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Evaluation of end-tidal carbon dioxide as a marker of fluid responsiveness in mechanically ventilated patients with shock: a prospective study in a tertiary care center in India

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Abstract

Shock management in intensive care unit (ICU) patients requires accurate assessment of fluid responsiveness to optimize outcomes. Dynamic indices, such as passive leg raising (PLR), are often underutilized due to invasive or complex techniques. Our aim was to evaluate change in end-tidal carbon dioxide (EtCO₂) during PLR as a non-invasive dynamic index of fluid responsiveness in mechanically ventilated ICU patients with shock. This was a prospective, observational cohort study conducted in a respiratory ICU at a tertiary care center in New Delhi, India. The study recruited adult patients on mechanical ventilation with shock between November 2022 and April 2024. After screening 340 ICU admissions during the recruitment period for inclusions and exclusions, a total of 90 adult patients on mechanical ventilation with shock were enrolled in the study. Measurements of EtCO₂ *via* mainstream capnography and cardiac output (CO) with transthoracic echocardiography (TTE) were done pre- and post-PLR. Fluid responsiveness was defined as a 10% increase in CO measured *via* TTE following PLR. Simultaneously, EtCO₂ was measured, with a 5% increase considered predictive of fluid responsiveness. Sensitivity, specificity, and the area under the receiver operating characteristic curve (AUROC) were calculated for EtCO₂. The study found a sensitivity of 86.8% and specificity of 88.5% for a 5% increase in EtCO₂, with an AUROC of 0.951, indicating high diagnostic accuracy. A significant correlation was observed between EtCO₂ changes and fluid responsiveness, validating EtCO₂ as a reliable predictor comparable to TTE. To conclude, EtCO₂ monitoring during PLR is a practical, non-invasive tool for assessing fluid responsiveness in ICU patients with shock. This method is suitable for bedside application, particularly in resource-limited settings, and supports informed fluid management decisions. Further multicenter studies are recommended to confirm its broader applicability.

Key words: capnography, fluid therapy, hemodynamics, mechanical ventilation, sensitivity and specificity, shock.

Introduction

Shock, characterized by inadequate tissue perfusion and oxygenation, is a critical concern in intensive care units (ICUs). Fluid administration is the cornerstone of shock management, aimed at increasing cardiac preload and cardiac output (CO). Dynamic indices have proven more reliable for evaluating fluid responsiveness than traditional static measures like central venous pressure, and inferior vena cava diameter. Current recommendations prefer dynamic assessments, including passive leg raising (PLR), mini fluid challenges, pulse pressure variation (PPV), and stroke volume variation (SVV) over static parameters such as central venous pressure (CVP) [1,2]. Despite their advantages, dynamic methods remain underutilized in ICUs due to the need for skilled operators, invasive techniques, or costly equipment [3].

To overcome these barriers, interest has grown in non-invasive methods like monitoring end-tidal carbon dioxide (EtCO₂) as a marker of fluid responsiveness. EtCO₂, the partial pressure of carbon dioxide (CO₂) at the end of expiration, can be measured via capnography, which is commonly available in mechanically ventilated patients. The physiological basis for using EtCO₂ is grounded in the Frank-Starling mechanism and Fick's principle, where increased CO from improved preload reduces alveolar dead space, raising EtCO₂ levels, provided CO₂ production by the body and its elimination via alveolar ventilation during controlled mechanical ventilation is relatively constant. This method is operator-independent, making it suitable for bedside use and an appealing alternative for fluid responsiveness evaluation in critically ill patients [4].

Despite the growing evidence supporting EtCO₂, its applicability in various patient populations, especially in resource-limited settings, remains underexplored. This study aims to evaluate the predictive value of EtCO₂ changes following PLR and fluid challenge in mechanically ventilated patients with shock in Indian ICUs, where a non-invasive, cost-effective method like EtCO₂ could significantly enhance bedside decision-making in critical care.

Materials and Methods

This was a single centre, prospective observational study conducted in a respiratory intensive care unit (RICU) in a tertiary care centre in New Delhi, India over a period of 18 months from November 2022 to April 2024. The sample size was determined based on a reference study by Monnet et al. (2013) that assessed the sensitivity and specificity of EtCO₂ as a marker of fluid responsiveness [5]. Using a sensitivity of 71% and specificity of 100%, and considering a 95% confidence interval with an α of 0.05 and a 10% dropout rate, the final sample size was

calculated as 90 participants. Consecutive patients admitted to the RICU with following inclusion & exclusion criteria were included in the study.

Inclusion criteria included adult patients admitted to the RICU who were on mechanical ventilation and presented with shock of any aetiology within 12 hours of onset, defined as: systolic blood pressure (SBP) <90 mm Hg, mean arterial pressure (MAP) <65 mmHg, or a decrease in SBP >40 mmHg from baseline [6,7]. Patient were excluded from the study if they met following exclusion criteria - cardiac arrhythmias; known mitral or aortic valve disease; factors limiting PLR manoeuvre such as orthopaedic deformities, lower limb fractures, or hip joint abnormalities; Pregnancy; Presence of intra-abdominal tumour, intestinal obstruction, or massive ascites; Deep venous thrombosis or the use of compression stockings; or those who had poor transthoracic echocardiographic window.

Ethics approval for the study was taken from Institutional Ethics Committee, Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi, dated 6th October 2022 (Serial number – EC/VMMC/SJH/Thesis/9/2022/CC-12).

Procedure and data collection

Equipment

The ultrasound equipment used was the Fujifilm Sonosite Edge II with a 1 to 5 Megahertz phased array probe (rP19x). EtCO₂ was measured using a Capnostat 5 mainstream capnograph integrated into an Avea ventilator.

Transthoracic echocardiography (TTE) for cardiac output

The left ventricular outflow tract (LVOT) diameter was measured from the parasternal long-axis view. The LVOT area was calculated using the following formula $LVOT\ area = [(LVOT\ diameter / 2)^2] \times \pi$.

Pulsed-wave Doppler was used to obtain velocity-time integrals (VTI) from the apical view at the LVOT. Stroke volume (SV) was calculated as $SV = LVOT\ area \times VTI\ of\ the\ LVOT\ blood\ flow$. The CO was calculated as $CO = Heart\ Rate \times SV$.

PLR test

The PLR manoeuvre was performed as follows:

1. Patients were initially positioned at a 45° semi-recumbent position.
2. Baseline CO as measured on TTE, and EtCO₂ levels were recorded.
3. The patient was then positioned supine with legs elevated at 45° using automated bed elevation.

4. Changes in CO and EtCO₂ were recorded at 1-minute intervals for 3 minutes.
5. The manoeuvre was repeated three times, and the average of the three readings was calculated.

A 10% or greater increase in CO following PLR was considered indicative of fluid responsiveness [8].

A 5% or greater increase in EtCO₂ during the PLR test was considered a positive response for fluid responsiveness [8].

Statistical analysis

Data was entered in Microsoft Excel and analysed using IBM SPSS statistics version 25. Normality of data - pre-PLR CO & EtCO₂ was tested with Kolmogorov Smirnov test. Primary objective was sensitivity and specificity of 5% rise in EtCO₂ to predict fluid responsiveness during PLR when compared to the 10% rise in CO measurement as measured by TTE. Receiver operating characteristic (ROC) curve analysis was performed to determine the diagnostic cutoff value for %change in EtCO₂ in predicting fluid responsiveness, and the area under the receiver operating characteristic curve (AUROC) was calculated. Kolmogorov Smirnov test showed non-normative distribution of all the parameters except age and MAP, which had normal distribution. Median and interquartile range (IQR) was calculated for all parameters except for age and MAP, where mean and standard deviation (SD) were calculated. Level of significance was calculated with Wilcoxon signed-rank test.

Results

The total number of admissions in the ICU during our study period was 340. Among 105 patients on mechanical ventilation with shock, 10 patients had poor acoustic window on transthoracic echo, two had severe aortic stenosis, one had immobilized legs post road traffic accident (RTA), one had undergone abdominal surgery and one had undergone leg amputation in the past, thus excluded from the study. Finally, 90 patients were included in the study after applying inclusion and exclusion criteria, as shown in Figure 1.

The mean age of the study population was 47.9 years with SD $\pm$ 18. 53 out of 90 study population had male gender, and 37 had female gender. The median sequential organ failure assessment (SOFA) score for the study population at the time of inclusion was 8 with IQR 3. The mean MAP was 68.1 mm Hg with SD $\pm$ 4.34. The median serum lactate level for the study population at the time of inclusion was 2.5 milli mole per liter with IQR 0.7 (Table 1).

Most common type of shock in the study was distributive shock followed by obstructive, cardiogenic, and hypovolemic shock. Septic shock was the most common cause of shock in the study population (N=68, 75.5%).

Wilcoxon signed-rank test was used to calculate level of significance. There was no significant difference in the baseline Pre PLR LVOT VTI, CO, and EtCO₂ between fluid-responsive and fluid-non-responsive groups. There was a significant difference in the post-PLR LVOT VTI, post-PLR CO, and post-PLR EtCO₂ between fluid-responsive and fluid-non-responsive patients (Table 2).

The sensitivity of Δ EtCO₂ 5% to predict fluid responsiveness was 86.8%, and the specificity was 88.5%.

AUROC calculated for Δ EtCO₂ 5% was 0.951. Youden's J Max Value for AUROC was 0.772. The corresponding optimal threshold for the percentage change in EtCO₂ is 5.41%, with the sensitivity and the specificity at this threshold being 86.8% & 90.4% respectively (Figure 2).

Discussion

Shock is a one of the most concerning complications in the ICUs. The incidence of shock, regardless of aetiology, ranges from 0.3 to 0.7 per 1,000 individuals, with septic shock being the most prevalent in ICU patients [9]. Fluid management is a critical component of shock management in the ICU, where both under- and over-resuscitation carry significant risks. Response of shock to intravenous fluid is time variable, with initial response followed by a decrease over time, as when the left ventricle functions near the plateau part of the Frank-Starling curve [2,9].

The change in EtCO₂ is a potential noninvasive alternative to more invasive techniques to assess fluid responsiveness in shock. Several studies have demonstrated the efficacy of EtCO₂ in predicting fluid responsiveness. Bhadade et al. (2022) reported that a 2 mmHg increase in EtCO₂ during PLR predicted fluid responsiveness with 74% sensitivity and 80% specificity in mechanically ventilated patients [10]. Similarly, Huang et al. (2022) confirmed moderate diagnostic accuracy with pooled sensitivity and specificity of 79% and 90%, respectively [11]. Monnet et al. (2012) found that EtCO₂ changes outperformed arterial pulse pressure in predicting fluid responsiveness, particularly in patients with spontaneous breathing or low tidal volume ventilation [5].

Our findings support that EtCO₂, measured during a passive leg raise (PLR), is an effective predictor, with a sensitivity and specificity of 86.8% and 88.5%, respectively, when a 5% change in EtCO₂ was used as the threshold. The strong performance of EtCO₂ in our study, as

indicated by a high AUROC and optimal sensitivity-specificity balance, positions it as a non-invasive, feasible bedside tool, especially relevant in settings with limited resources.

Sepsis was the most frequent cause of shock in our study population, contributing 75.5% of the study population. Nevertheless, the study did include patients with other types of shock as well, such as cardiogenic shock, obstructive shock and hypovolemic shock. Thus, the results of our study could indicate that EtCO₂ may serve as a universal marker across different shock types, leading to an easier method of establishing fluid responsiveness in settings with limited access to advanced hemodynamic monitoring.

Our study supports findings from other key studies like Monge Garcia et al. (2012) [4], Zang et al. (2013) [12], Xiao-ting et al. (2015) [13], Baloch et al. (2021) [14], and Özkarakas et al. (2024) [15]. However, our study differs by including patients with various shock types, in contrast to studies that focused on specific subgroups, such as septic shock (Zang et al. 2013; Özkarakas et al. 2024) or cardiogenic shock (Baloch et al., 2021) [12,14,15]. This inclusive approach underscores potential of EtCO₂ as a broad-spectrum marker of fluid responsiveness across shock etiologies.

In chronic respiratory disorders such as COPD, ventilatory limitations often lead to altered CO₂ elimination, potentially affecting EtCO₂ readings. Young et al. (2012) found that EtCO₂ and volumetric CO₂ were predictive of fluid responsiveness only in patients without underlying lung disease [16]. This aligns with Monnet et al.'s (2013) insights [5], which noted that COPD and other obstructive lung diseases might dampen EtCO₂ responsiveness due to altered ventilation-perfusion ratios. However, Monnet still reported a general predictive capability for EtCO₂ in a mixed cohort, which suggests that EtCO₂ still remains useful. Our study explored EtCO₂ dynamics in patients with primary respiratory conditions like chronic obstructive pulmonary disorder (COPD), asthma, and acute respiratory distress syndrome (ARDS), which comprised 42% of the cohort and still demonstrated good sensitivity and specificity of change in EtCO₂ as a marker of fluid responsiveness.

Despite promising results, several limitations need to be addressed. First, our single-center design in an ICU predominantly consisting of patients with respiratory disorders, limits the generalizability. Secondly, patients with certain physical limitations (e.g., orthopedic issues) or poor TTE windows were excluded, potentially influencing our results. Also, TTE was used to measure VTI and calculate CO based on VTI due to unavailability of continuous CO monitoring or other invasive methods of CO measurements such as pulmonary artery catheter (PAC). TTE has previously been shown to be comparable to PAC to measure cardiac output in critically ill [17-19]. Nonetheless, TTE remains an operator dependent tool [20], and so its use

for CO measurement as a comparator in current study may have led to less accurate diagnosis of fluid responsiveness.

Finally, while EtCO₂ provides a non-invasive solution, its efficacy in fluid responsiveness prediction could vary based on disease severity and ventilatory status, warranting further multi-center studies for validation. And last but not the least, in our study, patients were on controlled mechanical ventilation at the time of assessment, preventing the generalization to patients on assist-control mechanical ventilation and spontaneously breathing patient population.

Conclusions

This study demonstrates that %change in EtCO₂ of 5% pre and post PLR is an effective, non-invasive predictor of fluid responsiveness in mechanically ventilated ICU patients with shock, comparable to technically more challenging methods such as CO measurement by TTE. Our findings suggest EtCO₂'s potential as a universal fluid responsiveness marker across various shock types, including patients with primary respiratory conditions like COPD. This method, especially relevant in resource-limited settings, supports improved bedside decision-making for fluid resuscitation. Further multi-center studies could broaden its applicability, including validation in spontaneously breathing patients.

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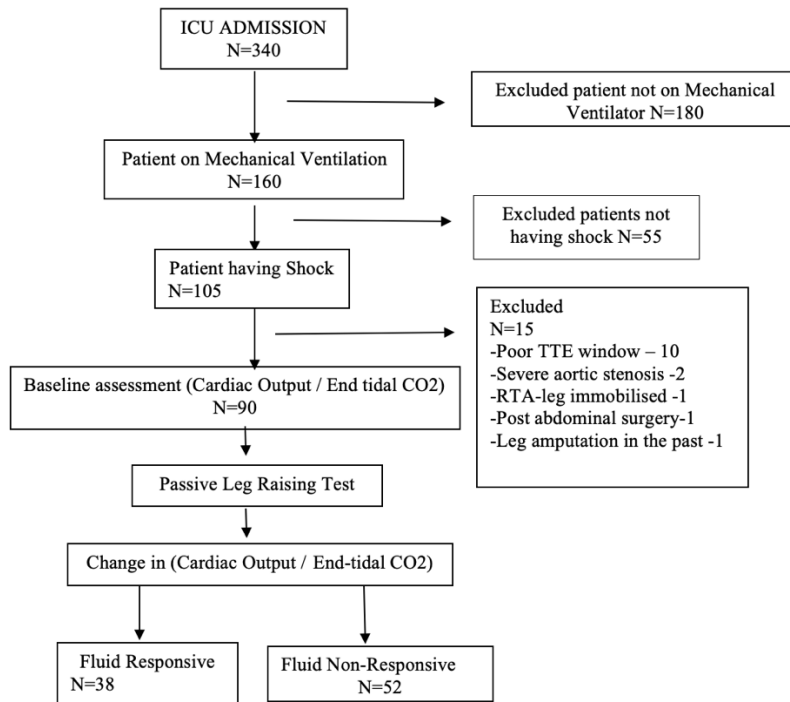


Figure 1. Flow chart for execution of the study.

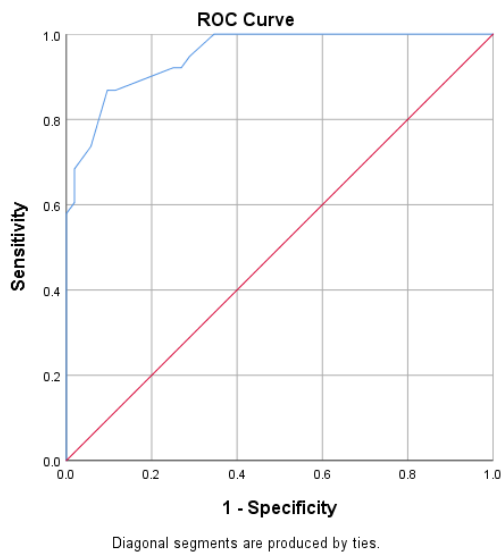


Figure 2. Area under receiver operating characteristic curve for change in end-tidal carbon dioxide to predict fluid responsiveness.

Table 1. Baseline study characteristics (n=90).

	Mean $\pm$ SD	Median (Minimum-Maximum, IQR)
Age (years)	47.9 $\pm$ 18	
Sex	Male Gender N=53 (58.89%) Female Gender N=37 (41.11%)	
ICU Length of stay at the point of inclusion (Days)		3 (1-45, 6)
SOFA score		8 (5-12, 3)
MAP (mm Hg)	68.1 $\pm$ 4.34	
Lactate (mmol/L)		2.5 (1.8-4.8, 0.7)
Urine output (ml/kg/hour)		0.3 (0.1-0.5, 0.13)
Admitting diagnosis	Number of patients (%)	
AECOPD	17 (18.8%)	
Pneumonia	15 (16.7%)	
ARDS	14 (15.6%)	
TB	12 (13.3%)	
Lung cancer	10 (11.1%)	
Myocardial infarction	7 (7.8%)	
AEILD	4 (4.4%)	
Asthma	3 (3.3%)	
Urosepsis	3 (3.3%)	
GBS	3 (3.3%)	
Traumatic Brain Injury	2 (2.2%)	
Type of shock	Number of patients (%)	
Distributive	70 (77.7%)	
Cardiogenic	7 (7.7%)	
Hypovolemic	4 (4.4%)	
Obstructive	9 (10%) – Pulmonary embolism n=7 (7.7%) and Cardiac tamponade n=2 (2.2%)	
Patients receiving vasopressors/inotropes (number of patients, %)	45 (50%)	

AECOPD, acute exacerbation of chronic obstructive pulmonary disorder; AEILD, acute exacerbation of interstitial lung disease; ARDS, acute respiratory distress syndrome; GBS, Guillain Barré syndrome; ICU, intensive care unit; MAP, mean arterial pressure; SD, standard deviation; SOFA, sequential organ failure assessment; TB, tuberculosis.

Table 2. Baseline and post-passive leg raising cardiac output and end-tidal carbon dioxide.

		Baseline (Pre-PLR)	Post-PLR	p-value
LVOT VTI (cm)	Fluid responsive (n=38)	17.3 (2.47)	21.35 (2.1)	0.001
Median (IQR)	Fluid non-responsive (n=52)	18.3 (1.95)	19.5 (1.9)	0.34
Cardiac Output (L/min),	Fluid responsive (n=38)	4.46 (2.83)	5.82 (2.79)	0.02
Median (IQR)	Fluid non-responsive (n=52)	4.93 (2.31)	5.19 (2.14)	0.39
EtCO ₂ (mm Hg)	Fluid responsive (n=38)	41 (7)	44 (8)	0.019
Median (IQR)	Fluid non-responsive (n=52)	39 (7)	40 (6)	0.51

EtCO₂, end-tidal carbon dioxide; IQR, interquartile range; LVOT VTI, left ventricular outflow tract velocity time integral; PLR, passive leg raising; SD, standard deviation.