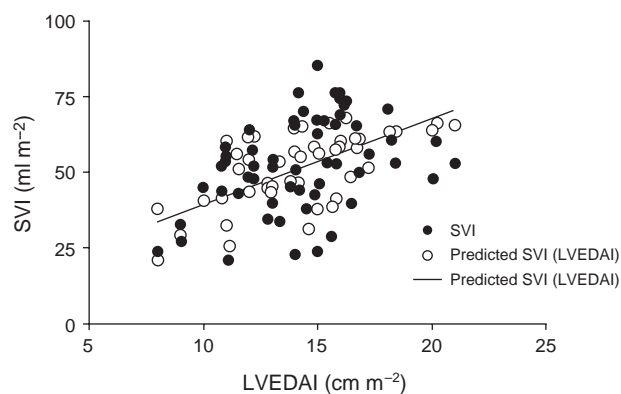
Variable		Regression coefficient (ml m ⁻²)	P	95% CI	r ²
cRVEDVI		0.21	<0.001	0.10 to 0.32	
cRVEF	31–40 vs. ≤30	7.99	0.10	–1.70 to 17.67	
	>40 vs. ≤30	19.95	<0.001	9.51 to 30.39	0.60

The regression coefficient is reported in ml m⁻², r², coefficient of determination; CI, confidence interval; cRVEDVI, continuous right ventricular end-diastolic volume index; cRVEF, continuous right ventricular ejection fraction.

Fig. 1

Scatter plot of stroke volume index against continuous right ventricular end-diastolic volume index. Predicted stroke volume index (SVI) was derived from the already mentioned multivariate regression model (see Table 3). For a visual representation of the relationship between continuous right ventricular end-diastolic volume index (cRVEDVI) and SVI in a two-dimensional space, the regression line of predicted SVI against cRVEDVI (according to the above mentioned model) is also shown.

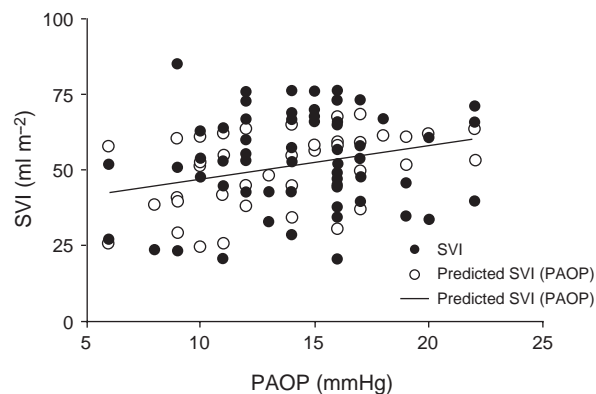
clinical indicator of right ventricular preload than end-diastolic volume. Recently, Hofer *et al.* [22], comparing two thermodilution-based volumetric preload assessment tools with echocardiographic preload monitoring, demonstrated that global end-diastolic volume index (GEDVI),

Fig. 2

Scatter plot of stroke volume index against left ventricular end-diastolic area index. Predicted stroke volume index (SVI) was derived from the already mentioned multivariate regression model (see Table 3). For a visual representation of the relationship between left ventricular end-diastolic area index (LVEDAI) and SVI in a two-dimensional space, the regression line of predicted SVI against LVEDAI (according to the above mentioned model) is also shown.

assessed by PiCCO system, reflected LV preload better than cRVEDVI measured by the modified PAC in cardiac surgical patients.

In the present study, we observed that an increase in LVEDAI of 1 cm m⁻² led to an increase in SVI of 1.47 ml m⁻², but this correlation did not reach statistical significance (Table 3, Fig. 2). TOE should be routinely used during liver transplant surgery even though a recent survey showed that TOE use is more frequent in small volume liver transplant centres [30,31]. The advantage of measuring the LVEDAI is the low probability of error due to the simplicity of the echographic view and of the planimetry tracing, and the disadvantages are that LVEDAI reflects the dimensional variation of a section of the LV in a unique plane and that it is operator dependent [32,33]. Moreover, it can be technically challenging to obtain a good transgastric view due to retraction of the stomach during surgical manoeuvres. Tousignant *et al.* [17] investigated the relation between LV stroke volume and LVEDAI in ICU patients and found only a moderate correlation ($r=0.60$) between single-point estimates of LVEDAI and response to fluid loading. Studies have demonstrated that changes in LVEDAI measured by TOE using endocardial border tracing are closely related to changes in CO and are superior to PAOP in predicting

Fig. 3

Scatter plot of stroke volume index against pulmonary artery occlusion pressure. Predicted stroke volume index (SVI) was derived from the already mentioned multivariate regression model (see Table 3). For a visual representation of the relationship between pulmonary artery occlusion pressure (PAOP) and SVI in a two-dimensional space, the regression line of predicted SVI against PAOP (according to the above mentioned model) is also shown.

the ventricular preload associated with maximum CO [18]. Although a small end-diastolic area generally indicates hypovolaemia, a large end-diastolic area may or may not indicate adequate preload in patients with LV dysfunction. This is analogous to LV end-diastolic pressure measurements in which an LV end-diastolic pressure of 15–20 mmHg, which usually represents a normal to hypervolaemic state in patients with normal LV function, can represent inadequate preload for patients with LV dysfunction. Also, in conditions presenting a low systemic vascular resistance, such as early sepsis, LV emptying will be improved because of the lowered afterload. In this situation, it may be difficult to differentiate hypovolaemia from low systemic vascular resistance by echocardiography alone, as both conditions will lead to a decreased end-diastolic area [34]. Moreover, it should be considered that the value of LVEDAI as a determinant of preload during surgery is dependent on the maintenance of contractility and compliance and must be matched with the first value [35].

Recently, Wiesenack *et al.* [24] showed that an increased cardiac preload was more reliably reflected by cRVEDVI than by LVEDI, CVP or PAOP in the preoperative cardiac surgery setting even if the response of SVI subsequent to fluid administration was better predicted by LVEDAI than by cRVEDVI. In the current study, we did not investigate fluid responsiveness. Even if a trend can be observed (Fig. 2), the correlation between LVEDAI and SVI did not reach statistical significance. These findings emphasize the application of TOE to detect acute and rapid changes in haemodynamics during liver transplant surgery. Although both thermodilution-derived cRVEDVI and TOE are interesting tools to evaluate preload, their use is still limited.

The conclusions drawn from these findings should be viewed with caution for several reasons. First, the small sample size observed in a single centre could limit the generalizability of the results and precision of the estimates. Second, the cRVEDVI was assessed here as a substitute for LV preload even if, of course, right ventricular function is different from LV function. The cRVEDVI value may be influenced by interventricular dependence, right ventricular dysfunction and increased right ventricular afterload. Therefore, the relationship between right ventricular preload assessment and CO readings may be weak; cRVEDVI has not been tested in patients with left or right-heart ventricular failure and its value in this context is unknown. Third, the use of the volumetric PAC requires correct catheter positioning to maximize the accuracy and reproducibility of measurements; assessment is recommended to ensure that this is the case. The administration of large volumes of fluid over a short period of time can result in erroneous measurements. Rapid changes in the baseline temperature of the body (hypothermia or hyperthermia) can contribute

to variations in measurements. Another situation that may contribute to unreliable measurements is the presence of a tachycardia (at rates of <150 beats min^{-1}).

In conclusion, cRVEDVI and LVEDAI better reflect preload than filling pressures during LTx surgery even if only cRVEDVI reached statistical significance. Instead of filling pressures, the use of thermodilution-derived cRVEDVI and/or TOE as direct preload monitoring in liver transplant surgery should be encouraged. Randomized clinical trials with larger populations are required to evaluate further the impact of cRVEDVI and LVEDAI in clinical practice.

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References

- 1 Shippy CR, Apple PL, Shoemaker WC. Reliability of clinical monitoring to assess blood volume in critically ill patients. *Crit Care Med* 1984; **12**:107–112.
- 2 Hansen RM, Viquerat CE, Matthay MA, *et al.* Poor correlation between pulmonary arterial wedge pressure and left ventricular end-diastolic volume after coronary artery bypass graft surgery. *Anesthesiology* 1986; **64**:764–770.
- 3 Kumar A, Anel R, Bunnell E, *et al.* Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects. *Crit Care Med* 2004; **32**:691–699.
- 4 Raper R, Sibbald WJ. Misled by the wedge? The Swan-Ganz catheter and left ventricular preload. *Chest* 1986; **89**:427–434.
- 5 Diebel LN, Wilson RF, Tagett MG, Kline RA. End-diastolic volume: a better indicator of preload in the critically ill. *Arch Surg* 1992; **127**:817–821.
- 6 Diebel L, Wilson RF, Heins J, *et al.* End-diastolic volume versus pulmonary artery wedge pressure in evaluating cardiac preload in trauma patients. *J Trauma* 1994; **37**:950–955.
- 7 Durham R, Neuaber K, Vogler G, *et al.* Right ventricular end diastolic volume as a measure of preload. *J Trauma* 1995; **39**:218–223.
- 8 Yu M, Takiguchi S, Takanishi D, *et al.* Evaluation of the clinical usefulness of the thermodilution volumetric catheters. *Crit Care Med* 1995; **23**:681–686.
- 9 Chang MC, Blinman TA, Rutherford EJ, *et al.* Preload assessment in trauma patients during large volume shock resuscitation. *Arch Surg* 1996; **131**:728–731.
- 10 Dhainaut JF, Brunet F, Monsallier JF, *et al.* Bedside evaluation of right ventricular performance using a rapid computerized thermodilution method. *Crit Care Med* 1987; **15**:148–152.
- 11 Cheatham ML, Safcsak K, Block EFJ, Nelson LD. Preload assessment in patients with an open abdomen. *J Trauma* 1999; **46**:16–22.
- 12 Cheatham ML, Nelson LD, Chang MC, Safcsak K. Right ventricular end diastolic volume index as a predictor of preload status in patients on positive end expiratory pressure. *Crit Care Med* 1998; **26**:1801–1806.
- 13 Diebel LN, Myers T, Dulchavsky S. Effects of increasing airway pressure and PEEP on the assessment of cardiac preload. *J Trauma* 1997; **42**:585–591.
- 14 De Wolf A. Transesophageal echocardiography and orthotopic liver transplantation: general concepts. *Liver Transpl Surg* 1999; **5**:339–340.
- 15 Buhre W, Buhre K, Kazmaier S, *et al.* Assessment of cardiac preload by indicator dilution and transesophageal echocardiography. *Eur J Anaesthesiol* 2001; **18**:662–667.
- 16 Hinder F, Poelaert JI, Schmidt C, *et al.* Assessment of cardiovascular volume status by transoesophageal echocardiography and dye dilution during cardiac surgery. *Eur J Anaesthesiol* 1998; **15**:633–640.
- 17 Tousignant CP, Walsh F, Mazer CD. The use of TEE for preload assessment in critically ill patients. *Anesth Analg* 2000; **90**:351–355.
- 18 Swenson JD, Harkin C, Pace NL, *et al.* Transesophageal echocardiography: an objective tool in defining maximum ventricular response to intravenous fluid therapy. *Anesth Analg* 1996; **83**:1149–1153.

- 19 Hourani JM, Bellamy PE, Tashkin DP, *et al.* Pulmonary dysfunction in advanced liver disease: frequent occurrence of an abnormal diffusing capacity. *Am J Med* 1991; **90**:693–700.
- 20 Chang MC, Black CS, Meredith JW. Volumetric assessment of preload in trauma patients: addressing the problem of the mathematical coupling. *Shock* 1996; **6**:326–329.
- 21 Nelson LD, Safcsak K, Cheatham ML, Block EF. Mathematical coupling does not explain the relationship between right ventricular end-diastolic volume and cardiac output. *Crit Care Med* 2001; **29**:940–943.
- 22 Hofer CK, Furrer L, Matter-Ensner S, *et al.* Volumetric preload measurement by thermodilution: a comparison with transoesophageal echocardiography. *Br J Anaesth* 2005; **94**:749–755.
- 23 Ishida T, Lee T, Shimabukuro T, Niinami H. Right ventricular end-diastolic volume monitoring after cardiac surgery. *Ann Thorac Cardiovasc Surg* 2004; **10**:167–170.
- 24 Wiesenack C, Fiegl C, Keyser A, *et al.* Continuously assessed right ventricular end-diastolic volume as a marker of cardiac preload and fluid responsiveness in mechanically ventilated cardiac surgical patients. *Crit Care* 2005; **9**:R226–R233.
- 25 Della Rocca G, Costa MG, Coccia C, *et al.* Preload and haemodynamic assessment during liver transplantation. A comparison between pulmonary artery catheter and transpulmonary indicator dilution technique. *Eur J Anaesth* 2002; **19**:868–875.
- 26 Urban P, Scheigger D, Gabathuler J, Rutishauser W. Thermodilution determination of right ventricular volume and ejection fraction: a comparison with biplane angiography. *Crit Care Med* 1987; **15**:652–655.
- 27 Jardin F, Gueret P, Dubourg O, *et al.* Right ventricular volumes by thermodilution in the adult respiratory distress syndrome: a comparative study using two-dimensional echocardiography as a reference method. *Chest* 1985; **88**:34–39.
- 28 Malm S, Frigstad S, Sasberg E, *et al.* Accurate and reproducible measurement of left ventricular volume and ejection fraction by contrast echocardiography: a comparison with magnetic resonance imaging. *J Am Coll Cardiol* 2004; **44**:1030–1035.
- 29 De Simone R, Wolf I, Mottl-Link S, *et al.* Intraoperative assessment of right ventricular volume and function. *Eur J Cardiothorac Surg* 2005; **27**:988–1033.
- 30 Schumann R. Intraoperative resource utilization in anesthesia for liver transplantation in the United States: a survey. *Anesth Analg* 2003; **97**:21–28.
- 31 Steltzer H, Blazek G, Gabriel A, *et al.* Two-dimensional transesophageal echocardiography in early diagnosis and treatment of hemodynamic disturbances during liver transplantation. *Transplant Proc* 1991; **23**:1957–1958.
- 32 Townend JN, Hutton P. Transoesophageal echocardiography in anaesthesia and intensive care. *Br J Anaesth* 1996; **77**:137–139.
- 33 Denault AY, Couture P, McKenty S, *et al.* Perioperative use of transoesophageal echocardiography by anesthesiologists: impact in noncardiac surgery and in the intensive care unit. *Can J Anesth* 2002; **49**:287–293.
- 34 Beaulieu Y, Marik PE. Bedside ultrasonography in the ICU: part 1. *Chest* 2005; **128**:881–895.
- 35 Cheung AT, Savino JS, Weiss SJ, *et al.* Echocardiographic and hemodynamic indexes of left ventricular preload in patients with normal and abnormal ventricular function. *Circulation* 2003; **108**:226–229.