

# Accuracy of arterial pressure waveform analysis for cardiac output measurement in comparison with thermodilution methods in patients undergoing living donor liver transplantation

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## Abstract

**Purpose** The aim of this study was to assess the accuracy of the first and third versions of arterial pressure waveform cardiac output (APCO<sub>v.1.0</sub> and APCO<sub>v.3.0</sub>) measurements in comparison with thermodilution methods in patients undergoing living donor liver transplantation.

**Methods** Twenty patients were anesthetized and mechanically ventilated. A radial arterial line was connected to a dedicated transducer for the APCO evaluation (FloTrac<sup>TM</sup>). A pulmonary artery catheter was placed and connected to a computer system (Vigilance<sup>TM</sup>) to measure intermittent thermodilution cardiac output (CO<sub>TD</sub>) and continuous cardiac output (CCO).

**Results** A total of 138 measurements were analyzed. Bland–Altman analysis showed that the mean biases for CO<sub>TD</sub>–APCO<sub>v.3.0</sub>, CO<sub>TD</sub>–APCO<sub>v.1.0</sub>, and CO<sub>TD</sub>–CCO were 0.89, 1.73, and –0.79 L/min, and the adjusted percentage errors were 37.5, 30.3, and 43%, respectively. While the variance for CO<sub>TD</sub>–APCO<sub>v.3.0</sub> was greater, the accuracy (bias) improved by 0.8 L/min as compared with CO<sub>TD</sub>–APCO<sub>v.1.0</sub>. The difference CO<sub>TD</sub>–APCO<sub>v.3.0</sub> became apparent when systemic vascular resistance was lower than 1000 dyne × s/cm<sup>5</sup>, especially below 700 dyne × s/cm<sup>5</sup>.

**Conclusion** These data suggest that the accuracy of APCO<sub>v.3.0</sub> has improved compared to APCO<sub>v.1.0</sub> due to the updated algorithm, but additional improvements should be evaluated, especially in patients undergoing living donor liver transplantation with low systemic vascular resistance.

**Keywords** Cardiac output · Arterial pressure waveform cardiac output · Thermodilution · Systemic vascular resistance

## Introduction

Accurate assessment of cardiac output (CO) is important for intraoperative circulatory management in patients undergoing liver transplantation. One most reliable and widely accepted method for the intraoperative measurement of CO is a thermodilution technique using a pulmonary artery catheter. However, pulmonary artery catheterization is a relatively invasive approach.

Recently, a CO monitoring system using arterial pulse waveform analysis (APCO, FloTrac<sup>TM</sup>/Vigileo<sup>TM</sup> system) has been developed, which does not require calibration or thermodilution. The accuracy and clinical applicability of this system with the original version of its software (APCO<sub>v.1.0</sub>) was evaluated and its clinical usefulness was demonstrated [1–3], but limitations have been reported in severe liver disease patients [4, 5]. To address these problems, a newer version of its software (APCO<sub>v.3.0</sub>) was developed. We designed this prospective study to compare the accuracy of APCO<sub>v.3.0</sub> with APCO<sub>v.1.0</sub> on the basis of CO measurements by intermittent and continuous thermodilution methods.

## Methods

### Patients and surgery

After obtaining approval from the ethics committee of our institution and informed consent, 20 consecutive patients

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scheduled to undergo living donor liver transplantation due to chronic liver failure or hepatocellular carcinoma were enrolled in this study. Patients with pre-existing pulmonary and/or cardiac disease, other than the common symptoms of end-stage liver dysfunction, and patients with fulminant hepatic failure or pulmonary hypertension were excluded from the study. The Child–Pugh grade and Model for End-Stage Liver Disease (MELD) score were defined for each patient. Protocols of this study did not require any changes from the standard intraoperative monitoring and critical care management adopted at our institution. Administration of vasoactive agents and fluids was done at the discretion of attending anesthesiologists.

Patients were anesthetized with fentanyl and propofol. Vecuronium was administered for muscle relaxation. After tracheal intubation, anesthesia was maintained with isoflurane (end-tidal 1–2%) and fentanyl (30–50 µg/kg). The lungs were ventilated using volume-controlled ventilation with an oxygen–air mixture to maintain an arterial oxygen partial pressure of >150 mmHg and an arterial carbon dioxide partial pressure of around 40 mmHg.

#### Continuous cardiac output monitor using a pulmonary catheter

After the induction of anesthesia, a pulmonary artery catheter (CCO, Continuous Cardiac Output VIP Catheter with SvO<sub>2</sub>, 746HF8, 8 Fr, Edwards Lifesciences, Irvine, CA, USA) was inserted via the right internal jugular vein through an introducer (Sheath Introducer, 9Fr, Nihon Sherwood, Shizuoka, Japan), and it was connected to the Vigilance™ monitor (Edwards Lifesciences, Irvine, CA, USA) for CO monitoring. The position of the catheter was confirmed by its pressure wave and chest X-ray. The pulmonary arterial pressure and central venous pressure were monitored continuously.

#### Arterial pressure wave cardiac output monitor (APCO)

A 20 gauge, 1.16 inch-long arterial catheter (BD Angio-cath™, BD, Franklin Lakes, NJ, USA) was inserted into the radial artery. A dedicated transducer was connected to the arterial line for APCO evaluation (FloTrac™, Edwards Lifesciences, Irvine, CA, USA). FloTrac™ was connected to Vigileo™ monitor (Edwards Lifesciences, Irvine, CA, USA) for CO monitoring. This FloTrac™/Vigileo™ system needs no external calibration and provides CO measurements from the arterial pressure waveform analysis. The Vigileo™ records hemodynamic variables at 20-s intervals, performing its calculations on the most recent 20 s of data. The system calculates the stroke volume (SV) using arterial pulsatility, resistance, and compliance. The CO is calculated as follows:  $CO = \text{heart rate} \times SV$ .

Stroke volume =  $k \times \text{Pulsatility}$ ,

where  $k$  is a constant quantifying arterial compliance and vascular resistance, derived from a multivariate regression model including Langewouter's aortic compliance, skewness and kurtosis of the pressure curve. The rate of adjustment of  $k$  was 1 min. Pulsatility is proportional to the standard deviation of the arterial pressure wave over a 20-s interval.

We prepared one Vigileo™ monitor that was equipped with version 1.1 of the software. Measurements on version 3 of the software were simulated on a dedicated laptop computer to simultaneously measure both APCO<sub>v1.0</sub> and APCO<sub>v3.0</sub>.

#### Thermodilution CO/CCO monitoring

A conventional pulmonary thermodilution method for CO monitoring (CO<sub>TD</sub>) was carried out with a bolus injection of 10 mL iced dextrose 5% solution. For CCO monitoring, a safe level of heat was transferred to the blood by a computer-controlled thermal filament mounted on the pulmonary artery catheter. To eliminate the natural temperature variations in the pulmonary artery, heat was transferred to the blood in a pseudo-random, on–off fashion. The observed changes in pulmonary artery temperature were recorded by the distal rapid-response thermister in the pulmonary artery. There was no need for user calibration because the Vigilance™ system automatically computed a cross-correlation between the filament input sequence, the power, and the distal thermister response to blood warming. From this cross-correlation, CO was calculated using a modified Stewart–Hamilton equation. CCO was time-averaged CO values over 3–6 min periods and was continuously updated (about every 60 s).

#### Study protocol

Synchronized measurements of cardiac output using four methods were made at the following predefined time points.

- T1: pre-operative
- T2: soon after operation started
- T3: pre-liver resection
- T4: during the anhepatic phase
- T5: post-reperfusion
- T6: end of operation

Between T2 and T3, the diseased liver was freed from the attachments and vascular structures of the liver were prepared for transplantation. After T4, the donor liver was revascularized and reperfused. In some patients with insufficient collateral circulation, venovenous bypass was

instituted from the femoral vein or portal vein to the subclavian vein. Patients were admitted to the intensive care unit under mechanical ventilation after the surgery was completed.

### Statistical analysis

All results are expressed as mean  $\pm$  standard deviation (SD) unless indicated otherwise. Statistical analysis was computed with JMP 7 (SAS Institute Inc., Cary, NC, USA). Data were analyzed using Student's *t* test, analysis of variance with the Bonferroni test for post-test comparisons, and the Bland–Altman method [6]. The bias (the mean difference between two methods) represents the systemic error between both methods. The limits of agreement were calculated as the bias  $\pm$ 2SD, and defined the range in which 95% of the differences between the methods were expected to fall. A *P* value  $<$ 0.05 was considered significant.

We used the Bland and Altman method modified by Myles and Cui [7], using a random effect model, to take account of repeated measures. The percentage error was calculated as the ratio of 2SD of the bias to the mean CO, and was considered clinically acceptable if it was below 30%, as proposed by Critchley and Critchley [8].

**Table 1** Patient characteristics and diseases leading to transplant

Characteristics	
Age (years)	54.5 $\pm$ 9.2
Gender (male/female)	12/8
Height (cm)	162.2 $\pm$ 9.8
Weight (kg)	60.7 $\pm$ 11.2
BSA (m <sup>2</sup> )	1.65 $\pm$ 0.20
ASA (1/2/3)	4/14/2
Child–Pugh classification (A/B/C)	2/6/12
Indication of liver transplantation ( <i>n</i> )	HCV cirrhosis 15 HBV cirrhosis 1 Others 4

Data are expressed as the mean  $\pm$  SD or a number

**Table 2** Comparison of biases, SDs, limits of agreement, mean COs and percentage errors among APCO<sub>v3.0</sub>, APCO<sub>v1.0</sub>, and CCO using the Bland–Altman method (compared with CO<sub>TD</sub> using a random effect model)

Method	Bias (l/min)	SD (l/min)	Above limit (l/min)	Below limit (l/min)	Mean CO (l/min)	Percentage error (%)
APCO <sub>v3.0</sub>	0.89	1.35	3.54	–1.76	6.3	37.5
APCO <sub>v1.0</sub>	1.73	1.09	3.87	–0.41	5.9	30.3
CCO	–0.79	1.55	2.25	–3.83	7.2	43.0

SD standard deviation, CO cardiac output, APCO<sub>v3.0</sub> cardiac output measured using the Vigileo<sup>TM</sup>/FloTrac<sup>TM</sup> system (software version 3.0), APCO<sub>v1.0</sub> cardiac output measured using the Vigileo<sup>TM</sup>/FloTrac<sup>TM</sup> system (software ver 1.0), CCO continuous cardiac output measured using the Vigilance<sup>TM</sup> system, CO<sub>TD</sub> cardiac output measured using intermittent thermodilution

### Results

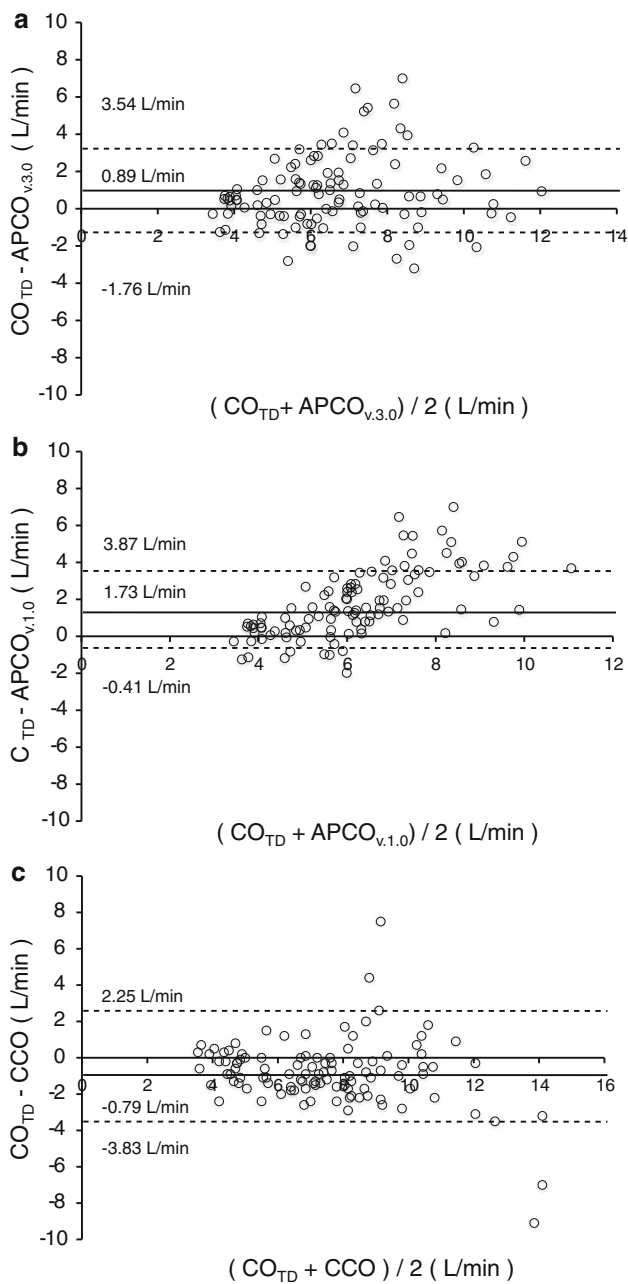
Patient characteristics are shown in Table 1. The mean MELD score was 11 (range 2–23). All patients finished the operations successively according to the protocol, and they were transferred to ICU and ventilated until they matched the extubation criteria. During surgery, cardiovascular agents such as phenylephrine, noradrenaline, dopamine, and dobutamine were used in all patients to obtain hemodynamic stability. There was no perioperative mortality.

A total of 150 data sets were collected. Twelve data sets were excluded because artifact measurements caused by electrical disturbances or surgical manipulations were seen in those data sets. Thus, a total of 138 data sets were available for the analysis.

Cardiac output data in CO<sub>TD</sub> was considered the standard CO. Numerical comparisons of these values are shown in Table 2. The overall median (range) cardiac output was 7.1 (3.0–12.5) L/min in CO<sub>TD</sub>, whereas it was 6.3 (3.4–11.6) L/min in APCO<sub>v3.0</sub>, 5.2 (3.4–9.2) L/min in APCO<sub>v1.0</sub>, and 7.9 (3.3–18.4) L/min in CCO. There were significant differences among these four cardiac output measurements (*P*  $<$  0.01).

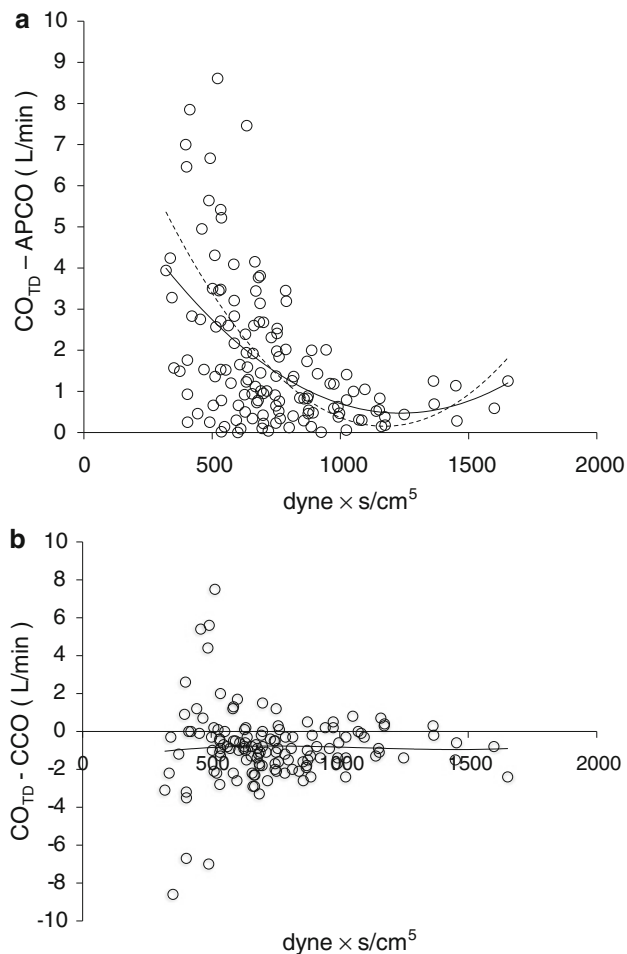
The mean bias between CO<sub>TD</sub> and APCO<sub>v3.0</sub> was 0.89 L/min, with 95% limits of agreement of –1.76 to 3.54 L/min. The adjusted percentage error was 37.5% (Fig. 1a). The mean bias between CO<sub>TD</sub> and APCO<sub>v1.0</sub> was 1.73 L/min, with 95% limits of agreement of –0.41 to 3.87 L/min. The adjusted percentage error was 30.3% (Fig. 1b). The mean bias between CO<sub>TD</sub> and CCO was –0.79 L/min, with 95% limits of agreement of –3.83 to 2.25 L/min. The adjusted percentage error was 43.0% (Fig. 1c). Because we expected to see differences in hemodynamic condition between before (T1–T4) and after reperfusion (T5–T6), we tried to compare the CO data between before and after reperfusion. However, the CO data could not demonstrate any differences between before and after reperfusion (data not shown).

Figure 2 shows regression analyses between systemic vascular resistance (SVR) and the differences in cardiac output CO<sub>TD</sub>–APCO<sub>v3.0</sub> (Fig. 2a) and CO<sub>TD</sub>–CCO (Fig. 2b). In Fig. 2a, we have also added the regression line



**Fig. 1a–c** Bland–Altman plots of  $CO_{TD}-APCO_{v3.0}$  (a),  $CO_{TD}-APCO_{v1.0}$  (b), and  $CO_{TD}-CCO$  (c). The *unbroken lines* show the mean difference and the *dotted lines* show the 2SD limits of agreement.  $CO_{TD}$  cardiac output measured using intermittent thermodilution,  $APCO_{v3.0}$  cardiac output measured using the Vigileo<sup>TM</sup>/FloTrac<sup>TM</sup> system (software version 3.0),  $APCO_{v1.0}$  cardiac output measured using the Vigileo<sup>TM</sup>/FloTrac<sup>TM</sup> system (software version 1.0),  $CCO$  continuous cardiac output measured using the Vigilance<sup>TM</sup> system

showing the correlation between SVR and the difference in cardiac output  $CO_{TD}-APCO_{v1.0}$ . The difference  $CO_{TD}-APCO_{v3.0}$  was correlated with SVR, leading to a larger difference at a lower systemic vascular resistance, although this difference was smaller than that observed for  $CO_{TD}-$



**Fig. 2a–b** Regression analyses between systemic vascular resistance (SVR) and the differences in cardiac output between  $CO_{TD}$  and  $APCO_{v3.0}$  or  $APCO_{v1.0}$  (a), or between  $CO_{TD}$  and  $CCO$  (b). The *solid lines* show the regression lines for  $CO_{TD}-APCO_{v3.0}$  and  $CO_{TD}-CCO$ , while the *dashed line* shows the regression line of  $CO_{TD}-APCO_{v1.0}$ .  $CO_{TD}$  cardiac output measured using intermittent thermodilution,  $APCO_{v3.0}$  cardiac output measured using the Vigileo<sup>TM</sup>/FloTrac<sup>TM</sup> system (software version 3.0),  $CCO$  continuous cardiac output measured using the Vigilance<sup>TM</sup> system

$APCO_{v1.0}$ . The difference  $CO_{TD}-CCO$  did not depend on SVR.

**Discussion**

Since the APCO system (FloTrac<sup>TM</sup>/Vigileo<sup>TM</sup>) was first introduced in 2004, there have been many reports investigating the accuracy of APCO for a cardiac output monitor [1–5, 9–12]. Results were inconsistent among the reports. Recently published studies showed clinically unacceptable bias and limits of agreement between APCO and  $CO_{TD}$ , and between APCO and transpulmonary thermodilution CO in patients undergoing cardiac surgery, liver transplantation, or in patients with septic shock [4, 5, 9–12]. On

the other hand, clinically acceptable bias and limits of agreement between APCO and  $CO_{TD}$  or between APCO and transpulmonary thermodilution CO have also been reported in surgical patients [1–3].

Our results suggest that the measurement of cardiac output using a newer version of APCO (APCO<sub>v3.0</sub>) still tends to underestimate cardiac output in patients undergoing living donor liver transplantation when compared with  $CO_{TD}$ . We found a bias of 0.9 L/min and 95% limits of agreement of  $-1.8$  to 3.5 L/min for  $CO_{TD}$ –APCO<sub>v3.0</sub>. Although the bias of APCO<sub>v3.0</sub> was better than that for APCO<sub>v1.0</sub>, which had a bias of 1.73 L/min and 95% limits of agreement of  $-0.41$  to 3.87 L/min for  $CO_{TD}$ , the percentage error for APCO<sub>v3.0</sub> was 37.5%, which exceeds the 30% limit of acceptability [8]. We found that these differences were caused by certain previously underestimated CO data points that were addressed in v.3.0. Some underestimated data points were still seen in the modified algorithm. This resulted in a higher calculated variance, though overall accuracy improved substantially, as indicated in the lower bias.

As shown in Fig. 2a, the difference  $CO_{TD}$ –APCO<sub>v3.0</sub> became apparent when systemic vascular resistance was lower than  $1000 \text{ dyne} \times \text{s/cm}^5$ , especially below  $700 \text{ dyne} \times \text{s/cm}^5$ . The APCO system calculates CO using arterial pressure waveform analysis in conjunction with patient demographic data such as height, weight, age, and gender to estimate arterial properties [13]. The relation between pressure pulse waveform and stroke volume depends on the properties of the arterial vascular tree, such as compliance and resistance. The cardiovascular system in cirrhotic patients is characterized by high CO and low systemic vascular resistance [14, 15], characteristics which were also observed in our patients. Because the extent of low vascular resistance has a certain impact on the arterial pressure waveform [16], the hemodynamic characteristics of our patients may explain the large value of the bias and the percentage error for  $CO_{TD}$ –APCO<sub>v3.0</sub>. Costa et al. [4] also demonstrated that during liver transplantation, the bias and the 95% limits of agreement between APCO and transpulmonary thermodilution CO increased significantly in hyperdynamic conditions ( $CO > 8 \text{ L/min}$ ).

The discordant results of the APCO system with thermodilution CO methods are in agreement with a study by Biais et al. [5], in which the APCO<sub>v1.0</sub> values differed significantly from CCO values in patients undergoing liver transplantation, with a bias of 0.8 L/min, 95% limits of agreement of  $-1.8$  to 3.5 L/min, and a percentage error of 43%. They also demonstrated that the bias was significantly correlated with systemic vascular resistance, with lower systemic vascular resistance yielding larger bias [5]. Instead of using CCO as the control, we used  $CO_{TD}$  as a standard CO because  $CO_{TD}$  is considered to be more

accurate than CCO. Interestingly, De Backer et al. [17] reported an improvement in measurement using the same version of the software (version 3) in septic patients who suffered from low systemic vascular resistance. Also, they reported that the discordance between  $CO_{TD}$  and APCO (version 3) was not correlated with the extent of lower systemic vascular resistance, although the previous software version was significantly correlated. This might lead us to explain why we did not observe an improvement with version 3 of the software in these patients. Although both septic patients and patients with severe liver disease often manifest systemic hyperdynamic circulation and low systemic vascular resistance, their pathogeneses are thought to be different. Most of our patients had been suffering from liver cirrhosis, which is slowly progressive, and their average follow-up after being diagnosed is 17.2 years, as compared to the time course of septic patients, which is usually limited. As liver disease progresses and liver function deteriorates, the systemic hyperdynamic circulation becomes more apparent with the activation of the renin–angiotensin–aldosterone system [18]. Despite the presence of highly elevated levels of circulating vasoconstrictors, the presence of lower systemic vascular resistance may be explained by vascular hyporesponsiveness due to increased levels of vasodilators such as nitric oxide, as well as an autonomic neuropathy [18]. Desensitization of adrenergic receptor is also reported [19]. The complicated pathophysiology of these patients, which is different from septic or healthy patients, may contribute to the discordance between  $CO_{TD}$  and APCO<sub>v3.0</sub> in patients undergoing living donor liver transplantation.

Therefore, the clinical limitations of the APCO<sub>v3.0</sub> and APCO<sub>v1.0</sub> software should be understood when monitoring cardiac output in patients undergoing living donor liver transplantation who exhibit low systemic vascular resistance. It is suggested that the discordance we observed in lower systemic vascular resistance in this study is specific to these patients.

It is noteworthy that there were also large bias and limits of agreement between CCO and  $CO_{TD}$  in this study. Although APCO<sub>v3.0</sub> underestimated CO by a mean bias of 0.89 L/min ( $CO_{TD}$ –APCO<sub>v3.0</sub>), CCO overestimated CO by a mean bias of  $-0.79 \text{ L/min}$  ( $CO_{TD}$ –CCO). The underestimation by APCO<sub>v3.0</sub> was correlated with systemic vascular resistance, as shown in Fig. 2a, whereas the overestimation by CCO was not correlated with systemic vascular resistance, as shown in Fig. 2b. It must be noted that the manufacturer of APCO cautions clinicians that inaccurate measurements can occur under hyperdynamic conditions. Accuracy may be improved by calibrating APCO under these conditions, because APCO does offer accurate measurement in these patients when systemic vascular resistances are within the normal range.

Therefore, if the low systemic vascular resistance state can be predicted and/or adjusted by the pressure waveform or other variables, APCO can be a more useful CO monitor than CCO in patients undergoing liver transplantation.

It also should be noted that we are aware of the limitations of our study design. Because we believe that systemic vascular resistance is an important factor in the accuracy of the APCO, cardiovascular agents we used intraoperatively, such as phenylephrine, noradrenaline, and adrenaline, can influence the accuracy of the measurement. Since it is not unusual for these patients to suffer from hypotension during liver transplantation, all of the patients received these vasoconstrictive drugs intraoperatively. Because the dosages and infusion timings differed among patients, we could not compare all time points under similar conditions. An additional concern is the simultaneousness of the measurements. It is widely accepted that the thermodilution method is the standard, but  $CO_{TD}$  is an average of measurements made over minutes and may not correspond the data obtained by APCO simultaneously. CCO measurement also has a delay. It is difficult to synchronize all of these measurements. Even though we collected data sets when patients' hemodynamic conditions were stable, these time delays might have influenced our results.

In conclusion, the FloTrac<sup>TM</sup>/Vigileo<sup>TM</sup> system equipped with its updated software (APCO<sub>v3.0</sub>) shows some advantages in accuracy compared to the system using the older version of the software (APCO<sub>v1.0</sub>). APCO<sub>v3.0</sub> still has limitations when used to monitor patients undergoing living donor liver transplantation who exhibit low systemic vascular resistance. Continued improvements to the algorithm should be evaluated clinically in this subset of patients.

**Conflict of interest** Edwards Lifesciences Japan Ltd. provided monitoring instruments that were used throughout this evaluation. We received financial support from Edwards Lifesciences Japan Ltd. to carry out this study.

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