Right Ventricular End-Diastolic Volume Index as a Predictor of Preload Status in Patients With Low Right Ventricular Ejection Fraction During Orthotopic Liver Transplantation

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ABSTRACT

Objective. The objective of this study was to compare the accuracy of 2 variables: pulmonary artery occlusion pressure (PAOP) and right ventricular end diastolic volume index (RVEDVI) as predictors of the hemodynamic response to fluid challenge as well as definition of the overall correlation between RVEDVI and change in PAOP, right ventricular ejection fraction (RVEF), central venous pressure (CVP), and determination of the right ventricular function during orthotopic liver transplantation.

Materials and Methods. A modified pulmonary artery catheter equipped with a fast response thermistor was used to determine RVEF, allowing calculation of RVEF end-diastolic volume index (EDVI, as the ratio of stroke index [SI] to EF). The above-mentioned hemodynamic measures were taken in 4 phases: T0, after induction of anesthesia; T1, during anhepatic phase; T2, 30' after graft reperfusion; and T3, at the end of surgery.

Results. The variation of the REF value was $36 \pm 4\%$ and $39 \pm 6\%$. Linear regression analysis showed a significant correlation between RVEDVI (range, 133 ± 33 – 145 ± 40 mL/m²) and stroke volume index (SVI) in each phase ($r^2 = 0.49$, $P < .01$; $r^2 = 0.57$, $P < .01$) at T0 and T1, respectively, and at T2 and T3 ($r^2 = 0.51$, $P < .01$; $r^2 = 0.44$, $P < .01$), respectively. No significant variations in the linear regression analysis between RVEDVI, PAOP, CVP, and RVEF were observed. No relationship was found between PAOP (range, 10 ± 2 – 6 ± 2 mm Hg) and SVI.

Conclusion. RVEDVI may be the best clinical estimate of right ventricular preload. In fact, minor changes of RVEF have been recorded, confirming that RV function was not altered during uncomplicated orthotopic liver transplantation.

A MODIFIED pulmonary artery catheter equipped with a fast response thermistor allows bedside assessment of right ventricular ejection fraction (RVEF), permitting the calculation of right ventricular end diastolic volume index (RVEDVI), and evaluation of ventricular filling.¹ A large number of articles have emphasized the right correlation between RVEDVI and cardiac index (CI) or stroke volume (SV).^{2,3} Others studies suggest a low response to fluid challenge associated with a low EF.^{4,5} Patients with a low EF showed a decrease in CI after volume expansion, which was probably attributed to right ventricular overdistension. Because of these observations, we designed a prospective study to further evaluate this volumetric assess-

ment of the hemodynamic response to fluid challenge during orthotopic liver transplantation (OLT) among patients with EF 40%. The 3 specific purposes of the study were to compare the accuracy of 2 variables: pulmonary artery occlusion pressure (PAOP) and RVEDVI, as predic-

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tors of the hemodynamic response to fluid challenge, as well as to define the overall correlation between RVEDVI and change in pulmonary artery occlusion pressure (PAOP), REF, and central venous pressure (CVP) as evidence of right ventricular function during OLT.

MATERIALS AND METHODS

Twenty-one patients undergoing liver transplantation had the indications: hepatitis C virus (HCV)-related cirrhosis ($n = 13$); hepatitis B virus (HBV)-related cirrhosis ($n = 2$); sclerosing cholangitis ($n = 3$); and exotoxic cirrhosis ($n = 3$). We excluded from this study patients with end-stage valvular heart disease, cardiac amyloidosis, hypertrophic cardiomyopathy, active alcoholism, chronic obstructive pulmonary disease, or recent acute myocardial infarction (<6 months). All patients underwent right catheterization, as directed by the diagnostic protocol for liver transplant eligibility. After induction of anesthesia, we inserted a modified Swan-Ganz thermodilution catheter, mounted with a rapid-response thermistor. The thermistor was connected to a dedicated computer to display on-line cardiac output (CO) and RVEF, allowing calculation of RVEDVI (EDV, as the ratio of SV to RVEF). Moreover, for each patient, we recorded, CO, heart rate, and mean systolic and mean pulmonary arterial pressure. RVEDV, CO, and SV were divided by body surface area to obtain index values. Hemodynamic parameters were recorded during the 4 phases: T0, after induction of anesthesia; T1, during the anhepatic phase; T2, 30' after graft reperfusion; and T3, at the end of surgery. Data are shown as mean values $\pm$ SD for continuous variables and absolute or relative frequencies for categorical variables. Correlations between RVEDVI, PAOP, CVP, and other hemodynamic variables were assessed using linear regression analysis. To compare coupled data, we used a t test (paired two sample for means, two-tail), with $P < .05$ considered statistically significant.

RESULTS

The research included 21 liver transplant recipients (mean age, 50.6 ± 13.4 ; 14 males, 7 females). A piggy-back surgical technique was used on all patients. Hemodynamic data are recorded in Table 1. A significant increase in the heart rate (HR) was observed: 84 ± 12 beats/min ($P < .01$), 85 ± 12 beats/min ($P < .01$), and 86 ± 12 beats/min ($P < .01$) at T1, T2, and T3, respectively. Mean arterial pressure decreased significantly 30' after graft reperfusion: 60 ± 10 mm Hg (P

$< .01$). Compared with T0, CVP decreased significantly at T1 4.5 ± 2.5 mm Hg ($P < .01$) and at T2 7.4 ± 3.6 mm Hg ($P < .01$). Even PAOP decreased significantly at T1 6.3 ± 2.2 mm Hg ($P < .01$) and at T2 8.0 ± 2.8 mm Hg ($P < .01$). Significant variation of RVEDVI value was observed at T1 133.1 ± 33 mL/m² ($P < .05$). Cardiac contractility, as shown by CI, was significantly increased at T1, T2, and T3: $3.94.5 \pm 1.6$ L/min/m² ($P < .05$), 4.5 ± 1.6 L/min/m² ($P < .01$), 4.7 ± 1.3 mm Hg ($P < .01$), respectively. SVI also increased at similar times: 57 ± 13 mL/m² ($P < .05$) at T1, 56 ± 17 mL/m² ($P < .05$) at T2, and 57 ± 18 mL/m² ($P < .05$) at T3. No patient had pulmonary hypertension during the study interval. Compared with T0, 22 ± 6 mm Hg, the mean pulmonary artery pressure (mPAP) decreased significantly during the anhepatic phase, 15 ± 5 mm Hg ($P < .01$). Significant variation of REF value was observed 30' after graft reperfusion, $39 \pm 6\%$ ($P < .01$). Linear regression analysis showed significant correlation between RVEDVI (range, 133 ± 33 – 145 ± 40 mL/m²) and SVI in each phase ($r^2 = 0.49$, $P < .01$; $r^2 = 0.57$, $P < .01$) at T0 and T1, respectively, ($r^2 = 0.51$, $P < .01$; $r^2 = 0.44$, $P < .01$) at T2 and T3, respectively. No significant variations of the linear regression analysis between EDVI, PAOP, CVP, and REF were observed. No relationship was found between PAOP (range, 10 ± 2 – 6 ± 2 mm Hg) and SVI (Table 2).

DISCUSSION

The advent of rapid response thermistors has allowed accurate bedside measurement of right ventricular volumes. Several studies^{6,7} have documented the scarce correlation between PAOP and RVEDVI. The advantage of volumetric measurements in assessing preload status has been noted by multiple authors, who have identified a significant correlation between RVEDVI and SVI^{2,3} and a lack of correlation between PAOP and SVI.⁸ We found a significant correlation between SVI and RVEDVI. In comparison with PAOP, RVEDVI seems a more reliable indicator of cardiac preload in patients undergoing liver transplantation. No correlation was observed between CVP, PAOP, and RVEDVI, indicating that CVP and PAOP are less reliable clinical indicators of RV preload. Wagner et al⁴ recorded that RVEDVI was not a reliable predictor of the

Table 1. Mean $\pm$ SD and t test of Hemodynamic Variables Recorded at Each Phase

	T0	T1	T2	T3
HR (beats/min)	70.33 $\pm$ 16.78	84.66 $\pm$ 12.96 [†]	85.66 $\pm$ 12.51 [†]	86.38 $\pm$ 12.65 [†]
mAP (mm Hg)	69.71 $\pm$ 10.53	64.38 $\pm$ 10.85	60.19 $\pm$ 10.76 [†]	67.23 $\pm$ 13.23
CVP (mm Hg)	10.09 $\pm$ 3.91	4.57 $\pm$ 2.56 [†]	7.42 $\pm$ 3.64 [†]	8.90 $\pm$ 3.41
mPAP (mm Hg)	22.14 $\pm$ 6.54	15.04 $\pm$ 5.41 [†]	19.61 $\pm$ 5.94	20.85 $\pm$ 5.75
PAOP (mm Hg)	10.19 $\pm$ 2.92	6.33 $\pm$ 2.26 [†]	8.09 $\pm$ 2.80 [†]	9.38 $\pm$ 3.12
REF (%)	36.23 $\pm$ 4.03	36.42 $\pm$ 5.96	39.85 $\pm$ 6.92 [†]	37.57 $\pm$ 7.64
EDVI (mL/m ²)	144.76 $\pm$ 24.37	133.57 $\pm$ 33.58*	134.57 $\pm$ 36.61	145.38 $\pm$ 40.51
CI (L/min/m ²)	3.48 $\pm$ 1.23	3.94 $\pm$ 1.03*	4.59 $\pm$ 1.61 [†]	4.71 $\pm$ 1.38 [†]
SVI (mL/m ²)	49.14 $\pm$ 8.97	57.33 $\pm$ 13.43*	56.04 $\pm$ 17.32*	57.71 $\pm$ 18.88*

Note: T0, after induction of anesthesia; T1, during anhepatic phase; T2, 30' after graft reperfusion; and T3, end of surgery.

* $P < .05$.

[†] $P < .01$.

Table 2. Linear Regression Analysis of Hemodynamic Data Recorded at Each Phase of the Surgical Intervention

	EDVI			
	T0	T1	T2	T3
SVI (mL/m ²)	$r^2 = 0.49$ $P = .0004$	$r^2 = 0.57$ $P = 6.31E-05$	$r^2 = 0.51$ $P = .0002$	$r^2 = 0.44$ $P = .0009$
PAOP (mm Hg)	$r = 0.06$ $P = .26$ (NS)	$r^2 = 0.09$ $P = .18$ (NS)	$r^2 = 0.12$ $P = .20$ (NS)	$r^2 = 0.10$ $P = .15$ (NS)

Abbreviation: NS, not statistically significant.

Note: T0, after induction of anesthesia; T1, during anephatic phase; T2, after graft reperfusion; and T3, end of surgery.

response to fluid. As a predictor of fluid responsiveness, PAOP was superior to RVEDVI. The overall EF in the patients of this study was $36 \pm 4\%$, compared with an EF of $31 \pm 10\%$ in the study performed by Wagner et al. It is possible that EDVI may better predict the response to volume in patients with higher RVEF. We found significant alterations of systemic hemodynamic parameters contemporaneously with minor changes in EF (range, 36%–39%). This confirms that EF reflects changes in preload and can be considered to be a measure of the contractility determining ventricular performance.^{9,10} The major findings of this study are that RV function appeared to be well preserved throughout the procedure, as indicated by a relatively constant increase in EF, suggesting that OLT is not associated with significant RV dysfunction. In conclusion, the relationship between EDVI and CI is conserved even at a low ejection fraction (<40%) among patients undergoing OLT. EDVI is a reliable predictor of the hemodynamic response to volume loading. RV function remains unaltered during uncomplicated OLT, showing that liver transplantation is not associated with significant RV dysfunction.

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