ORIGINAL ARTICLE

Continuous Right Ventricular End Diastolic Volume and Right Ventricular Ejection Fraction During Liver Transplantation: A Multicenter Study

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Cardiac preload is traditionally considered to be represented by its filling pressures, but more recently, estimations of end diastolic volume of the left or right ventricle have been shown to better reflect preload. One method of determining volumes is the evaluation of the continuous right ventricular end diastolic volume index (cRVEDVI) on the basis of the cardiac output thermodilution technique. Because preload and myocardial contractility are the main factors determining cardiac output during liver transplantation (LTx), accurate determination of preload is important. Thus, monitoring of cRVEDVI and cRVEF should help with fluid management and with the assessment of the need for inotropic and vasoactive agents. In this multicenter study, we looked for possible relationships between the stroke volume index (SVI) and cRVEDVI, cRVEF, and filling pressures at 4 predefined steps in 244 patients undergoing LTx. Univariate and multivariate autoregression models (across phases of the surgical procedure) were fitted to assess the possible association between SVI and cRVEDVI, pulmonary artery occlusion pressure (PAOP), and central venous pressure (CVP) after adjustment for cRVEF (categorized as ≤30, 31-40, and >40%). SVI was strongly associated with both cRVEDVI and cRVEF. The model showing the best fit to the data was that including cRVEDVI. Even after adjustment for cRVEF, there was a statistically significant ($P < 0.05$) relationship between SVI and cRVEDVI with a regression coefficient (slope of the regression line) of 0.25; this meant that an increase in cRVEDVI of 1 mL m^{-2} resulted in an increase in SVI of 0.25 mL m^{-2}. The correlations between SVI and CVP and PAOP were less strong. We conclude that cRVEDVI reflected preload better than CVP and PAOP. ***Liver Transpl 14: 327-332, 2008.*** © **2008 AASLD.**

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Major surgery may result in large decreases in preload and cardiac output (CO), potentially resulting in tissue hypoperfusion and poor outcome.[1,2] Maintaining a high CO in a liver transplant recipient is essential in order to ensure adequate tissue perfusion. To maintain a high CO, preload has to be maintained in the presence of hemorrhage, third space losses, and ongoing ascites

Abbreviations: CCO, continuous cardiac output; CI, cardiac index; CO, cardiac output; cRVEDVI, continuous right ventricular end diastolic volume index; cRVEF, continuous right ventricular ejection fraction; CVP, central venous pressure; HR, heart rate; LTx, liver transplantation; LVEDAI, left ventricular end diastolic area index; mAP, mean arterial pressure; mPAP, mean pulmonary arterial pressure; PAOP, pulmonary artery occlusion pressure; r^2, coefficient of determination; RVEDVI, right ventricular end diastolic volume index; RVEF, right ventricular ejection fraction; SVI, stroke volume index; τ, exponential decay time constant; T0, after anesthesia induction; Tc, during the anhepatic phase; Tr, at least 30 minutes after graft reperfusion; Tf, at the end of surgery.

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production. In addition, venous return can be further compromised by temporary clamping of the inferior vena cava, which also results in venous congestion of tissues below the diaphragm. However, overly aggressive volume replacement can cause significant volume overload and should be avoided.

Traditional estimates of intravascular volume status such as central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) correlate poorly with changes in CO in the critically ill patient.[3-6] According to the Frank-Starling principle, the vigor of cardiac contraction is related directly to muscle fiber length at end diastole. Although monitoring of muscle fiber length would be ideal, it has been shown that end diastolic volume of the left ventricle is a better indicator of muscle fiber length than PAOP. These relationships are also valid for the right ventricle. Current technology, based on echocardiography or thermodilution, allows us to estimate ventricular end diastolic volume.[7,8] There is indeed a renewed interest in pulmonary artery catheter (PAC) cardiac output that allows calculation of right ventricular volumes on the basis of the thermodilution technique, and end diastolic volume of the right ventricle reflects preload better than CVP.[9-12] The first generation of such catheters was introduced in the 1980s.[13-18] The current generation of PAC was introduced in the late 1990s. It allows dual monitoring of the continuous cardiac output (CCO) or index, continuous right ventricular ejection fraction (cRVEF), and continuous right ventricular end diastolic volume index (cRVEDVI).

The present study was designed to find relationships between the stroke volume index (SVI) and cRVEDVI, CVP, and PAOP in patients undergoing liver transplantation (LTx) with the current generation of technology.

PATIENTS AND METHODS

Patients

In this multicenter study, we obtained approval from the ethics committees and written informed consent from 244 consecutive patients who were about to undergo LTx. Patients with preexisting pulmonary and/or cardiac diseases and patients with fulminant hepatic failure, hepatopulmonary syndrome, or pulmonary hypertension were excluded.

Standard monitoring consisted of 2-lead electrocardiography (II/V5), pulse oximetry, direct arterial pressure monitoring (radial artery catheter), and multigas analysis. Anesthetic management was standardized and consisted of propofol (0.5 mg kg^{-1}) or midazolam (0.04-0.07 mg kg^{-1}) for the induction, cisatracurium besilate (0.15 mg kg^{-1}) as a muscle relaxant, and alfentanil (7-10 μg kg^{-1}) or fentanyl (2-5 μg kg^{-1}) for analgesia. Anesthesia was maintained with sevoflurane (0.8%) or desflurane (4%-6%) and remifentanil continuous infusion (0.1-0.5 μg kg^{-1} $minute^{-1}$). Mechanical ventilation with a volumetric anesthesia ventilator included an end expiratory pressure of 5 cm H_2O.

The surgical technique consisted of a piggyback procedure without venovenous bypass. Cathecholamines were administered on graft reperfusion when necessary.

Cardiopulmonary Monitoring

A 8.0 French PAC (Swan-Ganz CCOmbo CCO/SvO_2/CEDV/VIP 777HF8 catheter, Edwards Lifesciences, Irvine, CA) was placed through the right internal jugular vein. The PAC was connected to a Vigilance monitor (Edwards Lifesciences) for intermittent CO, CVP, PAOP, mean pulmonary artery pressure, body temperature, cRVEDVI, CCO, continuous mixed venous oxygen saturation, cRVEF, and continuous SVI measurements. Intermittent CO measurements were made by manual injection of a 10-mL saline solution, at room temperature, into the right atrium. Three consecutive boluses were injected without regard to the phase of the respiratory cycle over a 2-minute period and without interruption of mechanical ventilation. In cases in which there was a >10% discrepancy in the CO measurements, the measurements were repeated 5 times, and the lowest and highest results were discarded.

The new cRVEF algorithm generates a relaxation waveform resembling the bolus thermodilution washout decay curve. The waveform is based on the repeating on-off input signal and is generated by the accumulation of the temperature change for each on and off segment of the input signal. cRVEF is calculated on the basis of the estimation of the exponential decay time constant (τ) of this curve and heart rate (HR): $cRVEF = 1 - \exp[-60/(\tau \times HR)]$. cRVEDV is calculated as (CCO/HR)/cRVEF.

Experimental Procedure

After the induction of anesthesia and placement of the catheters, hemodynamic data were collected during hemodynamic stability at 4 time points: 30 minutes after the induction of anesthesia and catheter placement but before skin incision (T0), 20 minutes after the start of the anhepatic stage (Tc), at least 30 minutes after graft reperfusion (Tr), and finally at the end of surgery after skin closure (Tf). The following hemodynamic data were recorded: HR, mean arterial pressure, mean pulmonary arterial pressure, CVP, PAOP, cRVEF, and cRVEDVI. CO was measured with the intermittent thermodilution technique as this is considered to be the gold standard, and SVI was based on bolus CO measurements. Each set of measurements was performed during a steady-state period, that is, at least 15 minutes after any change in the infusion rate of catecholamines or sedatives and ventilator settings.

Statistical Methods

Descriptive statistics were used for demographic, clinical, and hemodynamic data. Means and standard deviations are presented for normally distributed data, and medians and interquartile ranges are presented for nonnormally distributed data. Minimum-maximum ranges are also presented. To describe the relationship between SVI and preload indices (cRVEDVI, PAOP, and

TABLE 1. Hemodynamic Data Reported as Mean (Standard Deviation) and Range

	T0	Tc	Tr	Tf
HR (b min^{-1})	76 (16)	90 (18)†	90 (16)	87 (15)‡
	47–140	53–139	51–133	48–150
mAP (mm Hg)	77 (16)	79 (17)	74 (17)†	78 (15)†
	58–132	40–124	27–128	45–126
mPAP (mm Hg)	23 (6)	20 (7)*	24 (7)†	24 (6)
	11–42	8–40	8–41	9–45
CVP (mm Hg)	12 (4)	10 (5)*	13 (5)†	13 (5)
	3–23	1–23	2–25	2–26
PAOP (mm Hg)	15 (5)	14 (5)*	16 (5)†	16 (5)
	4–26	4–25	5–27	4–28
CI (L min^{-1} m^{-2})	4.5 (1.6)	4.3 (1.4)§	5.3 (1.7)†	5.2 (1.5)
	(1.7–9.5)	(1.3–9.6)	(2.1–9.9)	(1.5–10.4)
SVI (mL m^{-2})	58 (16)	49 (16)†	58 (16)†	59 (16)
	23–106	20–98	17–99	15–99
cRVEDVI (mL m^{-2})	135 (26)	122 (29)†	128 (28)†	134 (26)‡
	82–198	63–195	53–195	74–198
cRVEF (%)	45 (11)	42 (10)*	46 (9)†	44 (9)‡
	25–79	23–75	25–72	20–74

NOTE: *P* values indicate a significant difference from the previous phase.
Abbreviations: CI, cardiac index; cRVEDVI, continuous right ventricular end diastolic volume index; cRVEF, continuous right ventricular ejection fraction; CVP, central venous pressure; HR, heart rate; mAP, mean arterial pressure; mPAP, mean pulmonary arterial pressure; PAOP, pulmonary artery occlusion pressure; SVI, stroke volume index; T0, after anesthesia induction; Tc, during the anhepatic phase; Tf, at the end of surgery; Tr, at least 30 minutes after graft reperfusion.
*$P \leq 0.001$.
†$P < 0.0001$.
‡$P < 0.01$.
§$P < 0.05$.

CVP) at different time points, Pearson's correlation coefficient was calculated.

Univariate panel-data fixed-effect autoregression models were fitted to assess associations between SVI and cRVEDVI, CVP, PAOP, and cRVEF; multivariate models were subsequently fitted. Because models including cRVEF (categorized as ≤30, 31-40, and >40%) showed a slight but significant improvement in goodness of fit (as tested by the likelihood ratio test), all multivariate models needed to include this variable. Panels were patients, and observation times were phases of the surgical procedure (T0, Tc, Tr, and Tf). Interaction between the putative predictor variables and cRVEF was also tested. Goodness of fit of the models was reflected by the coefficient of determination (r^2), which shows the proportion of the data variability explained by the fitted model (that is, in a model with r^2 of 0.4, the variables included in the model explain 40% of data variability; a model with r^2 of 0.5 fits the data better than one with r^2 of 0.4). The regression coefficient represents the slope of the regression line (the linear relationship between the 2 variables that is obtained by regression analysis) and indicates the increase in SVI (in mL m^{-2}) per unit increase in the putative predictor.

Model differences were tested by means of likelihood ratio tests. Statistical significance for the inclusion of a variable in the multivariate models was considered at the 0.05 level. Statistical analyses were performed with State 9.2 (StateCorp, Texas).

RESULTS

We enrolled 244 consecutive patients (173 male, 71 female) into the study. The mean age was 50.6 (8.1) years (range: 18-67); the mean body surface area was 1.79 (0.14) m^2 (range: 1.07-2.28). The underlying diseases necessitating LTx were viral cirrhosis (n = 135), hepatocellular carcinoma on cirrhosis (n = 48), alcoholic cirrhosis (n = 42), cryptogenic cirrhosis (n = 8), cholestatic cirrhosis (n = 6), and other (n = 5). The number of patients enrolled from each participating center was as follows: Padova, 68 patients; Modena, 48 patients; Udine, 47 patients; Pisa, 39 patients; Rome, 22 patients; and Bologna, 20 patients.

No inotropic drug was used during the study period. A total of 995 hemodynamic measurements were collected (Table 1). The cardiac index (CI), SVI, and cRVEF as well as preload indicators (CVP, PAOP, and cRVEDVI) decreased at the anhepatic stage in comparison with baseline values, they returned to baseline values upon reperfusion. HR increased only at the anhepatic stage. These parameters at the end of surgery were the same as at baseline.

According to univariate analysis, SVI significantly correlated with cRVEDVI, CVP, and PAOP (all $P < 0.05$), although the overall r^2 values of the models were low (0.23, 0.027, and 0.019, respectively); the regression coefficients were 0.26, 1.50, and 1.63, respectively. There was a correlation between SVI and cRVEF: in patients with cRVEF between 30 and 40%, the regres-

TABLE 2. Multivariate Regression Analysis Between SVI and cRVEDVI Including the Covariate cRVEF

Variable	Regression Coefficient (mL m^{-2})	*P*	95% Confidence Interval	r^2
cRVEDVI	0.25	<0.001	0.19–0.30	
cRVEF				
31-40 versus ≤30	8.28	0.001	3.26–13.31	
>40 versus ≤30	11.80	<0.001	6.24–17.35	
				0.30

Abbreviations: cRVEDVI, continuous right ventricular end diastolic volume index; cRVEF, continuous right ventricular ejection fraction; r^2, coefficient of determination; SVI, stroke volume index.

TABLE 3. Correlation Coefficients Between SVI and cRVEDVI, PAOP, CVP, and cRVEF at Four Predefined Steps

	Phases			
	T0	Tc	Tr	Tf
SVI versus cRVEDVI	0.40*	0.42*	0.46*	0.46*
SVI versus PAOP	0.21	0.23	0.1	−0.01
SVI versus CVP	0.18	0.15	0.19	−0.007
SVI versus cRVEF	0.43*	0.40*	0.31*	0.45*

NOTE: *P* values after Bonferroni correction are presented.
Abbreviations: cRVEDVI, continuous right ventricular end diastolic volume index; cRVEF, continuous right ventricular ejection fraction; CVP, central venous pressure; PAOP, pulmonary artery occlusion pressure; SVI, stroke volume index; T0, after anesthesia induction; Tc, during the anhepatic phase; Tf, at the end of surgery; Tr, at least 30 minutes after graft reperfusion.
*$P < 0.01$.

sion coefficient was 10.3 mL m^{-2} higher than in patients with cRVEF ≤ 30% (95% confidence interval: 5.40-15.20, $P < 0.001$); in those with cRVEF > 40%, the regression coefficient was 13.82 mL m^{-2} higher than in patients with cRVEF ≤ 30% (95% confidence interval: 8.42-19.21, $P < 0.001$).

With multivariate analysis (that is, after adjustment for cRVEF), the results did not change: SVI still correlated with cRVEDVI, CVP, and PAOP, again with the highest r^2 value for cRVEDVI. In Table 2, only the results for the correlations between SVI and cRVEDVI and between SVI and cRVEF are shown. The r^2 values for the regression analysis between SVI and cRVEDVI, CVP, and PAOP were not influenced by the subcategory of cRVEF. Finally, Pearson's correlation analysis at each of the 4 predefined time points between SVI and cRVEDVI, PAOP, CVP, and cRVEF revealed similar correlation coefficients (Table 3).

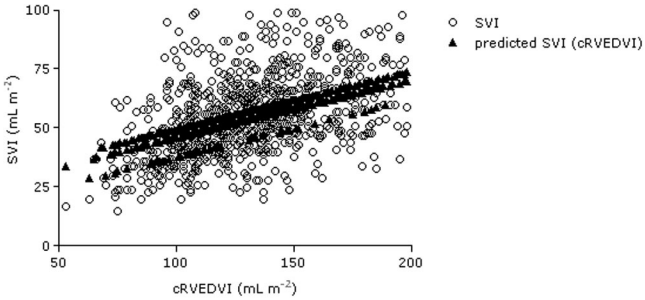

Figure 1. Predicted SVI was derived from the already mentioned multivariate regression model (see table 2). SVI: stroke volume index; cRVEDVI: continuous right ventricular end diastolic volume index. NB predicted values appear to lie on three lines, one for each range of cRVEF. These lines are parallel, indicating that no interaction between cRVEF and cRVEDVI was found.

DISCUSSION

Our results indicate that in LTx patients, cRVEDVI is a better reflection of preload than CVP and PAOP on the basis of the strong correlation between SVI and cRVEDVI. We observed that an increase in cRVEDVI of 1 mL m^{-2} leads to an increase in SVI of 0.25 mL m^{-2} (Fig. 1 and Table 2). The correlations between SVI and CVP and PAOP were less strong. We also found a strong correlation between SVI and cRVEF. The correlations between SVI and cRVEDVI, PAOP, CVP, and cRVEF were similar at different stages of the procedure, and the correlations between SVI and cRVEDVI, PAOP, and CVP were not influenced by cRVEF.

Patients undergoing LTx may develop significant hemodynamic instability, especially during the anhepatic stage, immediately after graft reperfusion, and in the early postoperative phase.[19] Optimal treatment requires diagnosis of the cause of the hypotension: low preload versus low CO versus low systemic vascular resistance. Monitoring of preload [right ventricular end

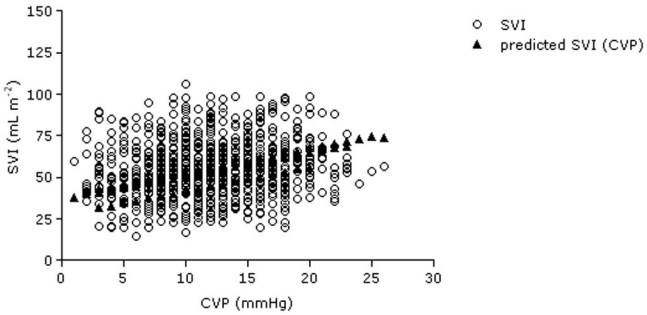

Figure 2. Predicted SVI was derived from the already mentioned multivariate regression model (see table 2). SVI: stroke volume index; PAOP: pulmonary artery occlusion pressure. NB predicted values appear to lie on three lines, one for each range of cRVEF. These lines are parallel, indicating that no interaction between cRVEF and PAOP was found.

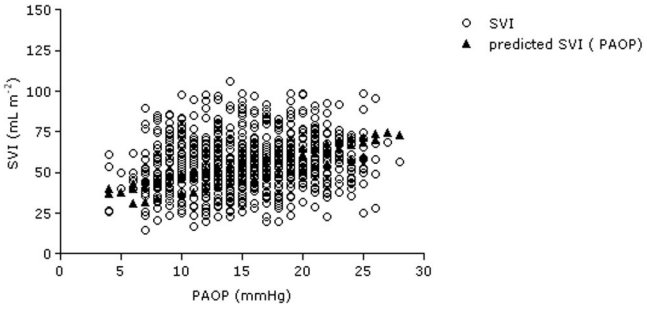

Figure 3. Predicted SVI was derived from the already mentioned multivariate regression model (see table 2). SVI: stroke volume index; CVP: central venous pressure. NB predicted values appear to lie on three lines, one for each range of cRVEF. These lines are parallel, indicating that no interaction between cRVEF and CVP was found.

diastolic volume index (RVEDVI)] and an index of contractility [right ventricular ejection fraction (RVEF)] helps in the differential diagnosis and guides hemodynamic management. Low CI can be treated by volume administration when RVEDVI is low and by inotropic support if RVEF is low after exclusion of other causes of decreased contractility (for example, low ionized calcium concentration or low pH) in order to avoid massive volume overload. Filling pressures do not reliably predict preload status, particularly in patients mechanically ventilated, with impaired heart function, or otherwise critically ill.[10,11] Several studies have confirmed that RVEDVI shows higher correlation with CO than CVP and PAOP.[5,6,14-16] Because of its geometrical complexity, the assessment of right ventricular volume remains today a very difficult task. Although recently De Simone and colleagues[20] demonstrated that intraoperative assessment of right ventricular volumes by means of thermodilution and transesophageal 3-dimensional echocardiography is feasible, they found that RVEDVI by thermodilution was larger than that determined by echocardiography, mainly because of the geometry of the right ventricle and the differences in technique. Despite these different results, the presence of significant agreement in measuring RVEF confirmed that these 2 methods can be reliably used for serial measuring of RVEDVI and right ventricular function during surgery.[20] Recently, Wiesenack and coworkers[21] showed that cardiac preload is more reliably reflected by cRVEDVI than by CVP, PAOP, or left ventricular end diastolic area index (LVEDAI), but cRVEDVI could not predict the response to a fluid challenge in patients undergoing elective coronary surgery.

In our patients who underwent LTx, we found higher cRVEDVI values than in other populations.[21,22] The high cRVEF, CI, and SVI confirm the presence of a hyperdynamic hemodynamic status in patients with end-stage liver disease. A slight decrease in cRVEF was observed only during the anhepatic stage. In 1993, De Wolf and coworkers[23] observed a hyperdynamic hemodynamic system in such patients with high RVEF, HR, and CI with low systemic vascular resisting index. Their study was performed with the first-generation thermodilution-based technology for the measurement of the right ventricular volumes and function. They found a significant correlation between SVI and RVEDVI over a wide range of RVEDVI (60-185 mL m^{-2}). The authors concluded that in patients undergoing LTx with normal right ventricular function, RVEDVI is a better clinical indicator of right ventricular preload than CVP. In 2005, Siniscalchi and coworkers[24] demonstrated in 21 LTx patients a linear relationship between SVI and cRVEDVI with r^2 values of 0.49 (T1), 0.57 (T2), 0.51 (T3), and 0.44 (T4). They did not find a correlation between SVI and CVP or PAOP. In this population, cRVEF was 36% (standard deviation: 4%). The authors concluded that cRVEDVI may be the best clinical estimate of right ventricular preload. However, they did not analyze the influence of time and of different levels of cRVEF on the relationship between SVI and cRVEDVI.

During anesthesia for LTx, 3-dimensional echocardiography can be used, although it is not considered to be a routine monitoring technique.[25-27] In particular, the short-axis view of the left ventricle (transgastric view) provides excellent visualization of the left ventricular dimension and function, and LVEDAI correlates well with changes in SVI during volume therapy.[28,29] The advantage of measuring LVEDAI is the low probability of errors due to the simplicity of the echographic view and of the planimetry tracing, whereas the disadvantage is that LVEDAI reflects the dimensional variation of a section of the left ventricle in a unique plane.[29] Unfortunately, the transgastric view is unavailable during most of the LTx procedure because of posterior retraction of the stomach.

Our study has several strengths. It is a large multicenter trial involving 244 patients undergoing LTx. Data were obtained with the last generation of CCO catheters and monitors. Also, to our knowledge, it is the first study analyzing the correlation between SVI and cRVEDVI in LTx patients using a multivariate regression model including cRVEF.

Some limitations of the present study have to be considered. cRVEDVI shows a delayed reactivity to rapid changes of intravascular volume. Also, inaccuracies in the measurement of cRVEDVI can result from poor po-

sitioning of the injectate port in relation to the tricuspid valve and of the thermistor in relation to the pulmonic valve. Additionally, sinus tachycardia, cardiac arrhythmias, hypovolemia, the timing of injection of the indicator in relation to the respiratory cycle, and the formation of thrombus on the catheter can also affect the accuracy of measurements. Finally, mathematical coupling (due to the fact that cRVEDVI is calculated with the SVI, which, in turn, is derived from the CI measurements) is a potential problem, but Chang et al.[30] and Nelson et al.[31] concluded that this was not a real concern.

In conclusion, cRVEDVI is a better preload index than filling pressures, and it is independent of cRVEF. Clinical management of low CO should be guided by both cRVEDVI and cRVEF to avoid underresuscitation or overresuscitation.

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