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Measurement of cardiac output: a comparison between transpulmonary thermodilution and uncalibrated pulse contour analysis[†‡]

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Background. Recently, continuous monitoring of cardiac output (CO) based on pulse contour analysis (Vigileo®) has been introduced into practice. In this clinical study, we evaluated the accuracy of this system by comparing it with the transpulmonary thermodilution technique (TPID) in septic patients.

Methods. We studied 24 mechanically ventilated patients with septic shock (16 male, 8 female, age 26–77 yr) receiving treatment with norepinephrine who for clinical indication underwent haemodynamic monitoring by the transpulmonary thermodilution technique using a PiCCO®plus system (Pulsion Medical Systems, Munich, Germany). In parallel, arterial pulse contour was applied using the femoral arterial pressure curve (FloTrac® pressure sensor, Vigileo® monitor, Edwards Lifesciences, Irvine, USA). After baseline measurement, mean arterial pressure was elevated by increasing norepinephrine dosage, and CO was measured again before mean arterial pressure was reduced back to baseline levels. Fluid status and ventilator settings remained unchanged throughout. At each time point, CO by transpulmonary thermodilution was calculated from three central venous bolus injections of 15 ml of saline ($<8^{\circ}$C). Linear regression and the Bland–Altman method were used for statistical analysis.

Results. Overall, CO was 6.7 (SD 1.8) (3.2–10.1) litre min^{-1} for CO(TPID) and 6.2 (2.4) (3.0–17.6) litre min^{-1} for CO(Vigileo®). Linear regression revealed: CO(Vigileo®)= 1.54+0.72×CO(TPID) litre min^{-1}, r^2=0.26 ($P<0.0001$). Mean bias between techniques [CO(TPID)–CO(Vigileo®)] was 0.5 litre min^{-1} (SD 2.3 litre min^{-1}). Correlation coefficients at the three time points were not significantly different from each other.

Conclusions. Pulse contour analysis-derived CO (Vigileo® system) underestimates CO(TPID) and is not as reliable as transpulmonary thermodilution in septic patients.

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Cardiac output (CO) is regarded as one of the most important haemodynamic variables for the assessment of cardiac function and guidance of therapy in critically ill patients. However, evidence of a positive influence on outcome by using invasive haemodynamic monitoring is

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lacking,[1 2] and alternative techniques have been suggested and introduced into clinical practice in recent years. For instance, measurement of CO by transpulmonary thermodilution has been proposed.[3 4] In this technique, a thermistor-tipped catheter is typically placed in the arterial system for the detection of the thermodilution time curve. Previously, a number of experimental and clinical studies showed that the measurement of CO by the transpulmonary thermodilution technique is comparable with that by a pulmonary artery catheter or other techniques.[5–9] Very recently, a new system based on pulse contour analysis without *in vivo* calibration (Vigileo®) has been developed and introduced into clinical practice. In this system, using the shape of the arterial pressure curve form, an arterial pressure CO algorithm is incorporated, which relates the blood flow to the arterial pressure using a haemodynamic model. The model uses basic cardiovascular haemodynamic concepts according to which the arterial circulation, acting as an elastic storage system, transforms the discontinuous flow owing to the pumping of the heart into steady flow in the peripheral organs.

The algorithm, which requires demographic patient data, is suggested to be robust to represent the flow–pressure relationship. So far, the data suggest that CO can be derived reliably by this technique when compared with pulmonary artery catheter (PAC)-based measurements.[10] However, these results as obtained in small patient populations are not convincing, and more recent studies[11 12] questioned the reliability of this system. In this study, we evaluated the Vigileo® system against transpulmonary thermodilution, which itself can be regarded as being as reliable as pulmonary arterial thermodilution in patients with septic shock under actual clinical conditions, i.e. changes in mean arterial pressure (MAP).

Methods

After approval by our local ethics committee, we enrolled 24 mechanically ventilated patients with septic shock (16 male, 8 female, age 26–77 yr) requiring treatment with norepinephrine, who for clinical indication underwent extended haemodynamic monitoring by the transpulmonary thermodilution technique (PiCCOplus®, Pulsion Medical Systems, Munich, Germany). All patients suffered from abdominal sepsis and were managed according to international guidelines on the treatment of patients with sepsis.[13]

Each patient had a 5F femoral arterial thermistor catheter (PV20L15, Pulsion Medical Systems, Munich, Germany) *in situ*. This system allows the determination of CO, intrathoracic blood volume (ITBV), extravascular lung water (EVLW) and global ejection fraction (GEF).[14–16]

In general, pressure transducers were calibrated against atmosphere at the mid-chest level. In parallel to haemodynamic monitoring by the transpulmonary thermodilution technique, we applied continuous CO measurements by arterial pulse contour analysis from the femoral arterial pressure curve (FloTrac® pressure sensor, Vigileo® monitor, Edwards Lifesciences, Irvine, CA, USA) with the software version V01.07, PIC V1.0. On the basis of the analysis of the shape of the arterial pressure form, an algorithm which relates the blood flow to the arterial pressure using a haemodynamic model enables determination of CO. The model uses basic cardiovascular haemodynamic concepts according to which the arterial circulation, acting as an elastic storage system, transforms the discontinuous flow owing to the pumping of the heart into steady flow in the peripheral organs. The algorithm requires demographic patient data, i.e. gender, age, height, weight and body surface area. This system enables the measurement of CO and also calculates the peripheral vascular resistance. The software for this device calculates CO every 20 s on the basis of the last 20 s interval of arterial waveform analysis.

Before the measurements, the pulse contour system was allowed to stabilize over a 10 min interval. No damping of the arterial pressure line, which could be achieved in all patients, was also a prerequisite for the measurements. After a baseline measurement of CO (baseline), MAP was elevated by increasing norepinephrine dosage (intervention), and control measurements were obtained after returning to baseline MAP values by reducing norepinephrine dosage (control). A MAP of approximately 90 mm Hg was targeted. At each time point, measurements were obtained about 5 min after reaching stable MAP, and the duration of the whole study period was about 15 min. Fluid status and ventilator settings remained unchanged throughout the study period. For clinical reasons, all patients were sedated and ventilated in a pressure-controlled mode (BiPAP, Evita 4, Draeger, Luebeck, Germany). In particular, airway pressures and the inspiratory oxygen fraction ($F_{I_{O_2}}$) remained constant in all patients. At each time point, CO by transpulmonary thermodilution was obtained from three central venous bolus injections of 15 ml of $<8^{\circ}$C cold normal saline. Before each injection, pulse contour CO was read from the Vigileo® monitor. Vigileo® CO values were obtained as the mean of the three values. Injections were made manually and not triggered by the respiratory cycle. The variation of the CO measurement was assessed by the coefficient of variation (CV), which was calculated within each patient and for each time point, and is calculated as CV=SD/mean (%).

Statistical analysis

All data are given as mean (SD). Haemodynamic parameters at the three time points were compared by an analysis of variance (ANOVA) on ranks and an all-pair-wise comparison procedure (Student–Newman–Keuls method). For the comparison of CO measurements and agreement between the two techniques, linear regression analysis and

the Bland–Altman method (mean bias, SD) were applied. Limits of agreement were defined by 2 SD. Furthermore, this type of analysis was performed separately for each time point, and a comparison between regression coefficients for all time points was made. Finally, changes in both techniques were correlated against each other by linear regression analysis. Statistical significance was considered at $P<0.05$. For the statistical analysis, we used SigmaStat® for Windows (version 1.0).

Results

Mean age was 58 (12) (58, range 26–77) yr, mean weight 83 (20) (77, range 56–150) kg and height 171 (12) (170, range 155–204) cm. Mean body surface area was 2.0 (0.3) (1.9, range 1.6–2.9) m^2. Severity of illness may be described by their mean APACHE II score of 26 (8) (25, range 7–43) and SAPS II score of 51 (15) (50, range 17–73), respectively. Patients' characteristics are summarized in Table 1. Heart rate, central venous pressure, GEF and EVLW were not different between the three time points. MAP was significantly higher during increased norepinephrine dosages as was systemic vascular resistance. It is noteworthy that, while CO(TPID) was unchanged, CO(Vigileo®) was not only different between baseline and intervention but also between baseline and control (Table 2).

Overall, the range of CO was 3.2–10.1 litre min^{-1} for CO(TPID) and 3.0–17.6 litre min^{-1} for CO(Vigileo®). Linear regression analysis revealed: CO(Vigileo®)= $1.54+0.72\times$CO(TPID) litre min^{-1}, $r^2=0.26$ ($P<0.0001$). The Vigileo® system underestimated CO(TPID), and the mean bias between both techniques was 0.5 litre min^{-1} (SD 2.3 litre min^{-1}) (Figs 1 and 2). With regard to each of the three time points, we found ranges of 3.5–13.4 litre min^{-1} CO(Vigileo®) and 3.4–9.7 litre min^{-1} CO(TPID) (baseline), 4.6–17.6 litre min^{-1} CO(Vigileo®) and 3.4–10.1 CO(TPID) (intervention), and 3.0–12.7 litre min^{-1} CO(Vigileo®) and 3.2–9.9 litre min^{-1} CO(TPID) (control), respectively (Figs 3–5). For each time point, r^2 was 0.10 ($P=0.13$), 0.30 ($P=0.006$) and 0.21 ($P=0.02$), respectively. When compared with each other, CVs were not statistically significantly different: the linear regression analysis between changes in CO(Vigileo®) and CO(TPID) revealed $r^2=0.14$ ($P=0.01$) (Fig. 6). As one patient (no. 10) was an extreme outlier, we re-analysed our data without this particular patient. However, the results were similar, as we found an overall correlation of $r^2=0.13$ ($n=69$).

As a measure of measurement variability, we calculated the CV for each time point and for each patient. Mean CVs were 5.8, 6.7 and 5.2% for CO(Vigileo®) and 6.7, 6.7 and 6.7% for CO(TPID) at the three time points.

Discussion

Our data show that the measurement of CO by pulse contour analysis using a system without *in vivo* calibration (Vigileo®) underestimates CO(TPID) and does not correlate well with transpulmonary thermodilution. Furthermore, changes in both techniques were also only poorly

Table 1 Patients' characteristics and data at baseline. M, male; F, female; HR, heart rate; MAP, mean arterial pressure; CO(TPID), cardiac output by transpulmonary thermodilution; CO(Vigileo®), cardiac output by the Vigileo® system

Number	Gender	Age (yr)	Weight (kg)	Height (cm)	HR (min^{-1})	MAP (mm Hg)	CO(TPID) (litre min^{-1})	CO(Vigileo®) (litre min^{-1})
1	M	48	103	184	77	83	7.9	4.8
2	M	64	75	175	71	75	8.8	4.8
3	F	72	85	161	97	77	5.0	4.1
4	M	68	65	175	100	60	8.2	6.5
5	M	53	70	170	85	74	7.8	4.4
6	F	70	87	158	100	65	7.5	6.6
7	M	57	108	190	81	62	9.7	6.0
8	F	72	56	170	73	51	6.0	3.6
9	M	54	75	170	98	59	3.5	4.6
10	M	46	150	204	124	67	8.4	13.4
11	M	54	80	158	100	57	6.3	5.9
12	M	71	78	172	135	65	7.4	7.2
13	M	53	70	169	86	61	5.5	5.8
14	F	45	80	160	122	74	4.3	5.8
15	F	26	85	170	71	67	7.1	4.7
16	M	60	75	175	100	54	8.8	4.9
17	M	43	95	180	107	66	7.2	6.4
18	M	62	70	169	61	64	3.4	3.5
19	M	66	93	172	101	43	5.7	4.3
20	F	77	70	165	119	73	6.7	5.9
21	M	58	115	185	94	74	7.0	8.3
22	F	39	70	165	115	73	5.7	5.8
23	F	72	70	155	114	66	3.3	5.8
24	M	56	70	150	109	74	7.9	5.0

Table 2 Results are mean (SD) [median]. MAP, mean arterial pressure; CVP, central venous pressure; CO(TPID), transpulmonary thermodilution-derived cardiac output; SVR, systemic vascular resistance; ITBVI, intrathoracic blood volume index; GEDVI, global end-diastolic volume index; GEF, global ejection fraction; EVLWI, extravascular lung water index; NEPI, norepinephrine. *$P<0.05$ *vs* baseline and control; $^{\#}P<0.05$ *vs* baseline and intervention. ANOVA on ranks for repeated measurements, all-pair-wise comparison procedure, Student–Newman–Keuls

Parameter	Baseline	Intervention	Control
Heart rate (min^{-1})	98 (19) [100]	95 (17) [94]	94 (19) [95]
MAP (mm Hg)	66 (9) [66]	92 (9)* [91]	68 (12) [66]
CVP (mm Hg)	12 (5) [12]	13 (7) [13]	12 (5) [13]
CO(Vigileo®) (litre min^{-1})	5.8 (2.0) [5.8]	7.6 (2.6) [7.6]*	5.3 (2.0) [4.8]$^{\#}$
CO(TPID) (litre min^{-1})	6.6 (1.8) [7.0]	6.8 (1.9) [6.8]	6.6 (1.8) [6.8]
SVR (dyn s cm^{-5})	551 (106) [535]	746 (91) [759]*	566 (138) [525]
GEF (%)	22 (6) [22]	22 (6) [23]	23 (7) [22]
EVLWI (ml kg^{-1})	7.3 (2.7) [7.0]	7.5 (3.0) [7.0]	7.4 (3.0) [7.0]
NEPI ($\mu g\ kg^{-1}\ min^{-1}$)	0.05 (0.04) [0.04]	0.11 (0.09) [0.09]*	0.05 (0.05) [0.05]

correlated. Thus, this new system is not as reliable as transpulmonary thermodilution in septic patients.

In this study, we used the transpulmonary thermodilution technique as the reference technique, which has been extensively compared with the clinical 'gold standard', i.e. pulmonary artery thermodilution. Previous experimental[5–7] and clinical studies[4 8] reported a good correlation between pulmonary artery and transpulmonary thermodilution for the measurement of CO. However, transpulmonary thermodilution CO is most often found to be higher than the corresponding pulmonary artery CO, and this is considered to be caused by the cold-induced reduction in the heart rate[17] and the loss of indicator.[18] Furthermore, Bock and colleagues[7] showed that the early recirculation of the cold is responsible for the broader thermodilution

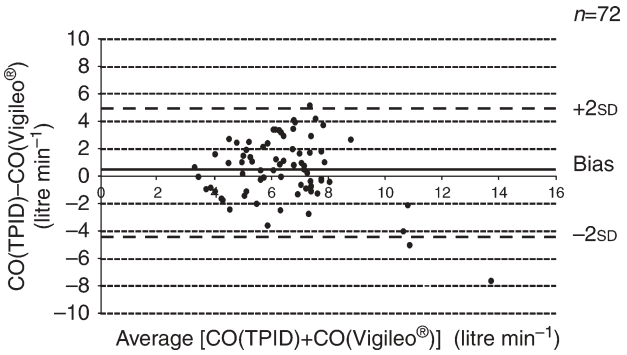

Fig 2 Bland–Altman plot for all CO measurements (n=72).

curve in the aorta, thus leading to about 3 (4)% higher values for CO.

Most recently, a less invasive device for continuous CO measurement (CCO), based on peripheral arterial pulse contour analysis (Vigileo®), has been introduced into clinical practice. This system does not require thermal or dye dilution, but rather bases its calculations on arterial waveform characteristics in conjunction with patient demographic data without requiring calibration against another method. The system has been assessed and found to be robust and accurate over a wide range of CO and clinical conditions.[19] However, only few clinical data have been available so far on the accuracy of this system, especially in critically ill patients. Manecke and colleagues[10] used data from 11 cardio-thoracic surgical patients with an unequal number of measurements per patient taken immediately after surgery and compared 65 pairs of CO measurements. The mean bias between pulse contour CO and intermittent pulmonary artery CO (ICO) was 0.04 (0.99) litre min^{-1}. However, because an unequal number of measurements per patient was used, which is a statistically inappropriate

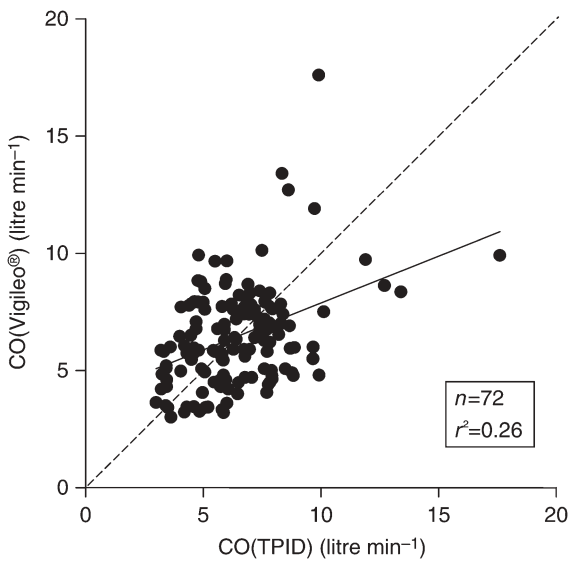

Fig 1 Linear regression between CO(Vigileo®) and CO(TPID) for all measurements (n=72). The dashed line indicates identity.

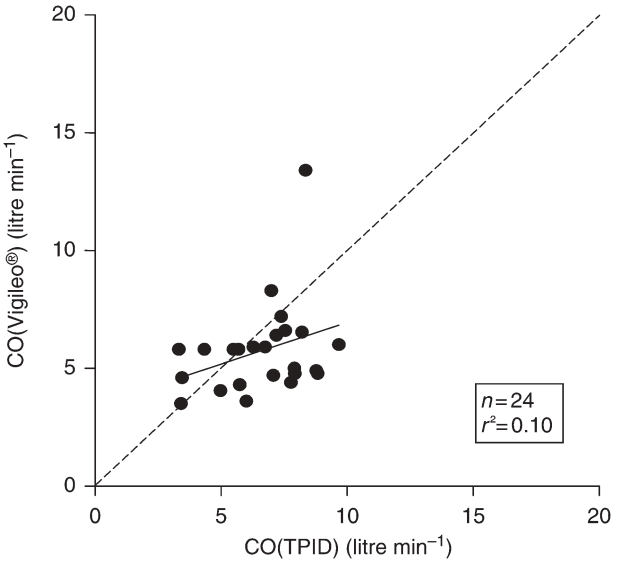

Fig 3 Linear regression between CO(Vigileo®) and CO(TPID) at baseline (n=24). The dashed line indicates identity.

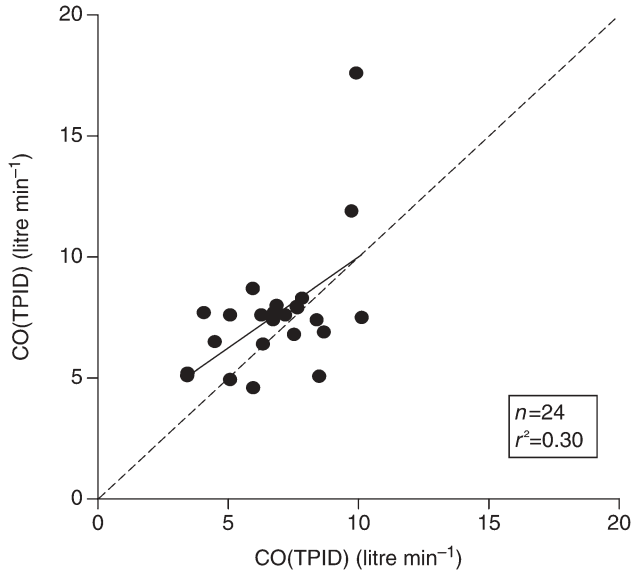

Fig 4 Linear regression between CO(Vigileo®) and CO(TPID) during increased MAP (n=24). The dashed line indicates identity.

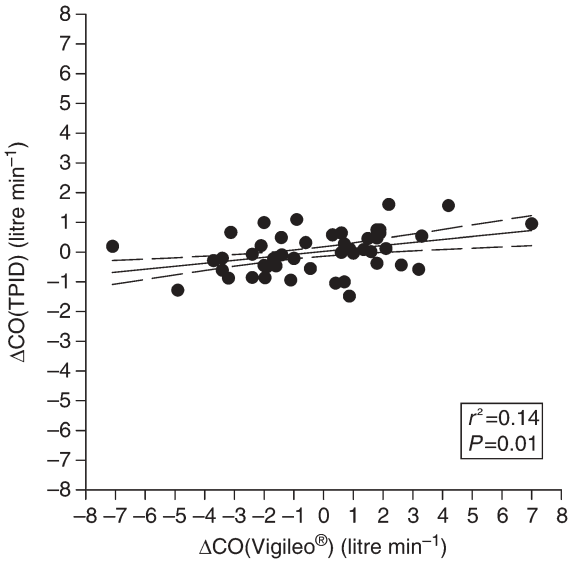

Fig 6 Linear regression between ΔCO(Vigileo®) and ΔCO(TPID) (n=72).

procedure, these results may be questionable. McGee and colleagues[20] analysed 252 data points in 36 patients [mean age 64 (14) yr, 72.2% male] and reported that pulse contour CO trends correlated with ICO in 98% of all data points collected.

In patients after off-pump coronary artery bypass grafting,[21] CO was described to be reliably monitored by pulse contour analysis (Vigileo®) during stable haemodynamic conditions. However, the Vigileo® system showed a tendency to overestimate rapid decreases and increases in CO when compared with the PiCCO®plus system. When compared with ICO bolus measurement, mean bias (2 SDS) (limits of agreement) was −0.13 (1.08) litre min^{-1} for Vigileo®-ICO. Costa and colleagues[22] studied 14 patients after liver transplantation, in whom ICO and Vigileo® CO measurements were collected after ICU admission and every 8 h until the 48th postoperative hour. The mean difference between Vigileo®–continuous CI [bias (2 SDS)] was 0.90 (1.49) litre min^{-1} m^{-2}, and 95% confidence intervals were −0.59 to 2.39 litre min^{-1} m^{-2}.

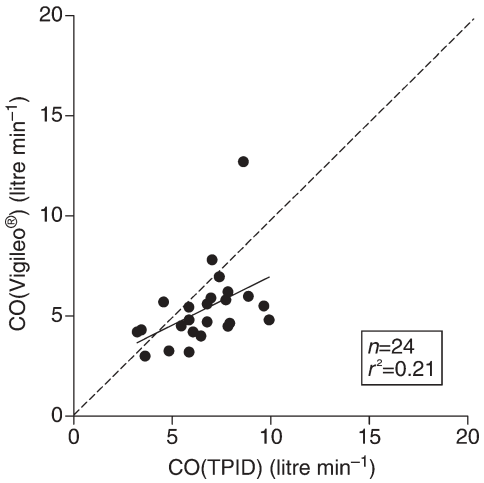

Fig 5 Linear regression between CO(Vigileo®) and CO(TPID) after returning to baseline MAP (n=24). The dashed line indicates identity.

More recently, the accuracy of this uncalibrated arterial waveform analysis system has been questioned. Sander and colleagues[11] studied CO measurements by a PAC, transpulmonary thermodilution and Vigileo® monitor in 30 cardiac surgical patients at four different time points. These authors reported r=0.53 between CO(Vigileo®) and ICO and r=0.84 between CO(Vigileo®) and CO(TPID). Mean bias and limits of agreement were 0.6 and −2.2 to 3.4 litre min^{-1} for ICO *vs* CO(Vigileo®) and −0.1 and −1.8 to 1.6 litre min^{-1} for CO(ICO) *vs* CO(TPID). As we found, their conclusion was that the Vigileo® system underestimated ICO to a clinically relevant extent and that the wide range of limits of agreement requires further evaluation. In six cardiac surgical patients, Opdam and colleagues[12] reported that ICO had better correlation with the Vigileo® values (r^2=0.27, bias=−0.006, 95% limits of agreement −1.2, 1.19 litre min^{-1}) than did those obtained with the CCO by PAC (r^2=0.056, bias=0.24, 95% limits of agreement −0.74, 1.22 litre min^{-1}). CO values measured during atrial pacing showed the highest correlation (r^2=0.38, bias=−0.02, 95% limits of agreement −0.53, 0.57 litre min^{-1}). These authors concluded that further evaluation is required before this device can be recommended for use in the clinical setting.

Besides a defined study protocol and the same number of measurements per patient, the strength of our study is the stability of CO measurements. In our study, the stability of measurements was in the range of that reported by other studies, i.e. CVs on average were clearly below 10% for both techniques at each of the three time points.

However, our study has several limitations. First, we did not use a further technique for comparison, which may independently allow assessment of CO under the study conditions. However, we used transpulmonary thermodilution and not PiCCO®-derived pulse contour, which itself may be influenced by the intervention of changing MAP. Furthermore, we used the Vigileo® system in a manner different from the manufacturer's recommendation. In detail, we used a central (i.e. femoral artery) and not a peripheral (A. radialis) pressure curve and this may have considerably influenced our findings. However, since there was no clinical need for an additional arterial catheter, we could not justify the placement of a second (i.e. peripheral) arterial cannula and we, therefore, used the curve already obtained from the femoral artery. Notably, a new software version for calculating CO is now available and, by applying this version, the results may be different from those in our study. In comparison with previous studies,[10 12] we enrolled a higher number of patients. We analysed a relatively high number of CO measurements with an equal number of measurements per patient under varying haemodynamic conditions while using a backward control. Nevertheless, the number of patients we enrolled (n=24) is still very limited. Finally, our results are limited insofar as we studied septic patients with reduced peripheral resistance, and these findings are probably not generally applicable to all patients.

In conclusion, pulse contour analysis-derived CO measurements (Vigileo® system) were not correlated with transpulmonary thermodilution and this new system is not as reliable as transpulmonary thermodilution in patients with septic shock.

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