ORIGINAL ARTICLE

Evaluation of Electrical Cardiometry to Assess Fluid Responsiveness in Patients with Acute Circulatory Failure: A Comparative Study with Transthoracic Echocardiography

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Abstract

Aim: Acute circulatory failure is commonly encountered in critically ill patients, that requires fluid administration as the first line of treatment. However, only 50% of patients are fluid-responsive. Identification of fluid responders is essential to avoid the harmful effects of overzealous fluid therapy. Electrical cardiometry (EC) is a non-invasive bedside tool and has proven to be as good as transthoracic echocardiography (TTE) to track changes in cardiac output. We aimed to look for an agreement between EC and TTE for tracking changes in cardiac output in adult patients with acute circulatory failure before and after the passive leg-raising maneuver.

Materials and methods: Prospective comparative study, conducted at a Tertiary Care Teaching Hospital.

Results: We recruited 125 patients with acute circulatory failure and found 42.4% (53 out of 125) to be fluid-responsive. The Bland–Altman plot analysis showed a mean difference of 2.08 L/min between EC and TTE, with a precision of 3.8 L/min. The limits of agreement (defined as bias ± 1.96SD), were −1.7 L/min and 5.8 L/min, respectively. The percentage of error between EC and TTE was 56% with acceptable limits of 30%.

Conclusion: The percentage error beyond the acceptable limit suggests the non-interchangeability of the two techniques. More studies with larger sample sizes are required to establish the interchangeability of EC with TTE for tracking changes in cardiac output in critically ill patients with acute circulatory failure.

Keywords: Acute circulatory failure, Bland–Altman plot, Electrical cardiometry, Fluid responsiveness, Transthoracic echocardiography.

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Highlights

- Sepsis with acute circulatory failure requiring ICU admission is common.
- Assessment of fluid responsiveness before administering fluids avoids harm due to overzealous fluid therapy.
- Electrical cardiometry (EC) is a non-invasive tool to assess fluid responsiveness.
- Future studies with larger sample sizes are needed to establish the interchangeability of EC with transthoracic echocardiography (TTE).

Introduction

Sepsis causing acute circulatory failure requiring admission to critical care units is not uncommon and fluid resuscitation is pivotal to achieve optimum organ perfusion and oxygen delivery.[1] The Aggressive fluid resuscitation during the early course of therapy improves cardiac output in 50% of cases and leads to undesired fluid overload and associated poorer outcomes in the remaining 50%, mandating an accurate assessment of fluid responsiveness on a real-time basis.[2–4] The various hemodynamic parameters to identify fluid responders include central venous pressure (CVP), stroke volume variation (SVV), pulse pressure variation (PPV), perfusion index (PI), and positive pressure ventilation-induced changes in superior and inferior vena cava diameter.[5–9] All these parameters have their merits and demerits. The PLR maneuver-induced change in cardiac output, with peak hemodynamic effect around 60 seconds; is a reliable predictor of fluid responsiveness in mechanically ventilated patients and patients with spontaneous breathing efforts.[10–12] The thermodilution technique with a pulmonary artery catheter (PAC), being the gold standard technique is no longer used barring its invasive nature, associated complications, and poorer outcome-benefit ratio. The search for less invasive techniques led to the development of devices based on transpulmonary thermodilution, pulse contour analysis, and bioreactance principle.[13] The echocardiography techniques offer an attractive alternative to PAC thermodilution for cardiac output measurement. Although transoesophageal echocardiography (TEE) is less invasive as compared to PAC, it is operator-dependent, requires patients to be unconscious, and its use is contraindicated

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in patients with esophageal diseases and bleeding disorders.[13] Transthoracic echocardiography is also operator-dependent but is simpler and can be performed even in conscious patients in contrast to esophageal echocardiography and multiple studies have validated it as an optimal tool to assess fluid responsiveness.[11–13] A recent meta-analysis also concludes that echocardiography is not significantly different from pulmonary thermodilution and now established as the gold standard for cardiac output measurement in current ICU practice.[14] The EC (OSYPKA Medical, Germany) device is a newer addition to the queue of non-invasive continuous cardiac output monitoring tools and works on the bioimpedance principle with advanced filtering techniques (to filter noise). It measures the change in electrical conductivity across the thorax based on the changing orientation of red blood cells during cardiac systole and diastole and calculates stroke volume based on a predefined algorithm.[15] The literature suggests that EC is a bedside, easy, portable, and operator-independent alternative to tracking a censorious patient's cardiac output.[15,16] Studies have also found EC to accurately measure cardiac output in patients undergoing anesthesia and surgery and even in pediatric cardiac patients.[17–20] Electrical cardiometry is comparable to echocardiography for cardiac output assessment in adolescents, pregnant, and neonates.[19–23] In a recent study, a change in cardiac output (≥12.5%), measured by EC, predicted fluid responsiveness in septic patients after fluid administration.[23]

Electrical cardiometry has also shown good agreement with carotid Doppler for predicting fluid responsiveness after PLR in patients with septic shock.[24]

In addition, EC has proven as good as echocardiography concerning mortality prediction in septic patients.[25] Although TTE is simple, easy to use, and portable, it requires experience and a good imaging window to measure cardiac output accurately. Besides, the patient's movement may alter the imaging window and require a longer time to measure the cardiac output, especially with a novice. Given the scarce literature on the use of EC in adult septic patients and the limitations posed by TTE, we tried to estimate the suitability of EC against TTE for continuous cardiac output measurement. We primarily aimed to gauge consensus between EC and TTE for cardiac output pre- and post-PLR maneuver. Secondarily, we aimed to compare hemodynamic, and ventilatory parameters before and after the PLR maneuver and to compare the dose and type of vasoactive agents between fluid responders and non-responders.

Materials and Methods

All septic patients admitted to the ICU of a Tertiary Care Teaching Hospital were screened for eligibility and 125 patients were taken in the study. All institutional ethical clearances (IECPG-675/23.12.2020) were taken before initiating the study and the same was registered. Informed written consent was acquired from the legally authorized representatives of all the participants. The trial was registered at the Clinical Trial Registry of India (CTRI/2021/03/031921) with the status being regularly updated.

All patients that were included in the study had features suggestive of acute circulatory failure (including SBP at least equal to or less than 90 mm Hg or drop of more than 50 mm Hg in patients with hypertension, the requirement of vasoactive support, heart rate >100 beats/minute, urine output less than 0.5 mL/kg/hour for two consecutive hours and delayed capillary refill time (>3 seconds) or evidence of skin mottling), requiring mechanical ventilation and, invasive arterial blood pressure monitoring.

Patients with any of the following were excluded from the study: Ages less than 18 years and more than 65 years, pregnant patients, patients with arrhythmias, known cases of heart failure on treatment, patients with valvular heart disease, patients with lower limb amputation, contraindications to performing PLR (lower limb DVT, limb/pelvic fracture, abdominal surgeries, intra-abdominal hypertension, intracranial hypertension), pulmonary hypertension (PH) with right ventricle dysfunction, presence of wound/dressing in the neck, difficulty in getting an adequate echo window [morbidly obese (BMI≥40 kg/m^2)] and chest wall trauma, were precluded from the study.

In a previous study, authors noted a mean difference (bias) of 0.15 L min^{-1} with a standard deviation (precision) of 0.53 L min^{-1} between cardiac output computed by EC (CO_{EC}) and TTE (CO_{TTE}).[23] The limits of agreement (±2 SD) at the upper and lower end were +1.21 and −0.91 L min^{-1}, respectively. At a set alpha error of 0.05 and to achieve 90% power, 125 patients were recruited to achieve a maximum allowable difference of 1.42 L min^{-1} in cardiac output measured by EC and TTE.

Study Protocol

Patients were included in the study within 6–12 hours of the onset of acute circulatory failure (either at ICU admission or during the ICU stay). The demographic details, comorbid illness, APACHE 2 score, vasoactive drugs (type and dosage), the time between the onset of circulatory failure and enrolment in the study (hours), cumulative fluid balance (in milliliters) at the time of enrolment, and parameters from arterial blood (PaO_2, $PaCO_2$, P/F ratio, lactate) were recorded for all the patients.

Adequate sedation was provided as per ICU protocol and volume control mode (tidal volume-6–8 mL/kg) was used to maintain constant minute ventilation in all the patients before initiating the study procedure.

The cardiac output was measured using two different techniques: Transthoracic echocardiography (using 1–5 MHz, phased array probe, Sonosite) and EC hemodynamic monitor (ICON® OSYPKA Medical, Germany). For TTE, LVOT VTI, LVOT diameter, and heart rate were combined to compute cardiac output, while EC (which works on bioimpedance principle) measures impedance due to the changing orientation of red blood cells in the aorta during cardiac systole and diastole and computes cardiac output utilizing Berstein OSYPKA equation.[26]

The patients were kept in a semi-recumbent position (45 degrees), and electrodes were applied to the neck and the thorax. The cardiac output was computed by TTE and simultaneously another person captured a video on the EC for 10 cardiac cycles. The cardiac output computed by TTE and the average of 10 cardiac cycles on the EC monitor served as the baseline value (baseline 1). subsequently, the standard PLR maneuver was performed by simultaneously bringing down the trunk and raising the lower limbs (with no alteration at the hip angle). The cardiac output with TTE was measured at one minute following the PLR maneuver, and simultaneously recording from the EC monitor was done as in the previous step. After PLR, the patient was brought back to the baseline position. The cardiac output by TTE was measured, and recording from EC (same way as during baseline 1 and PLR step) monitor was done again after 2 minutes of returning the patient to the original position (baseline 2). The baseline 2 measurements were done to ensure that the changes during PLR were only because of the PLR maneuver.

The hemodynamic variables including cardiac output, heart rate, oxygen saturation, invasive blood pressure, CVP, and ventilatory parameters (peak and mean pressure) were also recorded at various steps during the study procedure. The change in cardiac output>15% (with TTE) was considered a predictor of fluid responsiveness. Giving fluid to fluid responders or escalating the dose of vasopressor in non-responders was left at the discretion of the Intensivist on call.

Statistical Analysis

Mean standard deviation (SD) and frequency (%) were used to represent continuous and categorical data, respectively. The normality of quantitative data was checked by the Kolmogorov–Smirnov test of normality. The hemodynamic and ventilatory parameters were compared using the student's *t*-test while, and the categorical variables were scrutinized using the chi-square test. The Bland and Altman were applied to determine bias (ΔCO, mean CO_{EC} −mean CO_{Echo}), mean CO (CO_{EC} + CO_{Echo})/2), limits of agreement (ΔCO ± 1.96 SD), and the error percentage (1.96 SD/mean CO). The clinically acceptable limit of error between the two techniques was ±30%, based on a previous study.[26] The cardiac output with TTE and EC was weighed up using linear regression analysis, and Pearson's correlation coefficient was calculated. The statistical software, STATA (version 17) was used to analyze data, and a *p*-value with a cut-off of less than 0.05 was taken to differentiate statistically significant values from non-significant ones.

Results

A total of 145 patients were assessed for eligibility, out of which 20 were excluded for various reasons (Fig. 1).

Of 125 patients, 53 (42.4%) were found to be fluid responders, and 72 (58.6%) were fluid non-responders. The demographic, hemodynamic, and ventilatory parameters of all patients are shown in Table 1.

The groups (fluid responders and non-responders) were comparable for all parameters except mean MAP, which was slightly less in fluid responders (82 ± 10 mm Hg vs 84 ± 12 mm Hg, p = 0.04). The mean PEEP was slightly higher in fluid responders than non-responders (7.1 ± 1.2 cm H_2O vs 6.5 ± 1.5 cm H_2O, p = 0.04). The fluid responder group had fewer males and females than the fluid non-responder group. The differences in MAP, PEEP, and unequal distribution of males and females between the two groups can be attributed to the non-randomized nature of the study.

The mean difference (bias) and precision between CO_{EC} and CO_{TTE} were 2.08 L/min and 3.8 L/min, respectively. The limits of agreement at the lower and upper ends were −1.7 L/min and 5.8 L/min,

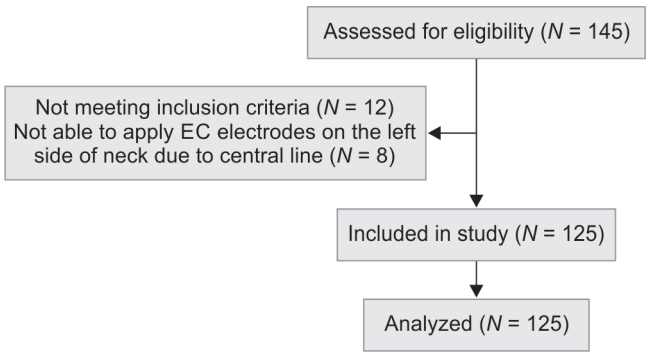


Fig. 1: CONSORT diagram

Table 1: Patient characteristics, hemodynamic and ventilatory parameters

Patient characteristics/parameters	*All patients (N = 125)*	*Fluid responders (N = 53)*	*Fluid non-responders (N = 72)*	**p-value*
Age (years)	39.9 ± 15.2	38.3 ± 15.9	41.0 ± 14.7	0.24
Males (*n*/%)	71 (57%)	33 (63%)	38 (52%)	0.20
Females (*n*/%)	54 (43%)	19 (37%)	35 (48%)	
Weight (kg)	65.3 ± 10.8	64 ± 10.7	66.4 ± 10.7	0.28
Height (cm)	164 ± 5.8	163 ± 6.7	164 ± 5.2	0.78
BMI (kg/m^2)	24.3 ± 3.3	24 ± 3.0	24.9 ± 3.4	0.10
APACHE 2 score	14.8 ± 4.7	14.7 ± 4.8	14.8 ± 4.6	0.84
Time gap (hours)	27.2 ± 20.9	27.9 ± 20.5	26.6 ± 21.3	0.50
Cumulative fluid balance (milliliters)	1915 ± 1890	1933 ± 1909	1902 ± 1889	0.77
PF ratio	262 ± 110	263 ± 107	261 ± 112	0.92
Lactate (mmol/L)	3.1 ± 3.8	3.2 ± 3.8	3.0 ± 3.9	0.70
Tidal volume (mL)	387 ± 55	383 ± 55	392 ± 55	0.27
PEEP (cm H_2O)	6.7 ± 1.8	7.1 ± 2.1	6.5 ± 1.5	0.04
Mean CVP (cm H_2O)	13.7 ± 2.0	13.3 ± 1.9	13.9 ± 2.0	0.09
Mean HR (beats/min)	107 ± 24	105 ± 21	108 ± 26	0.59
Mean SBP (mm Hg)	122 ± 18	122 ± 16	122 ± 19	0.88
Mean DBP (mm Hg)	66 ± 11	64 ±10	67 ± 11	0.38
Mean MAP (mm Hg)	84 ± 12	82 ± 10	84 ± 12	0.04
Mean PIP (cm H_2O)	27.9 ± 7.9	28.3 ± 6.7	27.6 ± 8.7	0.52
Mean P_{aw} (cm H_2O)	13.6 ± 3.5	13.9 ± 3.0	13.4 ± 3.8	0.25

Numerical values are shown as mean ± SD and number (%), *for comparison between fluid responders and non-responders. The *p*-value cut-off of <0.05 represents statistical significance

respectively. The SD and mean cardiac output (mean CO) were 1.9 L/min and 6.7 L/min, respectively. The percentage error between the methods was 56% [percentage error = (1.96*SD) %mean CO] (Fig. 2). The error between cardiac output measurement by EC and that by TTE is higher than acceptable limits (±30%).

The cardiac output measurement by EC(CO_{EC}) and TTE (CO_{TTE}) exhibited a moderate correlation ($r = 0.526$, 0.538, and 0.527 at different time points, $p = 0.001$) (Table 2 and Fig. 3).

The hemodynamic and ventilatory parameters before and after PLR are shown in Table 3. There was an increase in all parameters before and after the PLR. Although no patient touching was involved during the PLR- maneuver, the movement of the bed itself during the study period might have caused some degree of sympathetic stimulation causing a rise in heart rate and blood pressure. After PLR, the decrease in cerebral venous return compared to the upright position may explain the rise in CVP. For ventilatory parameters, pushing up of the diaphragm by abdominal contents after PLR probably led to a rise in Paw and PIP.

Both groups' changes at different time points are comparable except for peak inspiratory pressure ($p = 0.02$, not of clinical significance) (Table 4).

The doses of different inotropes/vasopressors were comparable between fluid responders and non-responders (Table 5).

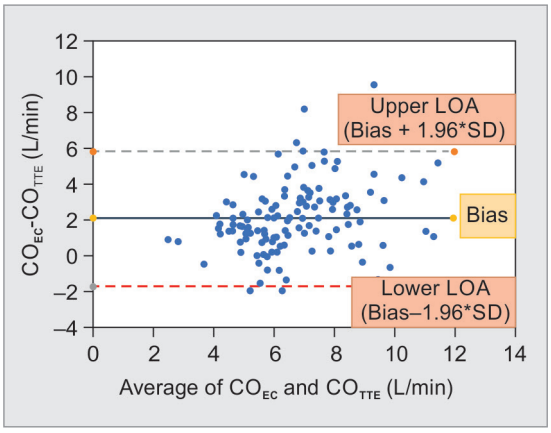


Fig. 2: Bland–Altman plot

Table 2: Cardiac output by EC and TTE at different time points for all patients

Time points	*COEC (N = 125)*	*COTTE (N = 125)*	*Correlation (r)*	*p-value*
B1	7.4 ± 2.1	5.4 ± 1.8	0.52 (0.38–0.64)	0.01
PLR	8.5 ± 2.3	6.2 ± 1.9	0.53 (0.40–0.65)	0.01
B2	7.4 ± 2.1	5.4 ± 1.8	0.52 (0.37–0.63)	0.01

The numerical values are shown as mean ± SD and mean (95% CI). The *p*-value with a cut-off of <0.05 represents statistical significance

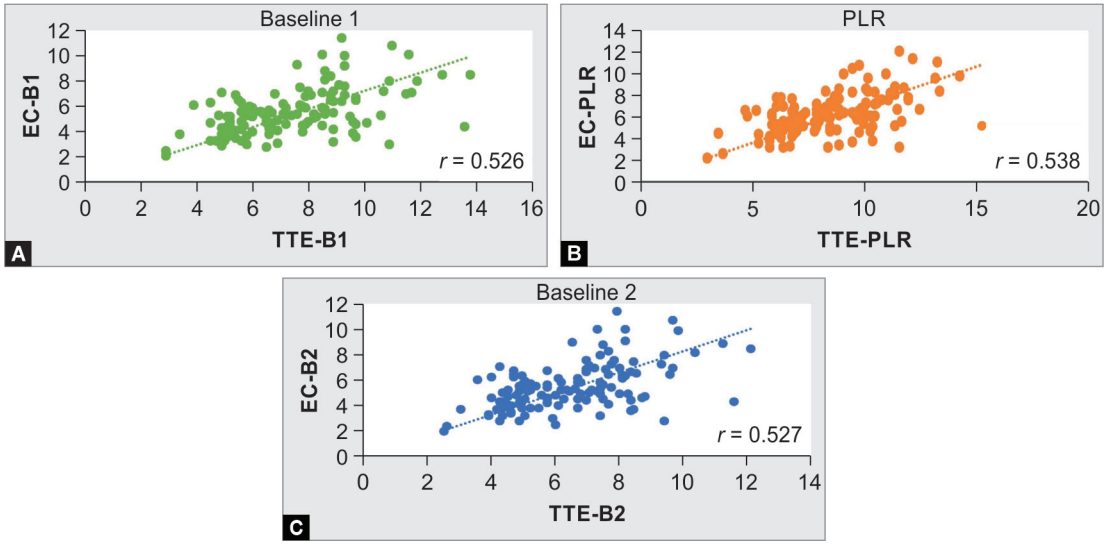
Discussion

The EC hemodynamic monitor (ICON® OSYPKA Medical, Germany) is a simple, non-invasive, and portable tool for continuous cardiac output monitoring and remains poorly utilized in adult septic patients. It is based on the bioimpedance principle and uses two pairs of surface electrodes with one pair applied at the lower part of the neck on the left side and; the other at the level of the xiphoid process along the left mid-axillary line; the electrodes of each pair apart by 5 cm. A small amplitude (2 milliamperes) alternating

Table 3: The hemodynamic and ventilatory parameters before and after PLR

Parameters	*Baseline 1 (N = 125)*	*PLR (N = 125)*	*p-value*
HR (beats/min)	106 ± 24	107 ± 24	0.001
CVP (cm H_2O)	12.5 ± 2.0	15.2 ± 2.2	0.001
SBP (mm Hg)	120 ± 18	124 ± 18	0.001
DBP (mm Hg)	65 ± 11	66 ± 11	0.001
MAP (mm Hg)	83 ± 11	84 ± 12	0.001
PIP (cm H_2O)	27.4 ± 8.0	27.7 ± 8.0	0.001
P_{aw} (cm H_2O)	13 ± 3.5	13.4 ± 3.5	0.001

Numerical values are shown as mean ± SD. The *p*-value with a cut-off of <0.05 represents statistical significance



Figs 3A to C: Correlation between cardiac output at different time points

Table 4: Changes in hemodynamic and ventilatory parameters from baseline to post-PLR in responders and non-responder groups

Parameters	*Fluid responders (N = 53) Mean ± SD, 95% CI*	*Fluid non-responders (N = 72) Mean ± SD, 95% CI*	*p-value*
ΔCVP	2.3 ± 1.0 (2.57–2.03)	2.3 ± 1.2 (2.57–2.03)	0.74
ΔHR	2.0 ± 2.0 (2.5–1.5)	2.0 ± 2.0 (2.4–1.6)	0.79
ΔSBP	4.2 ± 4.1 (5.3–3.1)	4.4 ± 6.0 (5.7–3.1)	0.88
ΔDBP	3.0 ± 3.1 (3.8–2.2)	2.7 ± 2.3 (3.2–2.2)	0.61
ΔMAP	3.0 ± 3.2 (3.8–2.2)	3.0 ± 3.0 (3.6–2.4)	0.89
ΔPIP	1.5 ± 1.4 (1.8–1.2)	0.9 ± 1.0 (1.1–0.7)	0.02
ΔP_{aw}	0.7 ± 0.9 (0.9–0.5)	0.6 ± 0.6 (0.7–0.5)	0.36

Δ = Difference between PLR and baseline 1 value. The *p*-value with a cut-off of <0.05 represents statistical significance

Table 5: Type and dose of vasopressors/inotropes

Inotrope/ vasopressor	*Fluid responders (N = 52)*	*Fluid non-responders (N = 73)*	*p-value*
Noradrenaline (μg/min)	15 ± 11.7	15.3 ± 11.2	0.85
Vasopressin (units/hour)	0.6 ± 0.9	0.5 ± 0.9	0.57
Adrenaline (μg/min)	0.7 ± 5.4	1.7 ± 5.8	0.06

Numerical values are shown as mean ± SD. The *p*-value with a cut-off <0.05 represents statistical significance

current travels from thorax electrodes toward neck electrodes. The changing orientation of red blood cells during the cardiac systole and diastole alters the conductivity of current and gives input to the EC device which in turn calculates Stroke volume and cardiac output using the Bernstein-OSYPKA equation.[27]

The present study compared EC with TTE for assessing fluid responsiveness in septic patients using a passive leg-raising maneuver. Of 125 patients, 53 (42.4%) were fluid responders based on a cut-off of 15% change in cardiac output (measured by TTE) pre- and post-PLR.

In the present study, the EC and TTE showed a mean difference of 2.08 L/min, concerning cardiac output with limits of agreement as –1.7 L/min and 5.8 L/min. The percentage error between the methods was 56% (the predefined acceptable limit was ±30%), with a moderate correlation ($r = 0.5 – 0.6$) between CO_{EC} and CO_{TTE} at different time points.

As per the meta-analysis by Critchley LA and Critchley JA, the limits of the agreement should be ±30% for a technique of cardiac output measurement to substitute the gold standard or nearly gold standard technique of measuring cardiac output.[28] In our study, the error between cardiac output measurement by EC and that by TTE is higher than the acceptable range, limiting the interchangeability of these two devices.

Effat H et al. compared EC with carotid Doppler for fluid responsiveness (after PLR and Fluid challenge) in 44 critically ill adult septic patients. Unlike ours, they found good agreement (kappa > 0.6 and $p < 0.01$) between EC and TTE for percentage change in cardiac output after PLR and fluid challenge. Only half of the patients in their study were in septic shock requiring vasopressors (47.7%), and only 45.5% were on positive pressure ventilation. In our study, on the other hand, all patients ($n = 125$) were in septic shock, requiring vasopressors, and were on mechanical ventilation. The differences in inclusion criteria in both studies limit comparison concerning the interchangeability of EC with TTE.[24]

Elsayed Afandy M et al. also analyzed 90 adult septic shock patients and randomized them into EC, TTE, and early goal-directed therapy using CVP values. Patients received fluid and vasopressor therapy according to the monitoring technique to which they were randomized. The authors primarily aimed to compare mortality at 30 days in three groups and found both techniques comparable for mortality. In addition, the dosage of vasopressors, time of liberation from vasopressors, days of mechanical ventilation, duration of ICU stays, and hospital stay, either before or after treatment were similar in all three groups, indicating that both the monitoring techniques were probably interchangeable. The authors, however, did not use all three monitoring parameters in all the patients and did not analyze them statistically for interchangeability.[25]

Kusumastuti NP and Osaki M demonstrated a strong correlation between cardiac output by EC and TTE in 30 patients (<18 years) post-cardiac surgery ($r = 0.831$, $p < 0.001$). Also, the authors noted a low bias (0.08 L/min/m^2) and a low percentage of error (13.19%) between the two techniques, when analyzed using the Bland–Altman plot. The low percentage of error and interchangeability of the two devices need reconsideration that only 50% of patients required hemodynamic support (in ours, all patients were on vasopressors/inotropes). Also, the patients were much younger (excluded from our study) and postoperative surgical cohort against medical cohorts in our study.[20]

We found an increase in hemodynamic parameters (HR, CVP, SBP, DBP, MAP) from baseline to post-PLR ($p < 0.001$). However, the increase was statistically comparable in fluid responders and non-responders. Similarly, the various authors observed an increase in hemodynamic parameters before and after PLR in their studies.[24,29–33]

In line with our study, Effat H et al. also noted an increase in CVP and MAP from baseline to post-PLR in 50 patients with septic shock.[24] The authors reported a statistical difference between fluid responders and fluid non-responders (conflicting with ours). However, this difference seems to be of little or no clinical importance.

Caille V et al. performed PLR and studied its hemodynamic effects in 40 patients with shock and receiving controlled mechanical ventilation. Similar to ours, the authors noted an increase in SBP, DBP, and MAP after PLR compared to baseline. Moreover, the increase was more appreciable in fluid responders than non-responders ($p < 0.05$) (unlike ours), but this change does not seem to have a bearing on clinical outcomes.[31]

Dong ZZ et al. studied 32 patients with septic shock and controlled mechanical ventilation for fluid responsiveness by performing PLR maneuver and found an increase in SBP, DBP, and MAP after PLR, compared to the baseline value in both groups (matching ours). However, only the fluid responder's group showed statistically significant change ($p < 0.05$) but appears to have negligible clinical significance.[32]

Compared to previous studies, the absence of difference between fluid responders and non-responders in the present study [concerning hemodynamic parameters (before and after PLR)] could be due to the timing of collecting hemodynamic parameters owing to the focus on collecting cardiac output values.[24,31,32] In some patients, it was collected at the time of peak effect of PLR

(around 60 seconds, where the increase in CVP and blood pressure is expected to be maximum) and delayed in others; this could have neutralized the appreciable difference between fluid responders and non-responders.

There was an increase in peak and mean airway pressure from baseline to post-PLR in both fluid responders and non-responder groups, and the increase was statistically significant for peak inspiratory pressure ($p = 0.02$). However, the actual difference was 0.6 cm H_2O and seems to have no clinical significance. However, we could not find a study concerning this outcome.

There was no difference concerning doses of noradrenaline, adrenaline, and vasopressin between the fluid-responder and non-responder groups. In line with our study, Effat H et al. and Elsayed Afandy M et al. also used EC in septic patients, but they did not compare fluid responders and non-responders concerning doses and types of inotropes/vasopressors.[24,25]

The present study had certain strengths and limitations. To mention strengths, firstly, we tried to investigate the suitability of EC as a simple, continuous, and operator-independent technique, against gold standard technique, i.e., TTE (which is operator-dependent and provides a non-continuous measurement of cardiac output). Secondly, we choose a population that requires fluid administration at admission and various points during ICU stay. Thirdly, the author performing TTE had already performed at least 50 such examinations before the start of this study, which minimizes errors in echocardiographic measurements. In addition, we performed standard passive leg-raising maneuvers which include no touching the patient hence avoiding any false rise in cardiac output that could occur because of the patient's stimulation.

It would be worth mentioning the limitations of our study. The significant deviation of EC from TTE for the percentage of error could be due to the following reasons. First, although, a paramedical staff was assigned to collect EC cardiac output, hemodynamic, and ventilatory parameters at the peak of PLR, the delay in collecting hemodynamic and ventilatory parameters in a few patients cannot be ruled out. Second, the impact of the thickness of subcutaneous tissue on electrical impedance in obese patients and females with thick infra-axillary fat pads needs consideration, as it may affect cardiac output data. Third, we did not record the temperature of our study participants which can affect conductivity across the electrodes of EC and hence the cardiac output values.[34,35]

Fourth, PH and increased right ventricular (RV) pressure decrease venous return during PLR and may lead to a false negative PLR-response.[36] Although we excluded patients with PH and RV dysfunction, we did not assess pulmonary artery pressure in at-risk patients, (e.g., chronic obstructive pulmonary disease and hypoxemia).

Fourth, the patients were included at different time points during ICU stay with varying positive cumulative fluid balance. Whether cumulative positive fluid balance affects fluid responsiveness or not, needs evaluation in future trials. Last but not least, being a single-center study, the generalizability of results is limited.

Conclusion

In the present study, EC and TTE showed poor agreement for the assessment of fluid responsiveness in adult patients with acute circulatory failure. Further studies with larger sample sizes are required to establish the interchangeability of these techniques for assessing fluid responsiveness.

Author Contributions

SS: Methodology; data collection; formal analysis; writing-original draft; RR: Supervision; Writing-review and editing; AT: Supervision; Writing- review and editing; VR: Conceptualization; methodology; Supervision; Writing-review and editing

Ethical Approval

Institute ethics committee (IECPG-675/23.12.2020), All India Institute of Medical Sciences, New Delhi, India.

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