

The comparison between stroke volume variation and filling pressure as an estimate of right ventricular preload in patients undergoing renal transplantation

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Abstract

Purpose The purpose of this prospective, observational study was to respiratory variation of stroke volume (stroke volume variation, SVV) against central venous pressure (CVP) and pulmonary artery diastolic pressure (PADP) as an estimate of right and left ventricular preload.

Methods With IRB approval and informed consent, 31 patients undergoing living related renal transplantation were analyzed. Under general anesthesia with positive pressure ventilation, stroke volume index and SVV were continuously monitored with FloTrac/Vigileo monitor. Right ventricular end-diastolic volume index (RVEDVI) as well as CVP and PADP were continuously monitored with volumetric pulmonary artery catheter. Data of every 30 min interval were used for analysis. The relationship between RVEDVI and CVP, PADP, SVV was analyzed with non-linear regression and the goodness-of-fit was assessed with coefficient of determination (R^2) of each regression curve. The ability of CVP, PADP and SVV to correctly differentiate RVEDVI <100 , <120 or >138 ml/m², which were used to guide fluid administration, was also assessed with ROC analysis.

Results Three hundred forty-eight data sets were obtained and analyzed. The goodness of fit between RVEDVI and SVV ($R^2 = 0.48$) was better than that between RVEDVI and CVP or PADP ($R^2 = 0.19$ and 0.33 , respectively). The

area under the ROC curve of SVV was significantly high compared to CVP or PADP.

Conclusions This study confirmed the theoretical framework of right ventricular preload and ventricular filling pressure and respiratory variation of stroke volume. The result also suggests that SVV can correctly predict preload status compared to pressure-based indices.

Keywords Preload responsiveness · Dynamic parameter · Static parameter · Right ventricular end-diastolic volume · Renal transplantation

Introduction

Conventionally, pressure-based static parameter such as central venous pressure (CVP) and pulmonary artery wedge pressure (PAWP) has been used to guide fluid therapy. However, the validity of CVP and PAWP as an index of fluid responsiveness has been questioned [1, 2]. Alternatively, several studies suggest that dynamic parameters including stroke volume variation (SVV) has been successfully predict the fluid responsiveness in anesthetized patients and critically ill patients [3, 4]. Furthermore, the recent study demonstrated that dynamic parameter-guided intraoperative fluid management reduced intraoperative lactate concentration [5]. The rationale of this study implies that there is a certain relationship between SVV and ventricular preload. However, the relationship between SVV and ventricular preload has not been clearly established. The purpose of this prospective, observational study was to compare SVV against CVP and pulmonary artery diastolic pressure (PADP) as estimates of right and left ventricular preload. To achieve this purpose in wide range of right ventricular preload, patients

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undergoing renal transplantation were studied since these patients were often hypovolemic before anesthesia due to preoperative hemodialysis and were intentionally fluid-loaded before graft reperfusion.

Subjects and methods

With institutional review board approval, patients undergoing elective renal transplantation from living-related donor were screened for eligibility in this study. Patients who were not eligible for pulmonary artery catheter insertion, with severe tricuspid regurgitation or any types of arrhythmia during preoperative evaluation were excluded from the study. Consequently, 33 patients were included in this study with written informed consent. All the subjects received chronic hemodialysis and underwent it on the day before surgery. The anesthetic management was at the discretion of one of the co-authors of this study (TT) in all the cases. Briefly, anesthesia was induced with intravenous fentanyl, propofol and rocuronium and was maintained with sevoflurane-oxygen-air mixture supplemented with either fentanyl or remifentanyl. If there were no contraindications, continuous epidural block was used both intraoperatively and postoperatively. Patients were mechanically ventilated with tidal volume of 8–10 ml/kg of actual body weight, PEEP of 5 cmH₂O and respiratory rate was adjusted to maintain normocapnia. After anesthetic induction, radial artery was cannulated with 22G plastic needle (Introcan safety, BBrown, Melsungen, Germany) and stroke volume index (SVI) and SVV were continuously monitored with FloTrac/Vigileo monitor (Ver. 3.02, Edwards Lifesciences, Irvine, CA, USA) [6, 7]. Additionally, volumetric pulmonary artery catheter (PAC) was inserted via right internal jugular vein (774HF75, Edwards Lifesciences, Irvine, CA, USA) and central venous pressure (CVP), pulmonary artery pressure and right ventricular end-diastolic volume index (RVEDVI) were continuously monitored. The principle of RVEDVI measurement with modified PAC is described elsewhere [8]. Briefly, recording of the blood temperature by fast-response thermister and R–R interval from electrocardiography, diastolic plateau of the thermodilution curve is identified. Total end-diastolic heat mass in the right ventricle is the sum of the heat mass at the end of the previous cycle (right ventricular end-systolic volume) and the colder blood during diastolic filling. The zero reference point of the pressure measurement was set at the mid-axillary level. To optimize renal blood flow after renal reperfusion, either saline, half saline, fresh frozen plasma, 5 % albumin or packed red cell are deliberately administered with the target of CVP >10 mmHg and PADP >15 mmHg irrespective of other hemodynamic data before the completion of

renal arterial anastomosis [9]. After the successful reperfusion of transplanted kidney, either fluid or fluid plus diuretics was administered to maintain adequate renal perfusion and urine output. At the completion of the transplantation, PAC was replaced with standard central venous catheter and the patients were transferred to the hospital ward after the regain of consciousness and the removal of endotracheal tube. Other clinical decisions were at the discretion of the attending anesthesiologist.

Hemodynamic data during the anesthesia were retrieved from the electronic archive and RVEDVI was used as an index of volume-based right ventricular preload. CVP and PADP were used as pressure-based static indicator of right ventricular and left ventricular preload, respectively. PADP was used as a surrogate pulmonary artery wedge pressure since it has been used as the target of fluid loading in our practice and is advantageous due to its continuous and operator-independent nature. Data were collected at 1 min interval in the archive but data sets obtained from the start of the surgery and every 30 min thereafter throughout the surgical procedure in each subject were used for analysis.

The number of the subjects was determined according to the previous report that stated sample size of 30 subjects had adequate statistical power for similar analysis [10]. Demographic and surgical data were expressed either as mean \pm SD or median (range) depending on the distribution of the data. The relationship between CVP, PADP, and SVV against RVEDVI were examined with non-linear curve fitting. Coefficient of determination (R^2) was used to quantify goodness-of-fit [11]. In order to speculate the ability of CVP, PADP and SVV for preload responsiveness or indication of fluid challenge, the ROC analysis of these parameters against several RVEDVI values was performed. Particularly, RVEDVI >138 ml/m² as the threshold of 100 % negative fluid responsiveness [12] and RVEDVI >100 ml/m² [13] and >120 ml/m² [11, 14] as a resuscitation goal during fluid resuscitation. The best cut-off value of CVP, PADP and SVV against these RVEDVI was determined by Youden index [15]. Prism (Ver. 5.0, Graphpad Software, San Diego, CA, USA) was used for the aforementioned statistical analysis.

Results

Two patients were excluded from the analysis due to intraoperative arrhythmia and the resultant 31 subjects were included in the analysis. The demographic and operative data are summarized in the Table 1. Number of available data sets from each subject was 11 ± 2 and total 348 data sets were used for analysis. The median (range) of RVEDVI, SVI, CVP, PADP and SVV was 142 (73–238) ml/m², 50 (22–135) ml/m², 9 (0–20) mmHg, 13 (3–28)

Table 1 Demographic and operative data

Age (years old)	42 ± 13
Gender (male/female)	20/11
Height (cm)	165 ± 9
Weight (kg)	60 ± 11
Pathology of end-stage renal disease (glomerulonephritis/diabetic nephropathy/others)	16/11/4
Duration of dialysis (years)	5.5 (3–15)
Presence of right heart dysfunction ^a	5
Presence of left heart dysfunction ^b	14
Duration of anesthesia (min)	488 ± 112
Duration of surgery (min)	334 ± 79
Intraoperative crystalloid (ml)	5270 (2450–14560)
Intraoperative colloid (ml)	860 (720–2400)
Intraoperative red blood cell (ml)	890 (0–3920)
Blood loss (g)	800 (50–5130)
Urine output (ml)	920 (30–2340)

Data are expressed as either mean ± SD, median (range) or number of subjects

^a Right heart dysfunction include right atrial dilatation, right ventricular hypertrophy, tricuspid regurgitation on preoperative transthoracic echocardiographic evaluation

^b Left heart dysfunction include left atrial dilatation, left ventricular hypertrophy, mitral regurgitation and regional wall motion abnormality on preoperative transthoracic echocardiographic evaluation

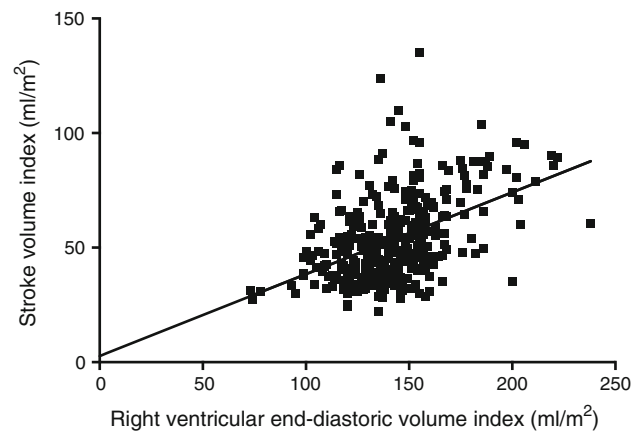
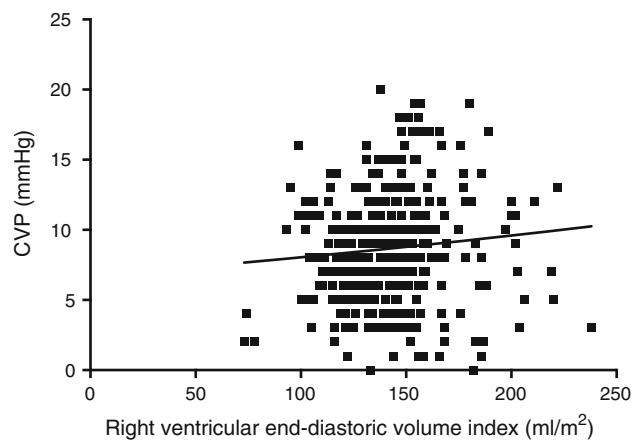
Table 2 Summary of hemodynamics

	Before fluid loading (n = 38)	During fluid loading (n = 181)	After fluid loading (n = 114)
HR (/min)	68 ± 9	71 ± 13	83 ± 13
MAP (mmHg)	78 ± 19	77 ± 18	89 ± 17
CVP (mmHg)	8 ± 3	8 ± 4	10 ± 4
PADP (mmHg)	11 ± 3	12 ± 4	15 ± 5
SVI (ml/m ²)	49 ± 16	51 ± 17	64 ± 15
SVV (%)	11 ± 4	10 ± 4	8 ± 4
RVEF (%)	40 ± 9	39 ± 10	43 ± 10
RVEDVI (ml/m ²)	131 ± 14	139 ± 25	150 ± 23
SvO ₂ (%)	85 ± 6	86 ± 6	89 ± 5

Data are expressed as mean ± SD

PADP pulmonary artery diastolic pressure, *SVI* stroke volume index, *RVEF* right ventricular ejection fraction, *RVEDVI* right ventricular end-diastolic volume index, *SvO₂* mixed venous oxygen saturation

mmHg and 9.0 (1.5–25.3) %, respectively. These data sets are summarized with the following three categories; before fluid loading, during fluid loading and after fluid loading (Table 2). The relationship between RVEDVI and SVI was most adequately represented by linear fitting. ($SVI = 0.36 \times RVEDVI + 2.7$, Fig. 1). The relationships between RVEDVI and CVP, PADP, SVV are most

**Fig. 1** Relationship between RVEDVI and SVI. The relationship between RVEDVI and SVI is best represented by linear regression line**Fig. 2** Relationship between RVEDVI and CVP. The relationship between RVEDVI and CVP is best represented by non-linear regression line. The coefficient of determination (R^2) is 0.19

adequately represented by curvilinear fitting. The regression equations of each relationship was as follows: $CVP = 3.8Xe^{(0.005 \times RVEDVI)}$, $PADP = 5.2Xe^{(0.006 \times RVEDVI)}$, $SVV = 50.8Xe^{(-0.009 \times RVEDVI)} - 5.0$, respectively (Figs. 2, 3, 4). The R^2 value that represented goodness of fit between CVP, PADP, SVV and RVEDVI was 0.19, 0.33 and 0.48, respectively, indicating that the relationship between RVEDVI and SVV was more closely represented by the fitting curve compared to CVP or PADP. The results of ROC analysis to detect various RVEDVI target for CVP, PADP and SVV are summarized in the Table 3 [12–14].

Discussion

The major findings of this study were as follows: (1) There was curvilinear relationship between filling pressures and

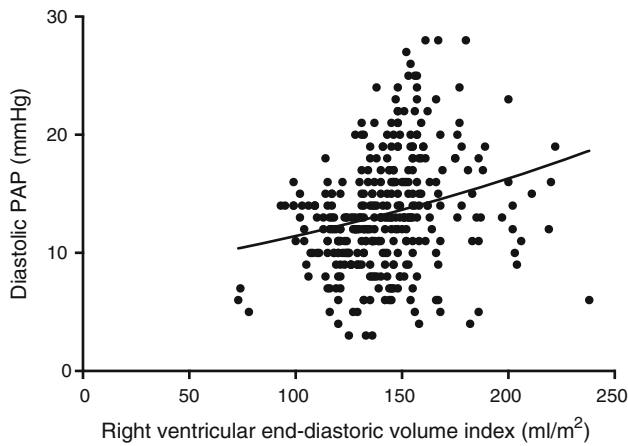


Fig. 3 Relationship between RVEDVI and pulmonary artery diastolic pressure. The relationship between RVEDVI and pulmonary artery diastolic pressure is best represented by non-linear regression line. The coefficient of determination (R^2) is 0.33

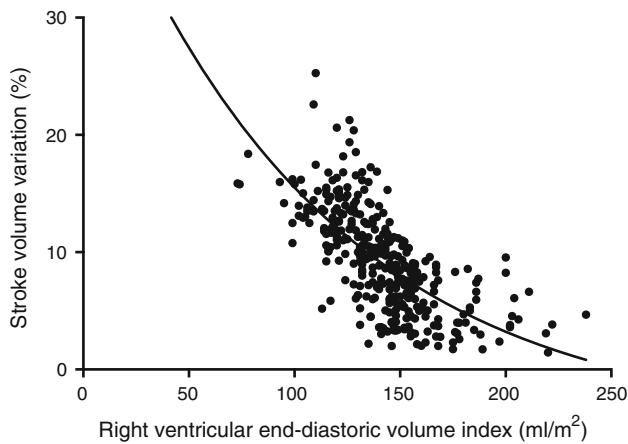


Fig. 4 Relationship between RVEDVI and stroke volume variation. The relationship between RVEDVI and pulmonary artery diastolic pressure is best represented by non-linear regression line. The coefficient of determination (R^2) is 0.48

RVEDVI. (2) There was curvilinear relationship between SVV and RVEDVI. (3) SVV was able to distinguish more accurately various RVEDVI threshold than ventricular filling pressure.

Terms such as static and dynamic parameters and preload responsiveness have been simultaneously used but indiscriminate use of these two different concepts is not warranted [16]. However, the relationship between these indices and preload itself has not been clearly demonstrated. Traditionally ventricular pressure and its surrogates have been used as the indicator of preload and preload responsiveness, but this concept has been repeatedly questioned [2, 10, 17, 18]. On the contrary, respiratory induced variation of stroke volume or arterial pressure has been successfully indicated the preload responsiveness

Table 3 ROC analysis of CVP, PADP and SVV to differentiate several threshold of RVEDVI

	RVEDVI <100 ml/m ² (indication of fluid challenge) ^a	RVEDVI <120 ml/m ² (indication of fluid challenge) ^a	RVEDVI >138 ml/m ² (negative fluid responsiveness) ^a
CVP			
AUC	0.58	0.58	0.57
Best cut-off value (mmHg)	<7	<8	>12
Sensitivity	0.33	0.53	0.29
Specificity	0.89	0.63	0.86
PADP			
AUC	0.58	0.64	0.67
Best cut-off value (mmHg)	<8	<13	>17
Sensitivity	0.33	0.58	0.34
Specificity	0.89	0.60	0.92
SVV			
AUC	0.88	0.86	0.88
Best cut-off value (%)	>16	>13	<9
Sensitivity	0.90	0.63	0.81
Specificity	0.75	0.84	0.84

PADP pulmonary artery diastolic pressure, *RVEDVI* right ventricular end-diastolic pressure, *SVV* stroke volume variation, *AUC* area under the curve

^a The rationale of these thresholds can be found in Refs. [12–14]

[19–22]. Spahn and his colleague graphically demonstrated the relationship between preload and these parameters and such theoretical framework is believed to be valid [23]. However, there is paucity of data supporting this concept.

Therefore, we tried to clarify the relationship between ventricular dimension and parameters of preload sensitivity, i.e., ventricular filling pressure and respiratory variation of left ventricular stroke volume in patients undergoing renal transplantation. These subjects undergo significant change of fluid status during surgery and may be appropriate to investigate the relationship in wide range.

In this study, we used RVEDVI measured with volumetric PAC as preload. This method enables automatic and continuous assessment of right ventricular preload and has been used in several clinical studies. Among them, RVEDVI below 90 ml/m² and over 138 ml/m² have been successfully diagnosed the presence of fluid responsiveness

with 100 % sensitivity and specificity in critically ill patients [12].

We found typical nonlinear relationship between CVP, diastolic PAP and RVEDVI in patients undergoing renal transplantation. The regression curve between RVEDVI and filling pressure theoretically represents right ventricular compliance and the interpretation of this relationship in perioperative hemodynamic management is summarized elsewhere [23]. The compliance is supposedly represented by curvilinear relationship, in which filling pressure exponentially increase when RV volume exceeds certain limit. Kincaid et al. [11] actually confirmed such relationship between CVP and RVEDVI and found individual coefficient of determination (R^2) in the range from 0.84 to 0.99 in trauma patients. However, several other studies differently assessed the relationship between filling pressure and RV volume. For example, Su et al. [24] demonstrated linear relationship between RV volume and CVP as well as PAWP. On the contrary, several studies reported no linear correlation between filling pressure and RVEDVI [17, 25]. Although these investigators did not apply nonlinear curve fit for the analysis, no evident relationship was found by manual inspection. It is not readily known the underlying reasons of such discrepancy. Nevertheless, our results correspond well with the current paradigm that rapid rise of filling pressure indicates possible hypervolemia. However, it is also clear that defining the threshold of filling pressure is difficult since there is no evident inflection point on the fitted curve.

We also found curvilinear relationship between SVV and RVEDVI in this study. On the contrary, Su et al. [24] demonstrated the linear relationship between preload and both pressure based static indicator and dynamic indicator of preload responsiveness in patients undergoing liver transplantation. Since the respiratory variations of stroke volume, pulse pressure or systolic pressure supposedly represent the slope of the Frank–Starling curve at certain cardiovascular status [26], the curve describing such relationship should be the inverted image of Frank–Starling curve. From this standpoint, we believe that the nonlinear fitting more appropriately describes the relationship. Additionally, the steep slope of the regression curve at the hypovolemic status suggests that SVV has high sensitivity to indicate preload responsiveness. On the contrary, the relationship between CVP, PADP and RVEDVI is flat at such status and suggests low sensitivity of the presence of preload responsiveness. We believe our findings correspond to the current paradigm that dynamic indicators such as SVV are advantageous to assess preload responsiveness than ventricular filling pressure. Furthermore, SVV more correctly differentiates several threshold of RVEDVI than filling pressure. Almost all the previous studies confirmed that SVV or its surrogates; pulse pressure variation and

systolic pressure variation successfully distinguish the presence of fluid responsibility. It is reasonable to assume that hypovolemic state such as $RVEDVI < 100 \text{ ml/m}^2$ can be correctly diagnosed by SVV than filling pressure. However, hypervolemic condition may be more correctly diagnosed by filling pressure due to steep slope of ventricular compliance curve at such condition [27]. Before this study, we were not sure whether hypervolemic state such as $RVEDVI > 138 \text{ ml/m}^2$ could be adequately predicted by SVV. However, our results suggest SVV is capable to indicate the increased RVEDVI more correctly than filling pressure. This finding also supports the claim that filling pressure is not suitable as a goal of fluid resuscitation [28].

This study also has several limitations. First, the current study design precludes providing the definitive cut-off value of these parameters about preload responsiveness. Currently available data generally support the application of goal-directed fluid management and assessment of the presence or absence of preload responsiveness plays a key role about the decision making whether the fluid should be given or not [22]. We acknowledge that it would be more clinically relevant if this study could provide additional information about the presence of preload responsiveness by standardized empiric fluid challenge. Nevertheless, we tried to compare the appropriateness of SVV and filling pressure as an indicator of preload responsiveness by the ROC analysis. In this analysis, we used certain values of RVEDVI obtained from critically ill patients since no intraoperative data are available for such purpose. In this regard, the cut-off value obtained by this study should be interpreted with caution. Second, the accuracy of the PAC-derived RVEDV is repeatedly questioned [8, 29–31]. Especially, the absolute value of RVEDVI may not be ideally accurate. Thus, our results summarized in the Table 3 should be interpreted with caution since these results are dependent on the accuracy of RVEDVI. However, we believe that the assessment of right ventricular preload is imperative to our study purpose and the relationship shown in Figs. 2, 3, 4 remains valid even if the absolute value of RVEDVI is not interchangeable to the value obtained by the other method. Third, the right ventricular function was not systematically evaluated in this study. Several previous studies demonstrated impaired right ventricular function in patients undergoing chronic hemodialysis [32, 33]. We tried to minimize such influences by excluding patients with evident right and left ventricular dysfunction but our results may be affected by the cardiac dysfunction in these specific patient group. However, selecting these subjects may provide unique opportunity since these subjects underwent significantly large change of fluid status during the study period. At the anesthetic induction,

subjects were usually hypovolemic due to the preoperative hemodialysis. However, subjects were deliberately fluid loaded before renal artery anastomosis to ensure adequate renal perfusion after reperfusion [9]. In this study, the range of RVEDVI was larger than previous studies and makes the interpretation of the relationship more robust. Despite these limitations, we believe the relationship shown in this study remains valid. Fourth, whether SVV obtained from arterial pulse contour method is superior to other dynamic parameters is not demonstrated. Respiratory variations of stroke volume and arterial pressure are consisted with the following two components; delta up caused by the increased left ventricular preload by enhanced pulmonary venous return and delta down caused by the reduced right ventricular preload by increased intrathoracic pressure. The relationship between RVEDVI and dynamic parameters may be more accurately assessed if delta down of stroke volume or arterial pressure were used. Unfortunately, the computation of delta down requires transient pause of mechanical ventilation and manual calculation and therefore, we used SVV that include both delta up and delta down of stroke volume.

In conclusion, this study demonstrated that there was curvilinear relationship between RVEDVI and SVV. The regression curve fits better with SVV compared to CVP or PADP over wide range of right ventricular preload. These results imply that SVV better represents right ventricular preload than filling pressure.

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Conflict of interest Dr. Kotake has received consultant fee from Edwards Lifesciences and unrestricted research fund from MSD, Nihon Kodan Corp. Dr. Kotake has also received speakers fee from MSD, Fresenius-Kabi, Otsuka Pharmaceuticals and Covidien.

References

1. Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest*. 2002;121:2000–8.
2. Osman D, Ridel C, Ray P, Monnet X, Anguel N, Richard C, Teboul JL. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. *Crit Care Med*. 2007;35:64–8.
3. Biais M, Nouette-Gaulain K, Cottenceau V, Revel P, Sztark F. Uncalibrated pulse contour-derived stroke volume variation predicts fluid responsiveness in mechanically ventilated patients undergoing liver transplantation. *Br J Anaesth*. 2008;101:761–8.
4. Cannesson M, Musard H, Desebbe O, Boucau C, Simon R, Henaine R, Lehot JJ. The ability of stroke volume variations obtained with Vigileo/FloTrac system to monitor fluid responsiveness in mechanically ventilated patients. *Anesth Analg*. 2009;108:513–7.
5. Forget P, Lois F, de Kock M. Goal-directed fluid management based on the pulse oximeter-derived pleth variability index reduces lactate levels and improves fluid management. *Anesth Analg*. 2010;111:910–4.
6. Biancofiore G, Critchley LA, Lee A, Yang XX, Bindi LM, Esposito M, Bisa M, Meacci L, Mozzo R, Filippini F. Evaluation of a new software version of the FloTrac/Vigileo (version 3.02) and a comparison with previous data in cirrhotic patients undergoing liver transplant surgery. *Anesth Analg*. 2011;113:515–22.
7. Tsai YF, Su BC, Lin CC, Liu FC, Lee WC, Yu HP. Cardiac output derived from arterial pressure waveform analysis: validation of the third-generation software in patients undergoing orthotopic liver transplantation. *Transpl Proc*. 2012;44:433–7.
8. Hein M, Roehl AB, Baumert JH, Rossaint R, Steendijk P. Continuous right ventricular volumetry by fast-response thermodilution during right ventricular ischemia: head-to-head comparison with conductance catheter measurements. *Crit Care Med*. 2009;37:2962–7.
9. Sprung J, Kapural L, Bourke DL, O'Hara JF Jr. Anesthesia for kidney transplant surgery. *Anesthesiol Clin North Am*. 2000;18:919–51.
10. He Z, Qiao H, Zhou W, Wang Y, Xu Z, Che X, Zhang J, Liang W. Assessment of cardiac preload status by pulse pressure variation in patients after anesthesia induction: comparison with central venous pressure and initial distribution volume of glucose. *J Anesth*. 2011;25:812–7.
11. Kincaid EH, Meredith JW, Chang MC. Determining optimal cardiac preload during resuscitation using measurements of ventricular compliance. *J Trauma*. 2001;50:665–9.
12. Diebel LN, Wilson RF, Tagett MG, Kline RA. End-diastolic volume. A better indicator of preload in the critically ill. *Arch Surg*. 1992;127:817–21.
13. Chang MC, Meredith JW. Cardiac preload, splanchnic perfusion, and their relationship during resuscitation in trauma patients. *J Trauma*. 1997;42:577–82.
14. Miller PR, Meredith JW, Chang MC. Randomized, prospective comparison of increased preload versus inotropes in the resuscitation of trauma patients: effects on cardiopulmonary function and visceral perfusion. *J Trauma*. 1998;44:107–13.
15. Akobeng AK. Understanding diagnostic tests 3: receiver operating characteristic curves. *Acta Paediatr*. 2007;96:644–7.
16. Reuter DA, Goetz AE. Differentiating “volumetric preload monitoring” and assessing “fluid responsiveness”. *Anesth Analg*. 2006;102:651–2.
17. Kumar A, Anel R, Bunnell E, Habet K, Zanotti S, Marshall S, Neumann A, Ali A, Cheang M, Kavinsky C, Parrillo JE. 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;32:691–9.
18. Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. *Chest*. 2008;134:172–8.
19. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. *Crit Care Med*. 2009;37:2642–7.
20. Zhang Z, Lu B, Sheng X, Jin N. Accuracy of stroke volume variation in predicting fluid responsiveness: a systematic review and meta-analysis. *J Anesth*. 2011;25:904–16.
21. Michard F. Stroke volume variation: from applied physiology to improved outcomes. *Crit Care Med*. 2011;39:402–3.
22. Biais M, Ouattara A, Janvier G, Sztark F. Case scenario: respiratory variations in arterial pressure for guiding fluid management in mechanically ventilated patients. *Anesthesiology*. 2012;116:1354–61.
23. Spahn DR, Chassot PG. CON: fluid restriction for cardiac patients during major noncardiac surgery should be replaced by

- goal-directed intravascular fluid administration. *Anesth Analg.* 2006;102:344–6.
24. Su BC, Tsai YF, Cheng CW, Yu HP, Yang MW, Lee WC, Lin CC. Stroke volume variation derived by arterial pulse contour analysis is a good indicator for preload estimation during liver transplantation. *Transpl Proc.* 2012;44:429–32.
 25. Spohr F, Hettrich P, Bauer H, Haas U, Martin E, Bottiger BW. Comparison of two methods for enhanced continuous circulatory monitoring in patients with septic shock. *Intensive Care Med.* 2007;33:1805–10.
 26. Michard F. Changes in arterial pressure during mechanical ventilation. *Anesthesiology.* 2005;103:419–28.
 27. Taguchi H, Ichinose K, Tanimoto H, Sugita M, Tashiro M, Yamamoto T. Stroke volume variation obtained with Vigileo/Flo-Trac system during bleeding and fluid overload in dogs. *J Anesth.* 2011;25:563–8.
 28. Ytrebo LM. Stop filling patients against central venous pressure, please! *Crit Care Med.* 2011;39:396–7.
 29. Wagner JG, Leatherman JW. Right ventricular end-diastolic volume as a predictor of the hemodynamic response to a fluid challenge. *Chest.* 1998;113:1048–54.
 30. Zink W, Noll J, Rauch H, Bauer H, Desimone R, Martin E, Bottiger BW. Continuous assessment of right ventricular ejection fraction: new pulmonary artery catheter versus transoesophageal echocardiography. *Anaesthesia.* 2004;59:1126–32.
 31. De Simone R, Wolf I, Mottl-Link S, Bottiger BW, Rauch H, Meinzer HP, Hagl S. Intraoperative assessment of right ventricular volume and function. *Eur J Cardiothorac Surg.* 2005;27:988–93.
 32. Arinc H, Gunduz H, Tamer A, Ozhan H, Akdemir R, Saglam H, Oguzhan A, Uyan C. Use of tissue Doppler to assess right ventricle function in hemodialysis patients. *Am J Nephrol.* 2005;25:256–61.
 33. Paneni F, Gregori M, Ciavarella GM, Sciarretta S, De Biase L, Marino L, Tocci G, Principe F, Domenici A, Luciani R, Punzo G, Mene P, Volpe M. Right ventricular dysfunction in patients with end-stage renal disease. *Am J Nephrol.* 2010;32:432–8.