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Uncalibrated pulse contour-derived stroke volume variation predicts fluid responsiveness in mechanically ventilated patients undergoing liver transplantation

M. Biais, K. Nouette-Gaulain, V. Cottenceau, P. Revel and F. Sztark*

Service d'Anesthésie Réanimation 1, Hôpital Pellegrin, CHU Bordeaux, Place Amélie Raba-Léon, 33076 Bordeaux Cedex, France

**Corresponding author. E-mail: francois.sztark@chu-bordeaux.fr*

Background. Stroke volume variation (SVV) is able to predict adequately the individual response to fluid loading. Our objective was to assess whether the SVV measured by a new algorithm (Vigileo^{TM}; Flotrac^{TM}) can predict fluid responsiveness.

Methods. Forty mechanically ventilated patients undergoing liver transplantation, who needed volume expansion (VE), were included. VE was done with albumin (4%) 20 ml×BMI over 20 min. SVV, pulse pressure variation (PPV), central venous pressure (CVP), and pulmonary artery occlusion pressure (PAOP) were measured immediately before and after VE. Cardiac output (CO) measured by transthoracic echocardiography (CO-TTE) was used to define responder patients if CO increased by 15% or more after VE, or non-responder otherwise. CO obtained with the pulmonary artery catheter (CO-PAC) and with Vigileo (CO-Vigileo) were also recorded.

Results. Five patients were excluded. Seventeen patients were responders (Rs) and 18 were non-responders (NRs). Before VE (i) SVV and PPV were higher in Rs and (ii) CVP and PAOP were lower in Rs. Baseline SVV and PPV correlated with change in CO induced by VE (respectively, r^2=0.72, $P<0.0001$; r^2=0.84, $P<0.0001$). An SVV threshold of >10% discriminated Rs with a sensitivity of 94% and a specificity of 94%. After VE, the decrease in SVV was significantly correlated with the increase in CO (r^2=0.51; $P<0.0001$). There was no difference between the area under the ROC curves of SVV and PPV. After VE, the change in CO-Vigileo was closely correlated with change in CO-TTE (r^2=0.74, $P<0.0001$) and with change in CO-PAC (r^2=0.77, $P<0.0001$).

Conclusions. The SVV obtained by the Vigileo system may be used as a predictor of fluid responsiveness in patients with circulatory failure after liver transplantation. CO-Vigileo is able to track the change in CO induced by VE.

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Optimal monitoring of cardiac preload in the critically ill is paramount for precise haemodynamic management, particularly in the postoperative course of liver transplantation. Indeed, this surgery is associated with massive bleeding during dissection and with a decrease in systemic vascular resistance after graft reperfusion.[1 2] In these patients, an adequate preload is of utmost importance for optimizing cardiac performance and organ perfusion. None of the routinely used static variables of cardiac preload, such as filling pressures (central venous pressure, CVP, and pulmonary artery occlusion pressure, PAOP), reliably predict fluid responsiveness.[3–5] In contrast to static indices of preload, dynamic indices such as stroke volume variation (SVV) are able to predict adequately the individual response to fluid loading.[6–10]

The recently introduced Vigileo monitor (Vigileo^{TM}; Flotrac^{TM}; Edwards Lifesciences, Irvine, CA, USA), which allows continuous cardiac output (CO) monitoring,

is based on the analysis of the systemic arterial pressure wave and does not require pulmonary artery catheterization or calibration with another method.[11] In addition, the monitor continuously displays the SVV, which is the percentage variation of stroke volume (SV) over a floating period of 20 s.

Several studies have demonstrated the usefulness of SVV calculated with the PiCCO^{TM} system (Pulsion SG, Munich, Germany), to predict fluid responsiveness in patients undergoing cardiac surgery, neurosurgical procedures, and in the intensive care unit.[6 12–14] This device needs a femoral artery catheter and a frequent recalibration.[15] The potential advantages of the Vigileo system are that it needs no external calibration and only a radial artery catheter.

The aim of this study was to assess whether SVV obtained with this new technology and algorithm (Vigileo; Flotrac; Edwards Lifesciences) can predict fluid responsiveness in patients undergoing liver transplantation and to compare its predictive value to the commonly measured haemodynamic variables. Furthermore, we compared CO obtained with Vigileo device (CO-Vigileo) and CO obtained (i) with transthoracic echocardiography (CO-TTE) and (ii) with pulmonary artery catheter (CO-PAC).

Methods

Patients

We studied 40 consecutive patients in the postoperative period of liver transplantation, for whom the decision to give fluid was taken by the physician. This decision was based on the presence of clinical signs of acute circulatory failure (low blood pressure or urine output, tachycardia, mottling), biological signs of organ dysfunction, need of vasopressive drugs or all. This prospective observational study was approved by our local ethics committee and patients consented to the study.

Exclusion criteria were: hypoxaemia: $Pa_{O_2}/FI_{O_2} < 100$ mm Hg blood volume overload defined by PAOP$\geq$18 mm Hg, hydrostatic pulmonary oedema on chest radiography, patients younger than 18 yr, arrhythmias, BMI $>$40 or $<$15 kg m^{-2}, significant aortic or mitral valvulopathy, intracardiac shunt, spontaneous breathing activity, and unsatisfactory cardiac echogenicity.

Mechanical ventilation

All patients were studied immediately after admission to the intensive care unit. At this time, all patients were sedated with propofol and sufentanil to ensure that there was no evidence of spontaneous breathing effort (identified by clinical examination and visual examination of respiratory curves). Mechanical ventilation was performed in a volume-controlled mode with a tidal volume of 8–10 ml kg^{-1}, a PEEP of 3 cm H_2O, and an inspiratory/expiratory ratio of 0.5. The inspired oxygen concentration was adjusted to maintain an arterial oxygen partial pressure $>$90 mm Hg. Respiratory rate was adjusted to maintain an arterial carbon dioxide pressure between 35 and 40 mm Hg.

The total PEEP ($PEEP_{tot}$) and the plateau pressure (P_{plat}) were measured using an end-expiratory and end-inspiratory occlusion manoeuvre of 5 s. Tidal volume (V_t) was measured by means of the ventilator transducer. The static compliance of the respiratory system ($C_{st,rs}$) was calculated as follows: $C_{st,rs}=V_t/(P_{plat}-PEEP)$.

Haemodynamic monitoring

Pulmonary artery catheter

Before surgery, a PAC (CCOmbo, 744HF75, 7.5Fr, Edwards Lifesciences) was inserted through the left subclavian vein through an introducer (M3L9FHSI, 9Fr, Edwards Lifesciences), and positioned under guidance of the pressure curve measured at the proximal and distal port of the catheter and chest X-ray. The correct position of the PAC in West's zone 3 was checked using a method previously described.[16] Semi-continuous CO was calculated using a modified Stewart–Hamilton equation (CO-PAC).[17 18]

Patients were studied in a supine position, all transducers were positioned at the level of the fourth intercostal space in the middle axillary line, and zero was measured at atmospheric pressure.

Vigileo monitor

A 3Fr, 8-cm-long arterial catheter (115.09, Vygon, Ecouen, France) was inserted in the left radial artery. A dedicated transducer (FloTrac, Edwards Lifesciences) was connected to the radial arterial line on one side and to the Vigileo System (Edwards Lifesciences) on the other side. The system enables the continuous monitoring of arterial pressure, CO, SV, and SVV by pulse contour analysis. This system needs no calibration and provides continuous CO measurements from the arterial pressure wave. The Vigileo (Software version 1.07) analyses the pressure waveform 100 times per second over 20 s, captures 2000 data points for analysis, and performs its calculations on the most recent 20 s data. The device calculates SV as $k\times$pulsatility, where pulsatility is the standard deviation of arterial pressure over a 20 s interval, and k is a factor quantifying arterial compliance and vascular resistance. The k value is derived from a multivariate regression model, including (i) Langewouter's aortic compliance,[19] (ii) mean arterial pressure, (iii) variance, (iv) skewness, and (v) kurtosis of the pressure curve. The rate of adjustment of k is 1 min (Software 1.07).

The CO-Vigileo was calculated as follows: CO=heart rate$\times$SV. CO obtained with this device was recorded, but

not used in this study, to discriminate responder (R) and non-responder (NR) patients after volume expansion (VE).

Stroke volume variation is calculated as the variation of beat-to-beat SV from the mean value during the most recent 20 s data: $SVV=(SV_{max}-SV_{min})/SV_{mean}$.

ΔSVV was defined as the difference between SVV before and after VE. The mean values of the three consecutive SVV determinations were used for statistical analysis (more than 1 min).

Calculation of pulse pressure variation

Pulse pressure (PP) was defined as the difference between systolic and diastolic arterial blood pressure. Maximal (PP_{max}) and minimal (PP_{min}) values were determined over the same respiratory cycle. Pulse pressure variation (PPV) was then calculated as: $PPV=(PP_{max}-PP_{min})/[(PP_{max}+PP_{min})/2]$ as previously described.[8] PPV was evaluated in triplicate over each of three consecutive respiratory cycles. The mean values of the three determinations were used for statistical analysis.

Pressure measurements

Central venous pressure, systolic, diastolic, and mean arterial pressure were recorded continuously. PAOP was determined at end-expiration and averaged from three consecutive respiratory cycles.

Echocardiographic measurements

Cardiac output (CO-TTE). Doppler echocardiography was performed by the same operator using an ultrasound device (EnVisor C, Philips Medical System) equipped with a phased array transthoracic probe (2.5 MHz). The SV was calculated as the product of the aortic valve area by the velocity time integral of aortic blood flow (VTIAo). Using the parasternal long-axis view, the diameter of the aortic cusp and the aortic valve area was calculated [π(diameter^2)/4]. As the diameter of the aortic orifice is assumed to remain constant in a given patient, the diameter was measured once at baseline. Using the apical five-chamber view, the VTIAo was computed from the area under the envelope of the pulsed-wave Doppler signal obtained at the level of the aortic annulus. The VTIAo value was averaged over five consecutive measurements. CO was calculated as the product of heart rate by SV.[20] The operator was unaware of PPV values and of variables measured by Vigileo (SVV, CO-Vigileo) and by CAP (CO-PAC, CVP, and PAOP).

Left ventricular ejection fraction. Left ventricular ejection fraction (LVEF) was measured using the biplane Simpson's method from the apical two- and four-chamber views.

Study design

For VE, we used 20 ml×BMI of albumin 4% (Albumine-LFB® 4%). The fluid bolus was administered rapidly over 20 min. Two sets of measurements were performed: the first before VE and the second immediately after VE. CO-TTE, CO-Vigileo, CO-PAC, SVV, PPV, CVP, and PAOP were simultaneously measured. Ventilatory settings and dosages of inotropic and vasopressive drugs were kept constant during the study period.

Statistical analysis

Results were expressed as mean (SD) if the data were normally distributed or median [25–75% interquartile range] if not. The effects of VE on haemodynamic parameters were assessed using a non-parametric Wilcoxon rank sum test. Assuming that a 15% change in CO was required for clinical significance, patients were separated into Rs and NRs by change in CO-TTE$\geq$15% and $<$15% after the volume challenge.[7 8] Haemodynamic parameters before VE in Rs and NRs were compared with a non-parametric Mann–Whitney test. The relationship between (i) SVV, changes in SVV, PPV, CVP, and PAOP, and changes in CO, (ii) baseline SVV and baseline PPV, and (iii) changes in SVV and in PPV were evaluated using a Spearman correlation.

Cardiac output obtained with TTE, PAC, and Vigileo at baseline and after VE was compared using Bland and Altman method.[21]

The relationship between the change in CO-TTE and CO-Vigileo and between the change in CO-PAC and CO-Vigileo after VE were evaluated using a Spearman correlation.

Receiver operating characteristic (ROC) curves were generated for CVP, PAOP, PPV, and SVV varying the discriminating threshold of each parameter, and area under the ROC curves (95% CI) were calculated and compared.[22] Values for each area can be between 0 and 1. A value of 0.5 indicates that the screening measure is no better than chance, whereas a value 1 implies perfect performance. In our study, the area under the ROC curve represented the probability that a random pair of R and NR after VE would be correctly ranked by the haemodynamic variable measurement.

A P-value of <0.05 was considered to be statistically significant. Statistical analysis was performed using Statview (software 5.0; SAS Institute Inc.) and Medcalc (software 8.1.1.0; Mariakerke, Belgium).

Results

Global analysis

Forty patients were initially included. Five patients were excluded from analysis for arrhythmia during the protocol (two patients) or difficulties in transthoracic echocardiographic images analysis (three patients). The characteristics of the 35 final studied patients and their diseases leading to transplantation are reported in Table 1.

Table 1 Patient characteristics and diseases leading to transplant (n=35). Mean (SD) or number. HCV, hepatitis C virus; HBV, hepatitis B virus; LVEF, left ventricular ejection fraction; MELD, model for end-stage liver disease

Characteristics	
Age (yr)	51 (11)
Gender	
Male	23
Female	12
Weight, kg	67 (14)
Body mass index (kg m^{-2})	23 (3)
Postoperative plasma albumin concentration (g $litre^{-1}$)	21.7 (3.2)
LVEF (%)	71 (7)
Norepinephrine ($\mu g\ kg^{-1}\ min^{-1}$)	0.31 (0.2)
Tidal volume (ml kg^{-1})	8.4 (0.6)
Respiratory rate (cycles min^{-1})	15 (2)
Plateau pressure (cm H_2O)	19 (3)
Underlying disease	
Alcoholic cirrhosis	19
HCV cirrhosis	11
HBV cirrhosis	2
Other	3
Child Pugh classification (A/B/C)	7/14/14
MELD score	17 (6)

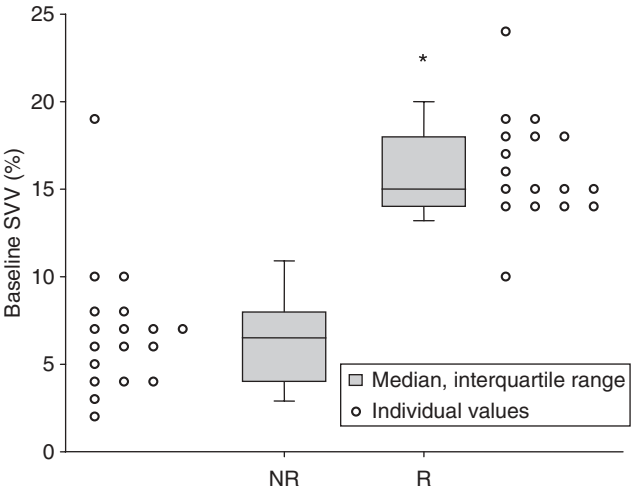

Fig 1 Median values, interquartile range and individual values of baseline values of stroke volume variation (SVV) in responders (R) and non-responders (NR). * P<0.001 *vs* NR.

Twelve patients received β-blockers (oesophagus varicose rupture prevention) and five patients received calcium blockers (arterial hypertension n=3, and supraventricular arrhythmia n=2). The decision to give fluid was made for tachycardia (eight patients), mottling (seven patients), low urine output (14 patients), and functional renal impairment (six patients). Seventeen patients were Rs (CO-TTE increase ≥15%) and 18 were NRs. Haemodynamic measurements in Rs and NRs at baseline and after VE are given in Table 2. The static compliance of the respiratory system was not different at baseline and after VE: 28 (4) *vs* 29 (4) ml cm H_2O^{-1}, respectively, P>0.05.

Fluid responsiveness

Before VE, SVV, and PPV were significantly higher and CO, PAOP, and CVP were significantly lower in Rs than in NRs (Fig. 1 and Table 2). After VE, CVP, PAOP, PPV, and SVV presented significant changes in Rs and NRs.

There was no correlation between baseline values of both CVP and PAOP and the per cent change in CO-TTE after fluid challenge (P>0.05). In contrast, the baseline SVV and PPV correlated significantly and closely with the change in CO-TTE induced by fluid challenge (respectively, r^2=0.72, P<0.0001; r^2=0.84, P<0.0001) (Fig. 2).

Volume expansion induced a significant decrease in SVV (ΔSVV) (P<0.005), which was significantly correlated with the VE-induced increase in CO (r^2=0.51; P<0.0001).

Baseline SVV was correlated with baseline PPV (r^2=0.67; P<0.0001).

The change in SVV after VE was correlated with the change in PPV (r^2=0.53, P<0.001) after VE.

A 10% SVV threshold discriminated between Rs and NRs with a sensitivity of 94% (95% CI: 71–99) and a specificity of 94% (95% CI: 73–99) (Fig. 3).

The area under the ROC curve, showing the ability of the haemodynamic parameters to discriminate between Rs

Table 2 Haemodynamic variables before and after volume expansion in fluid responders and fluid non-responders. HR, heart rate; MAP, mean arterial pressure; CO-TTE, cardiac output obtained with transthoracic echocardiography; SVR, systemic vascular resistance; CVP, central venous pressure; MPAP, mean pulmonary arterial pressure; PAOP, pulmonary arterial occlusion pressure; SVV, stroke volume variation; PPV, pulse pressure variation. Median [25–75% interquartile range]. P1, volume expansion value *vs* baseline value in non-responders; P2, baseline value in responders *vs* baseline value in non-responders; P3, volume expansion value *vs* baseline value in responders

Variables	Fluid non-responders (n=18)			Fluid responders (n=17)			
	Baseline	Volume expansion	P1	Baseline	P2	Volume expansion	P3
HR (beats min^{-1})	62 [55–69]	58 [55–63]	<0.05	72 [75–84]	NS	65 [62–79]	<0.05
MAP (mm Hg)	90 [80–104]	91 [85–102]	NS	83 [65–94]	NS	91 [80–106]	<0.05
CO-TTE (litre min^{-1})	6.3 [5.9–7.1]	6.8 [6–7.8]	<0.05	5.8 [5.2–6.7]	<0.05	6.9 [6.4–8.1]	<0.05
SVR (dyn s^{-1} cm^{-5})	975 [821–1200]	921 [741–1177]	NS	970 [820–1238]	NS	812 [742–1025]	NS
CVP (mm Hg)	7 [5–10]	12 [8–14]	<0.05	6 [2–8]	<0.05	10 [6–11]	<0.05
MPAP (mm Hg)	18 [14–24]	22 [16–25]	<0.05	15 [11–19]	<0.05	22 [16–24]	<0.05
PAOP (mm Hg)	12 [10–12]	13 [11–14]	<0.05	10 [7–12]	<0.05	12 [10–13]	<0.05
SVV (%)	6 [4–8]	4 [4–5]	<0.05	15 [14–18]	<0.05	7 [5–12]	<0.05
PPV (%)	6 [5–9]	4 [3–5]	<0.05	16 [15–19]	<0.05	7 [3–11]	<0.05

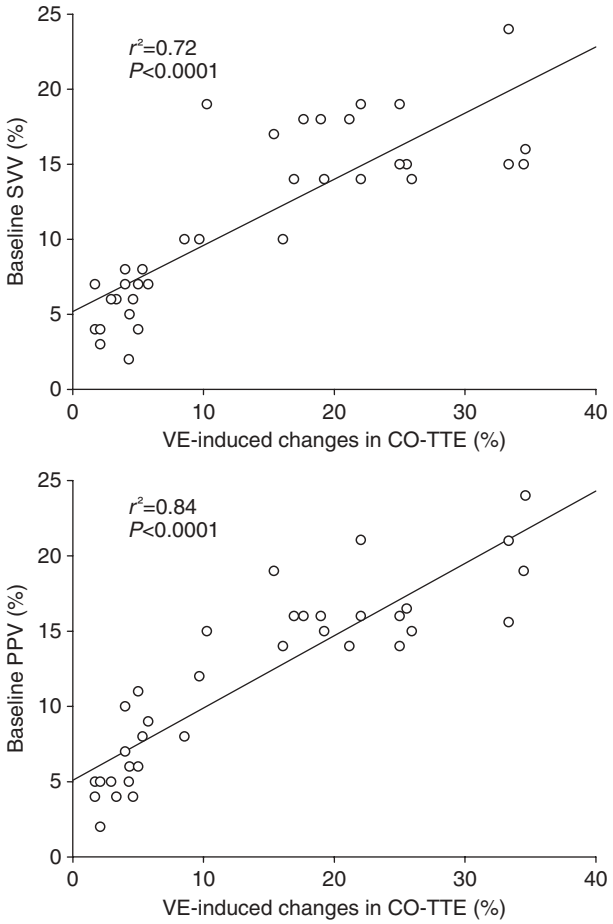

Fig 2 Relationship between baseline values of stroke volume variation (SVV) and volume expansion (VE)-induced changes in cardiac output measured by TTE (CO-TTE), and between pulse pressure variation (PPV) and VE-induced changes in CO-TTE.

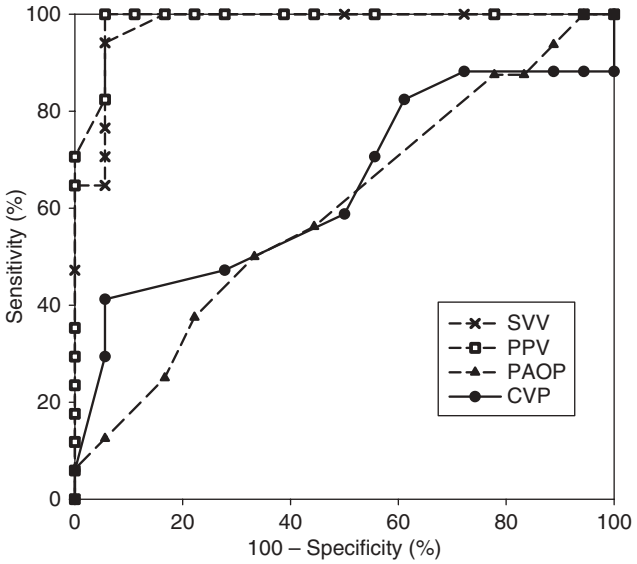

Fig 3 Receiver operating characteristic curves comparing the ability of stroke volume variations (SVVs), pulse pressure variations (PPVs), central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) before volume expansion to discriminate Rs and NRs (cardiac output measured with TTE). The best threshold values showing the ability of various haemodynamic parameters to predict fluid responsiveness were >10% for SVV, >12% for PPV, ≤10 mm Hg for PAOP, and ≤3 mm Hg for CVP. Areas under the ROC curve (95% CI) were 0.95 (0.81–0.99) for SVV, 0.98 (0.87–0.99) for PPV, 0.60 (0.42–0.76) for PAOP, and 0.64 (0.44–0.78) for CVP.

and NRs, is shown in Figure 3. There was no significant difference between the area under the ROC curve for SVV and PPV.

CO comparison

The values of CO-TTE, CO-PAC, and CO-Vigileo, before and after VE, are shown in Table 3. Bias and 95% limit of agreement between CO-Vigileo and CO-TTE, and between CO-Vigileo and CO-PAC are reported in Figure 4. After VE, the percentage change in CO-Vigileo correlated with the percentage change in CO-TTE (r^2=0.74, P<0.0001) and with the percentage change in CO-PAC (r^2=0.77, P<0.0001).

Table 3 Values of cardiac output obtained with transthoracic echocardiography (CO-TTE), with pulmonary artery catheter (CO-PAC) and with Vigileo device (CO-Vigileo). Median [25–75% interquartile range]. *P<0.05 volume expansion *vs* baseline

	Baseline	After volume expansion
CO-TTE (litre min^{-1})	6.0 [5.2–7.1]	6.9 [6.2–7.8]*
CO-PAC (litre min^{-1})	6.0 [5.3–7.2]	7.3 [6.4–8.0]*
CO-Vigileo (litre min^{-1})	5.8 [5.3–6.5]	6.6 [6.2–7.3]*

Responder and NR classification was similar using CO-TTE or CO-PAC. Using CO-Vigileo, 34 patients were well classified (97%). Only one patient was classified as an NR with CO-Vigileo and as R with CO-TTE and CO-PAC.

Discussion

Our study demonstrates that uncalibrated SVV measurement by arterial waveform analysis (Vigileo System, FloTrac) can be used to predict the effects of VE in mechanically ventilated patients after liver transplantation, as well as PPV.

To our knowledge, two studies investigated the ability of the SVV obtained with the Vigileo system to predict the fluid responsiveness.[23 24] Hofer and colleagues compared the prediction of fluid responsiveness using SVV, as determined by the Vigileo system (software version 1.07) and the PiCCOplus system. The authors found, in 40 patients undergoing elective cardiac surgery, that SVV assessed using these two device exhibited similar performance in terms of fluid responsiveness. The optimal SVV-Vigileo threshold value discriminating R and NR was 9.6%. Conversely, de Waall and colleagues found that, in 18 patients undergoing coronary artery bypass grafting, SVV obtained with Vigileo was unable to predict fluid responsiveness. However, they used the first software generation (version 1.01) that operates with a re-calibration

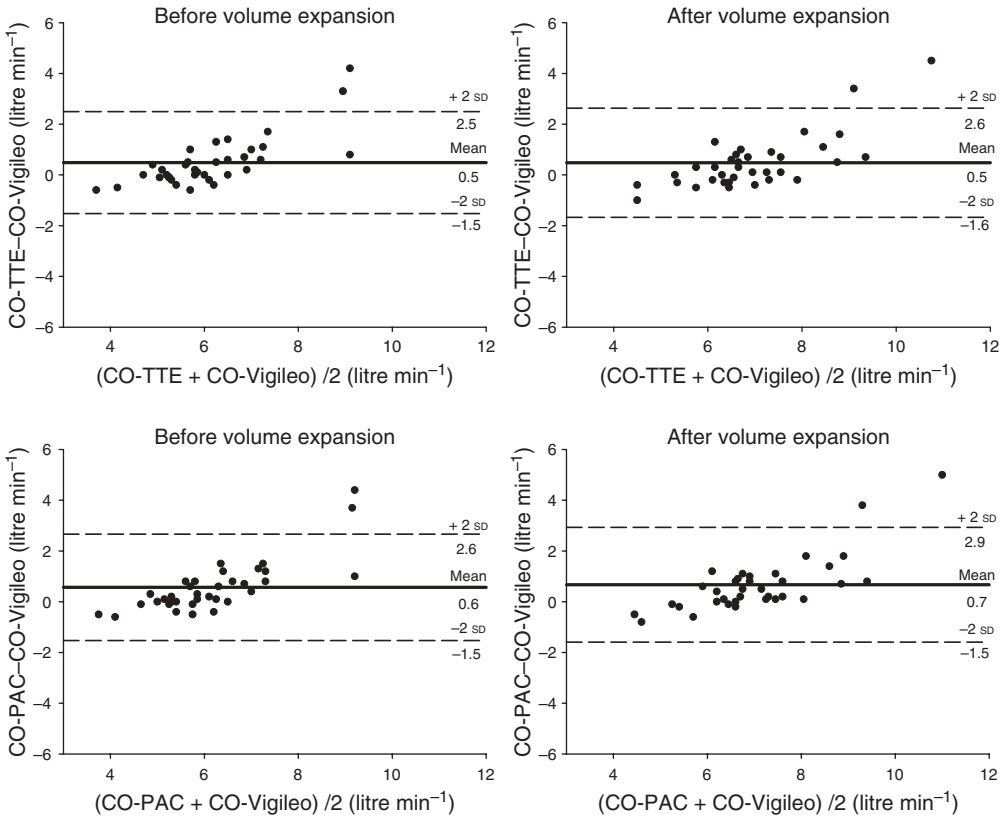

Fig 4 Bland–Altman plots between (upper panels) cardiac output measured by transthoracic echocardiography (CO-TTE) and by Vigileo™ device (CO-Vigileo) and between (lower panels) cardiac output measured by pulmonary artery catheter (CO-PAC) and by Vigileo™ device (CO-Vigileo), before and after fluid challenge. The continuous lines show the mean difference (bias) and the dotted lines show the 95% limits of agreement (2×SD).

interval of 10 min. This may, at least in part and as suggested by the authors, explain their negative findings. In our study, we used a software generation (version 1.07) that operates with a re-calibration interval of 1 min, and our results are consistent with those recently reported by Hofer and colleagues in a different patient population.

The best cut-off value of 10%, we report in the present study, is also in accordance with previous studies using another device (PiCCO™ system, Pulsion SG).[6 9] Indeed, this threshold is close to the thresholds of 9.5% described by Berkenstadt *et al.*[6] and Reuter *et al.*[9] and 12.5% found by Hofer *et al.*[25] with the PiCCO system.

The failure of CVP and PAOP to predict fluid responsiveness is in accordance with increasing evidence that static preload indicators are not suited for functional haemodynamic monitoring.[3]

Stroke volume variation and PPV have previously been studied in patients during brain surgery, in critically ill patients after cardiac surgery and in patients suffering from septic shock.[6 8 9 25] These indices are highly sensitive in predicting fluid responsiveness in these settings. In the present study, we found a correlation between baseline SVV and baseline PPV, in accordance with previously published data.[9 25] Indeed, aortic pulse pressure is directly proportional to left ventricular stroke volume and inversely related to aortic compliance. Respiratory changes in left ventricular stroke volume have been shown to be reflected by the changes in peripheral pulse pressure during the respiratory cycle.[26]

A cut-off of 15% is usually used to cope with the intrinsic variability of CO measurements and to define a clinically relevant change. However, NRs presented statistically, but not clinically, the relevant changes in CO. Furthermore, we observed statistically significant changes in HR, CVP, MPAP, PAOP, SVV, and PPV after VE in NRs.

Invasive pressures (CVP, MPAP, and PAOP) showed a statistical difference between baseline values in Rs and NRs. However, their interquartile ranges were large with an overlapping. In contrast, interquartile ranges for SVV and PPV did not overlap.

In addition to intravascular volume status, SVV is affected by the depth of airway pressure and tidal volume.[27 28] Large tidal volumes, reduced chest wall compliance, and air trapping may cause exaggerated SVV values. In the present study, mechanical ventilation was performed in a volume-controlled mode with a tidal volume of 8–10 ml kg^{-1}, a PEEP of 3 cm H_2O, and an inspiratory/expiratory ratio of 0.5. These parameters remained stable during the procedure. Furthermore, the static compliance of the respiratory system was not different before or after VE.

In the present study, all patients received norepinephrine before VE. Nouira and colleagues[29] showed in an experimental study in six dogs that norepinephrine could significantly reduce the value of PPV. Our findings confirm that while norepinephrine may affect the absolute PPV value, it does not affect its clinical value as a predictor of fluid responsiveness.

We found that CO-Vigileo was able to track the changes in CO induced by VE. To our knowledge, this is the first clinical study to investigate this issue. Correlations between the changes in CO-Vigileo and the changes in CO-TTE and in CO-PAC were very close. Furthermore, the R classification using CO-Vigileo was exact in 97% of patients. Only one patient was classified as R using CO-TTE or CO-PAC and as an NR using CO-Vigileo. This patient presented a percentage change in CO induced by VE just over 15% (CO-TTE: 16.9%, CO-PAC: 15.6%, CO-Vigileo: 13.3%), thus explaining the classification mistake.

Our study has some limitations. First, Rs and NRs were defined by CO obtained by TTE. We did not use CO obtained with PAC because it was semi-continuous CO determination and was not suitable for tracking rapid changes in CO.[30–32] However, the classification of Rs and NRs using CO-TTE or CO-PAC was similar. TTE has its inherent limitations but we took care to obtain interpretable measurements: the VTIAo was averaged over five consecutive measurements and three patients were excluded for unsatisfactory cardiac echogenicity. However, four patients presented a VE-induced change in CO just over 15% (15.4–17.6%). The accuracy and the reproducibility of CO measurement by TTE were not sufficient to guarantee that they were all Rs. Second, we excluded patients with spontaneous breathing activity or cardiac arrhythmias because respiratory variations in haemodynamic signals are ineffective.[4 33] Indeed, in patients with cardiac arrhythmia, the beat-to-beat variation in stroke volume may no longer reflect the effects of mechanical ventilation. This is particularly true in patients with atrial fibrillation or frequent extrasystoles. In patients with few-and-far-between extrasystoles, the arterial pressure curve can still be analysed if the cardiac rhythm is regular during at least one respiratory cycle. Third, as we studied patients with an LVEF of $>50\%$, our results cannot be extrapolated to patients with heart failure. Finally, the study was performed in patients sedated and mechanically ventilated with a tidal volume ≥ 8 ml kg^{-1}, and it has been shown that SVV is affected by the depth of tidal volume under mechanical ventilation.[27 28] The ability of SVV calculated by Vigileo to predict fluid responsiveness in patients ventilated with low tidal volume (<8 ml kg^{-1}) remains to be studied.

In conclusion, our findings suggest that, in mechanically ventilated patients with circulatory failure after liver transplantation, (i) SVV calculated by the Vigileo monitor is a useful predictor of increased CO in response to VE and (ii) CO-Vigileo is able to track changes in CO induced by VE.

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