

Effective arterial elastance as a predictor of fluid responsiveness in patients with septic shock—a prospective observational study

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Abstract

Background

Assessment of fluid responsiveness is essential in the resuscitation of patients with septic shock. Effective arterial elastance (Ea) is a lumped index of arterial load; however, its ability to predict fluid responsiveness in these patients remains unknown.

Methods

This prospective observational study was conducted in the intensive care unit of Zhejiang Cancer Hospital. All enrolled patients underwent a 500mL fluid challenge (FC), and hemodynamic parameters, including cardiac output (CO) and mean arterial pressure (MAP), were recorded using the PiCCO system. Fluid responsiveness was defined as an increase in CO of > 10% after the fluid challenge, and arterial pressure responsiveness as an increase in MAP of > 10%. Receiver operating characteristic (ROC) curve was performed to evaluate the predictive ability of Ea. Correlations between Ea and FC-induced changes in CO and MAP were assessed.

Results

Forty-one patients were analysed, of whom 20 (49%) were fluid responders. Baseline Ea was significantly higher in fluid responders than in fluid nonresponders [2.43 (2.07–4.04) vs 1.82 (1.61–2.47) mmHg/mL, $p = 0.022$]. Baseline Ea predicted fluid responsiveness with an area under the curve (AUC) of 0.710 (95% CI: 0.549–0.870, $p = 0.022$) and an optimal cut-off value of 2.00 mmHg/mL (sensitivity: 80%; specificity: 62%), but lacked predictive ability for arterial pressure responsiveness (AUC = 0.583, 95% CI: 0.361–0.781, $p = 0.427$). FC-induced percentage change in CO ($\Delta\text{CO}\%$) was moderately correlated with baseline Ea ($\rho = 0.45$, $p = 0.004$), whereas the percentage change in MAP ($\Delta\text{MAP}\%$) was not ($\rho = -0.28$, $p = 0.080$). Among the fluid responders, 15 (75%) were arterial pressure nonresponders. In this subgroup, Ea decreased significantly after the fluid challenge [1.91 (1.67–2.45) vs 2.43 (2.07–4.29) mmHg/mL, $p = 0.001$], and the percentage change in Ea ($\Delta\text{Ea}\%$) was strongly correlated with both $\Delta\text{CO}\%$ ($\rho = -0.65$, $p = 0.002$) and $\Delta\text{MAP}\%$ ($r = 0.67$, $p = 0.001$).

Conclusion

In patients with septic shock, baseline Ea > 2.00 mmHg/mL predicts fluid responsiveness but not arterial pressure responsiveness. A significant decrease in Ea after a fluid challenge may explain the dissociation between fluid responsiveness and arterial pressure responsiveness.

Introduction

According to the early goal directed therapy strategy, fluid resuscitation has been considered a cornerstone of treatment for patients with septic shock. Fluid resuscitation increases cardiac preload, thereby enhancing cardiac output (CO) and oxygen delivery, ultimately improving tissue perfusion in these patients[1]. However, fluid overload has attracted increasing attention because it may lead to cardiac dysfunction, pulmonary edema, pleural effusion, and even adverse clinical outcomes[2]. Therefore, assessment of fluid responsiveness, defined as the ability to significantly increase CO in response to fluid expansion[3], is recommended by current guidelines to optimize fluid management[4, 5, 6].

The goal of fluid resuscitation is not only to achieve adequate CO but also to maintain sufficient mean arterial pressure (MAP), thereby ensuring adequate tissue perfusion pressure. However, an increase in CO does not necessarily result in a concomitant increase in arterial blood pressure, because the relationship between CO and arterial pressure is also influenced by the vascular system. Arterial pressure reflects the interaction between the heart and the arterial system. During a fluid challenge (FC), arterial pressure is determined not only by cardiac preload and myocardial contractility, but also by cardiac afterload and the mechanical properties of the arterial system[7]. Previous studies have demonstrated that the dissociation between CO and arterial pressure responses to a fluid challenge is largely attributable to changes in arterial load[7, 8].

Arterial load encompasses the extracardiac factors that influence ventricular ejection, including the steady component, clinically represented by systemic vascular resistance (SVR), and the pulsatile components (arterial compliance, characteristic aortic impedance, and wave reflections)[9]. Effective arterial elastance (Ea) is a lumped index of the arterial load imposed on the left ventricle during ejection. Ea is defined as the ratio of end-systolic pressure (Pes) to stroke volume (SV) ($Ea = Pes / SV$), representing the slope of the arterial end-systolic pressure-volume relationship[10]. Previous studies have shown that Pes can be estimated as 90% of systolic arterial pressure (SAP), allowing Ea to be calculated as $(0.9 \times SAP) / SV$ [11, 12]. This approach permits bedside measurement and continuous monitoring of Ea, thereby facilitating hemodynamic assessment in clinical practice. Ea may help explain the dissociation between CO and arterial pressure responses to fluid resuscitation in patients with septic shock. While few studies have evaluated whether Ea can predict fluid responsiveness, most research has focused on dynamic arterial elastance (Eadyn) as a predictor of arterial pressure responsiveness[13, 14]. Understanding the determinants of Ea during a fluid challenge could help identify patients who may develop a decrease in arterial pressure despite adequate preload, thereby guiding individualized hemodynamic management. Therefore, the present study aimed to investigate the value of Ea in predicting fluid responsiveness in patients with septic shock.

Materials and Methods

Study population

This was a prospective, observational study. Patients with septic shock admitted to the intensive care unit of Zhejiang Cancer Hospital between October 2023 and November 2025 were enrolled. The inclusion criteria were as follows: (1) age ≥ 18 years; (2) fulfillment of the diagnostic criteria for septic shock[15], which include hypotension, sustained vasopressor requirement to maintain MAP ≥ 65 mmHg and serum lactate > 2 mmol/L; (3) central venous catheter in place for measurement of central venous pressure (CVP); (4) availability of hemodynamic monitoring using the Pulse index Continuous Cardiac Output (PiCCO) system (PULSION Medical Systems SE, Feldkirchen, Germany); (5) a fluid challenge of 500 mL of crystalloid solution was administered over 10 minutes based on PiCCO-derived hemodynamic parameters and clinical judgment by the attending physician. The exclusion criteria were as follows: (1) age < 18 years; (2) pregnancy or perinatal status; (3) coexistence or diagnosis of other types of shock, including cardiogenic, hemorrhagic, or obstructive shock; (4) hemodynamic instability during the fluid challenge requiring adjustment of vasoactive drug dosage; (5) a fluid challenge was not clinically indicated by the attending physician.

Definitions

Fluid responsiveness was defined as an increase in CO of $> 10\%$ after the fluid challenge, during which patients received 500 mL of crystalloid solution over 10 minutes[16]. Similarly, arterial pressure responsiveness was defined as an increase in MAP of $> 10\%$ after the fluid challenge[17].

Data collection

Data collected included demographic characteristics (age, sex, body mass index [BMI]), comorbidities, Acute Physiology and Chronic Health Evaluation II (APACHE II) score, vasopressor use and other clinical variables. Hemodynamic parameters, including heart rate (HR), diastolic arterial pressure (DAP), SAP, MAP, CVP, CO, SV, Ea, SVR, net arterial compliance (C) and arterial time constant (Tau), were recorded before and immediately after the fluid challenge. These data were recorded using the PiCCO system for analysis. The following formulas were used: $Ea = (0.9 \times SAP) / SV$; $C = SV / (SAP - DAP)$; $Tau = SVR \times C \times (60 / 80,000)^{\dagger}$. $\dagger$ SVR was expressed in $\text{dyn} \cdot \text{s} \cdot \text{cm}^{-5}$. The constant 80,000 was used to convert SVR to $\text{mmHg} \cdot \text{s} \cdot \text{mL}^{-1}$; the factor 60 converted minutes to seconds.

Statistical analysis

Statistical analyses were performed using SPSS version 26.0 (IBM Corp, Armonk, NY, USA) and GraphPad Prism version 10.0 (GraphPad Software, Boston, MA, USA). The normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed continuous variables were expressed as mean $\pm$ standard deviation and compared using the independent-samples t-test (for between-group comparisons) or the paired t-test (for before-and-after fluid challenge comparisons). Non-normally distributed continuous variables were expressed as median (interquartile range, IQR) and compared using the Mann-Whitney U test (independent samples) or the Wilcoxon signed-rank test (paired samples). Categorical variables were presented as n (%) and compared using the Pearson chi-square test or Fisher's exact test when expected cell counts were < 5 . Receiver operating characteristic

(ROC) curves were generated to evaluate the discriminatory ability of Ea and arterial load-related parameters for fluid responsiveness and arterial pressure responsiveness. The area under the ROC curve (AUC) with 95% confidence interval (CI) was calculated, and the optimal cutoff was determined using the Youden index. Correlations were assessed using Spearman's rank correlation coefficient (ρ) for non-normally distributed variables and Pearson's correlation coefficient (r) for normally distributed variables. A two-tailed P value < 0.05 was considered statistically significant.

Results

Demographic and clinical features

A total of 41 patients with septic shock were included in the analysis, with a median age of 69 years and a mean APACHE II score of 24.1 ± 4.8 . Twenty patients were fluid responders and 21 were fluid nonresponders. Baseline characteristics were comparable between the two groups (Table 1). The most common site of infection was the abdomen (20, 48.8%), followed by pulmonary infection (13, 31.7%) and bloodstream infection (8, 19.5%). There were no statistically significant differences between the two groups in demographic and clinical features, including APACHE II score, use of vasoactive agents and comorbidities (all $p > 0.05$).

hemodynamic parameters before and after the fluid challenge

Changes in hemodynamic parameters before and after the fluid challenge are presented in Table 2. Compared with baseline, CO and SV increased significantly after the fluid challenge in fluid responders (both $p < 0.001$). Concurrently, in fluid responders, Ea decreased significantly from 2.43 (2.07–4.04) to 2.14 (1.70–2.55) mmHg/mL ($p = 0.004$) and SVR from 1161 (899–1461) to 981 (725–1136) $\text{dyn} \cdot \text{s} \cdot \text{cm}^{-5}$ ($p < 0.001$) after the fluid challenge, whereas C increased from 0.74 ± 0.37 to 0.90 ± 0.33 mL/mmHg ($p < 0.001$). In fluid nonresponders, SV increased after the fluid challenge ($p = 0.028$), whereas the remaining hemodynamic parameters did not differ significantly from baseline (all $p > 0.05$).

Intergroup comparison of hemodynamic parameters before and after the fluid challenge

As shown in Table 3, before the fluid challenge, SV was significantly higher in fluid nonresponders than in fluid responders ($p = 0.047$). In addition, no statistically significant differences in the other hemodynamic variables were observed between the two groups either before or after the fluid challenge (all $p > 0.05$). At baseline, Ea was significantly higher in fluid responders than in fluid nonresponders [2.43 (2.07–4.04) vs 1.82 (1.61–2.47) mmHg/mL, $p = 0.022$], and C was significantly lower [0.71 (0.43–0.90) vs 1.15 (0.80–1.36) mL/mmHg, $p = 0.003$]. After the fluid challenge, however, Ea and C did not differ significantly between the two groups ($p = 0.411$, $p = 0.054$, respectively). Tau was significantly lower in fluid responders than in fluid nonresponders both before and after the fluid challenge ($p = 0.019$ and $p = 0.011$, respectively). No statistically significant between-group differences were observed in the other arterial load parameters at either time point (all $p > 0.05$).

Hemodynamic parameters in arterial pressure responders vs nonresponders

Among the 20 fluid responders, 5 (25%) were arterial pressure responders and 15 (75%) were arterial pressure nonresponders. As shown in Table 4, in arterial pressure responders, the fluid challenge significantly increased CO, SAP, DAP and MAP ($p = 0.043$, $p = 0.002$, $p = 0.043$, $p = 0.042$, respectively), with no significant changes in arterial load parameters (all $p > 0.05$). In arterial pressure nonresponders, the fluid challenge significantly increased CO and SV ($p = 0.001$, $p < 0.001$, respectively), significantly decreased Ea [from 2.43 (2.07–4.29) to 1.91 (1.67–2.45) mmHg/mL] and SVR [from 1092 (890–1419) to 942(736-1005) dyn·s·cm⁻⁵] (both $p = 0.001$), and significantly increased C (from 0.74 ± 0.41 to 0.95 ± 0.34 mL/mmHg) ($p < 0.001$). As shown in Table 5, no significant differences in baseline hemodynamic parameters were observed between arterial pressure responders and nonresponders (all $p > 0.05$). However, after the fluid challenge, arterial pressure nonresponders had significantly lower Ea, SAP and MAP than arterial pressure responders ($p = 0.033$, $p = 0.020$, $p < 0.001$, respectively).

Predictive value of Ea for fluid responsiveness

The predictive value of Ea for fluid responsiveness was shown in Figure 1. ROC curve analysis showed that baseline Ea was predictive of fluid responsiveness in patients with septic shock, with an AUC of 0.710 (95% CI: 0.549–0.870, $p = 0.022$). At a cutoff value of 2.00 mmHg/mL, the sensitivity was 80% and the specificity was 62%.

Predictive value of arterial load parameters for arterial pressure responsiveness

The predictive value of arterial load parameters for arterial pressure responsiveness was shown in Figure 2. ROC curve analysis showed that baseline Ea did not predict arterial pressure responsiveness after the fluid challenge (AUC = 0.583, 95% CI: 0.361–0.781, $p = 0.427$). Similarly, neither C nor Tau predicted arterial pressure responsiveness in patients with septic shock (C: AUC = 0.430, 95% CI: 0.244–0.628, $p = 0.508$; Tau: AUC = 0.465, 95% CI: 0.277–0.651, $p = 0.746$, respectively).

Correlation Analyses

Baseline Ea showed a moderate positive correlation with the percentage change in CO ($\Delta\text{CO}\%$) after the fluid challenge ($\rho = 0.45$, $p = 0.004$), but not with the percentage change in MAP ($\Delta\text{MAP}\%$) ($\rho = -0.28$, $p = 0.080$) (Figure 3). Among the fluid responders, the percentage change in Ea ($\Delta\text{Ea}\%$) was negatively correlated with $\Delta\text{CO}\%$ ($\rho = -0.65$, $p = 0.002$) and positively correlated with $\Delta\text{MAP}\%$ ($r = 0.67$, $p = 0.001$) after the fluid challenge. (Figure 4)

Discussion

Assessing fluid responsiveness is recommended by the current guidelines to optimize fluid management. CO is regarded as the gold standard for assessing fluid responsiveness and the PiCCO system has been established as a reliable method for CO evaluation. In the present study, CO was monitored using the PiCCO system, which integrates transpulmonary thermodilution calibration with

arterial waveform analysis to provide continuous, real-time hemodynamic data. This approach enables reliable quantification of key parameters, including CO, SV, SVR, MAP, and global end-diastolic volume, thereby offering a comprehensive assessment of circulatory status in critically ill patients[18]. However, assessment of fluid responsiveness based on arterial pressure may lead clinicians to overlook patients who could potentially benefit from fluid therapy, as the hemodynamic responses of CO and arterial pressure to fluid challenge are not always concordant. Ranjit et al.[19] found that changes in cardiac index did not correlate with changes in MAP following fluid administration ($r = 0.035$, $p = 0.79$). Yang et al.[20] further found that Ea decreased after a fluid challenge despite an increase in CO, suggesting that the reduction in arterial load might explain why some patients increased their CO without a concomitant increase in arterial blood pressure. Importantly, arterial pressure should not be regarded as a reliable substitute for CO when evaluating fluid responsiveness. During a fluid challenge, the arterial pressure response is determined not only by the change in CO, but also by concurrent changes in arterial elastance[21].

Ea, calculated as the ratio of Pes to SV, serves as a lumped afterload index of arterial load[22]. Compared with SVR or arterial pressure, Ea provides a more integrative characterization of ventricular load. Beyond serving as an afterload index, Ea reflects the energetic coupling between the heart and the arterial tree. Together with cardiac contractile performance, it also contributes to regulating arterial pressure and blood flow[23]. Despite these theoretical advantages, Ea has not been widely adopted in clinical practice, largely because its mathematical similarity to SVR has led to the misconception that it is merely a surrogate for vascular resistance. In our study, we observed that Ea and SVR decreased significantly, while C increased significantly in fluid responders after the fluid challenge. Several earlier investigations involving patients with septic shock reported similar findings[7,24]. In those studies, fluid responders exhibited declines in Ea and SVR with an increase in C after a fluid challenge. The decline in arterial load may be largely explained by suppression of sympathetic outflow as circulating volume was replenished. Several physiological mechanisms may account for the reduction in arterial load. First, fluid challenge may alleviate baroreflex-mediated vasoconstriction induced by hypovolemia. Second, reopening of collapsed vascular beds after a fluid challenge could increase the effective arterial cross-sectional area[7]. Third, the augmented blood flow caused by fluid challenge may enhance endothelial shear stress, thereby upregulating nitric oxide production and facilitating vascular relaxation[25].

Studies on the role of Ea in fluid responsiveness remain notably limited, Yang et al.[26] used a pulmonary artery catheter to monitor CO and demonstrated that a baseline Ea > 1.97 mmHg/mL predicted fluid responsiveness in patients with septic shock, with a sensitivity of 71% and a specificity of 60%. They also demonstrated that Ea did not predict arterial pressure responsiveness in patients with septic shock. To the best of our knowledge, this is the first study to evaluate the predictive value of Ea, derived from PiCCO monitoring, for fluid responsiveness in patients with septic shock. We found that Ea was significantly higher in fluid responders than in fluid nonresponders [2.43 (2.07–4.04) vs 1.82 (1.61–2.47) mmHg/mL, $p = 0.022$]. After the fluid challenge, Ea decreased significantly in fluid responders, whereas it remained unchanged in fluid nonresponders. Moreover, as shown in Figure 3A, higher baseline Ea was associated with a greater percentage increase in CO after the fluid challenge. This finding may be

explained by the greater reduction in Ea observed in patients with elevated baseline arterial tone, whereby afterload reduction contributed to improved ventricular ejection. These findings suggest that a higher baseline Ea was associated with a greater likelihood of fluid responsiveness. Specifically, in our study, a baseline Ea value > 2.00 mmHg/mL predicted fluid responsiveness in patients with septic shock. We also found that Ea failed to predict arterial pressure responsiveness in patients with septic shock (AUC = 0.583). There was no significant correlation between baseline Ea and Δ MAP% ($\rho = -0.28$, $p = 0.080$). Further analysis in the fluid responder group revealed a positive correlation between Δ Ea% and Δ MAP% ($r = 0.67$, $p = 0.001$), indicating that a smaller reduction in Ea was associated with a greater increase in MAP, whereas a greater reduction in Ea corresponded to a smaller increase or even a decline in MAP. Mechanistically, Ea reflects the capacity of the arterial system to convert SV into pressure. Accordingly, a higher Ea yields a greater arterial pressure for a given SV. Conversely, a decrease in Ea after a fluid challenge implies that arterial pressure may not increase appreciably or may even decline, indicating arterial pressure nonresponsiveness to fluid administration. Overall, the reduction in Ea after a fluid challenge may partly explain why some patients who exhibit fluid responsiveness do not demonstrate a concomitant increase in arterial pressure.

The interaction between the ventricle and the arterial system is termed ventricular-arterial coupling (VAC), which is quantified by the ratio of Ea to ventricular end-systolic elastance (Ees)[27]. Previous studies have reported the normal Ea / Ees range as 0.5–1.3, with a ratio of approximately 1 generally considered to reflect optimal VAC[28,29,30]. Under this condition, administration of a fluid bolus that augments CO would be expected to produce a corresponding increase in MAP. Septic shock is characterized by ventricular-arterial decoupling. In the early phase of shock, parallel reductions in Ea and Ees tend to preserve the Ea / Ees ratio near baseline. As shock progresses to the decompensated phase, however, Ees continues to decline, whereas Ea remains relatively unchanged or may even increase, partly due to the administration of vasoactive agents. This divergence consequently elevates the Ea / Ees ratio, reflecting progressive ventricular-arterial decoupling[30,31]. A recent study demonstrated that fluid expansion enhanced CO in fluid responsive patients, thereby improving ventricular-arterial coupling in patients with shock. This improvement was mainly attributable to a reduction in Ea[24]. Consistent with previous findings, our results demonstrated that Ea was significantly reduced after the fluid challenge in fluid responders[24,32,33]. These findings suggest that vascular autoregulatory function is relatively preserved in fluid responders and that the reduction in Ea may decrease ventricular afterload and thereby improve cardiac efficiency. Conversely, during fluid resuscitation, the use of vasoactive agents to increase vascular resistance and achieve target arterial pressure may aggravate ventricular-arterial decoupling, thereby accelerating circulatory deterioration. In addition, a subgroup analysis of fluid responders based on arterial pressure responsiveness revealed that Ea decreased significantly only in those who were fluid responders but arterial pressure nonresponders, whereas it remained unchanged in patients responsive to both fluid and arterial pressure. Correlation analysis showed that among the fluid responders, a greater reduction in Ea after the fluid challenge was associated with a greater increase in CO, whereas MAP improved to a lesser extent or even declined. This may explain why CO increased in some patients after the fluid challenge without a concomitant increase in arterial pressure. In the

present study, baseline Ea did not predict arterial pressure responsiveness in patients with septic shock, which is consistent with the findings of several previous studies[20,26]

This study has several limitations that should be acknowledged. First, the binary categorization of fluid responsiveness may be criticized, given that this parameter is physiologically continuous rather than dichotomous. Nevertheless, such a classification provides a practical framework for addressing the research question. Second, cardiac function parameters were not continuously monitored, and VAC was not dynamically assessed. Consequently, whether changes in Ea were accompanied by corresponding changes in VAC after a fluid challenge remains unclear and warrants further investigation. Third, this was a single-center study with a relatively small sample size, which may limit the statistical power and generalizability of the findings. Therefore, further large-scale, multicenter studies are needed to confirm the value of Ea in predicting fluid responsiveness. Fourth, this study was conducted primarily from a systemic hemodynamic perspective, and the underlying mechanisms warrant further investigation through in-depth basic research. Despite these limitations, the predictive value of Ea in patients with septic shock remains promising and warrants further investigation before broader clinical application.

Conclusion

In summary, baseline Ea > 2.00 mmHg/mL predicts fluid responsiveness but not arterial pressure responsiveness in patients with septic shock. A significant decrease in Ea after a fluid challenge may explain why CO improved without a concomitant increase in arterial pressure.

Abbreviations

Ea: effective arterial elastance; CO: cardiac output; MAP: mean arterial pressure; FC: fluid challenge; PiCCO: Pulse index Continuous Cardiac Output; ROC: receiver operating characteristic; AUC: area under the curve; CI: confidence interval; SVR: systemic vascular resistance; HR: heart rate; BMI: body mass index; Pes: end-systolic pressure; SV: stroke volume; CVP: central venous pressure; APACHE II: Acute Physiology and Chronic Health Evaluation II; SAP: systolic arterial pressure; Eadyn: dynamic arterial elastance; DAP: diastolic arterial pressure; C: net arterial compliance; Tau: arterial time constant; IQR: interquartile range; VAC: ventricular-arterial coupling; Ees: end-systolic elastance.

Declarations

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Ethics statement

This study was approved by the ethics committee of Zhejiang Cancer Hospital Involving Human Subjects (Approval No. IRB-2023-861). Written informed consent was obtained from the patient or, from next of kin or an independent patient advocate if the patient lacked this capacity. All clinical investigations adhered to the principles outlined in the Declaration of Helsinki. Clinical trial number: not applicable.

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Consent for publication

Not applicable.

Author Contributions

Conceptualization, JFT, KZL, JS; Methodology, JFT, JS; Formal analysis, JS, LHS; Investigation, LHS, YQC; Validation, YQC, JS; resources, JFT, LHS; data curation, JFT, JS, YQC, LHS; writing-original draft preparation, JFT, JS; writing-review and editing, JFT, KZL; visualization, JFT, JS; supervision, KZL; project administration, JFT, KZL; funding acquisition, JFT. All authors have read and agreed to the published version of the manuscript.

Data Availability Statement

The datasets used and/or analysed in the current study are available from the corresponding author upon reasonable request.

Competing Interests

The authors have declared that no competing interest exists.

References

1. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Eng J Med*. 2001; 345(19): 1368-77.
2. Pfortmueller CA, Dabrowski W, Wise R, et al. Fluid accumulation syndrome in sepsis and septic shock: pathophysiology, relevance and treatment-a comprehensive review. *Ann Intensive Care*. 2024; 14(1): 115
3. Monnet X, Malbrain M, Pinsky MR. The prediction of fluid responsiveness. *Intensive Care Med*. 2023; 49(1): 83-86.
4. Evans L, Rhodes A, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. *Intensive Care Med*. 2021; 47(11): 1181-1247.

5. Mekontso Dessap A, AlShamsi F, Belletti A, et al. European society of intensive care medicine (ESICM) 2025 clinical practice guideline on fluid therapy in adult critically ill patients: part 2-the volume of resuscitation fluids. *Intensive Care Med.* 2025; 51(3): 461-477.
6. Prescott HC, Antonelli M, Alhazzani W, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2026. *Intensive Care Med.* 2026; 52(5): 863-936.
7. Monge García MI, Guijo González P, Gracia Romero M, et al. Effects of fluid administration on arterial load in septic shock patients. *Intensive Care Med.* 2015; 41(7): 1247-1255.
8. Valverde A. Fluid Resuscitation for Refractory Hypotension. *Front Vet Sci.* 2021; 8: 621696.
9. Monge García MI, Saludes Orduña P, Cecconi M. Understanding arterial load. *Intensive Care Med.* 2016; 42(10): 1625-1627.
10. Sunagawa K, Maughan WL, Burkhoff D, et al. Left ventricular interaction with arterial load studied in isolated canine ventricle. *Am J Physiol.* 1983; 245(5 Pt 1): H773-780.
11. Kelly RP, Ting CT, Yang TM, et al. Effective arterial elastance as index of arterial vascular load in humans. *Circulation.* 1992; 86(2): 513-521.
12. Gaczoł M, Rajzer M, Wojciechowska W. Ventricular-arterial coupling: changes with ageing and implications across cardiovascular conditions. *Blood Press.* 2025; 34(1): 2457698.
13. Paphawin S, Theerawit P, Junhasavasdikul D, et al. The Accuracy of Dynamic Parameters for Prediction of Fluid Responsiveness in Elderly Patients with Septic Shock. *Int J Gen Med.* 2025; 18: 2333-2342.
14. Zhou X, Pan W, Chen B, et al. Predictive performance of dynamic arterial elastance for arterial pressure response to fluid expansion in mechanically ventilated hypotensive adults: a systematic review and meta-analysis of observational studies. *Ann Intensive Care.* 2021; 11(1): 119.
15. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA.* 2016; 315(8): 801-10.
16. De Backer D, Aissaoui N, Cecconi M, et al. How can assessing hemodynamics help to assess volume status? *Intensive Care Med.* 2022; 48(10): 1482-1494.
17. Caicedo Ruiz JD, Aldana JL, Kattan E, et al. Left ventricular-arterial coupling in septic shock: A physiological review. *J Crit Care.* 2026; 93: 155486.
18. Persona P, Valeri I, Saraceni E, et al. Cardiac Output Evaluation on Septic Shock Patients: Comparison between Calibrated and Uncalibrated Devices during Vasopressor Therapy. *J Clin Med.* 2021; 10(2): 213.
19. Ranjit S, Natraj R, Kissoon N, et al. Variability in the Hemodynamic Response to Fluid Bolus in Pediatric Septic Shock. *Pediatr Crit Care Med.* 2021; 22(8): e448-e458.
20. Yang T, Weng L, Jiang W, et al. Hemodynamic effects of different fluid volumes for a in septic shock patients. *Chin Med J (Engl).* 2022; 135(6): 672-680.
21. Messina A, Collino F, Cecconi M. Fluid administration for acute circulatory dysfunction using basic monitoring. *Ann Transl Med.* 2020; 8(12): 788.

22. Protti I, van den Enden A, Van Mieghem NM, et al. Looking Back, Going Forward: Understanding Cardiac Pathophysiology from Pressure-Volume Loops. *Biology*. 2024; 13(1): 20-24.
23. Yang T, Jiang W, Yang D, et al. Predictive value of change in effective arterial elastance in norepinephrine weaning: a retrospective study. *Ann Palliat Med*. 2021; 10(6): 6325-6335.
24. Zhou X, Fang H, Pan T, et al. Hemodynamic effects of volume expansion on left ventricular-arterial interactions in circulatory shock-a prospective observational study. *Front Cardiovasc Med*. 2026; 13: 1726410.
25. Losser MR, Forget AP, Payen D. Nitric oxide involvement in the hemodynamic response to fluid resuscitation in endotoxic shock in rats. *Crit Care Med*. 2006; 34(9): 2426-31.
26. Yang T, Huang H, Weng L, et al. effective arterial elastance in evaluating the in septic shock patients. *Chin Crit Care Med*. 2021; 33(3): 269-275.
27. Zhou X, Zhang Y, Pan J, et al. Optimizing left ventricular-arterial coupling during the initial resuscitation in septic shock-a pilot prospective randomized study. *BMC Anesthesiol*. 2022; 22(1): 31.
28. Monge García MI, Santos A. Understanding ventriculo-arterial coupling. *Ann Transl Med*. 2020; 8(12): 795.
29. Chantler PD, Lakatta EG. Arterial-ventricular coupling with aging and disease. *Front Physiol*. 2012; 3: 90.
30. Demailly Z, Besnier E, Tamion F, et al. Ventriculo-arterial (un)coupling in septic shock: Impact of current and upcoming hemodynamic drugs. *Front Cardiovasc Med*. 2023; 10: 1172703.
31. Guarracino F, Ferro B, Morelli A, et al. Ventriculoarterial decoupling in human septic shock. *Crit Care*. 2014; 18(2): R80.
32. Huette P, Abou-Arab O, Longrois D, et al. Fluid expansion improve ventriculo-arterial coupling in preload-dependent patients: a prospective observational study. *BMC Anesthesiol*. 2020; 20(1): 171.
33. Li S, Wan X, Laudanski K, et al. Left-Sided ventricular-arterial coupling and volume responsiveness in septic shock patients. *Shock*. 2019; 52(6): 577-582.

Tables

Tables 1 to 5 are available in the Supplementary Files section.

Figures

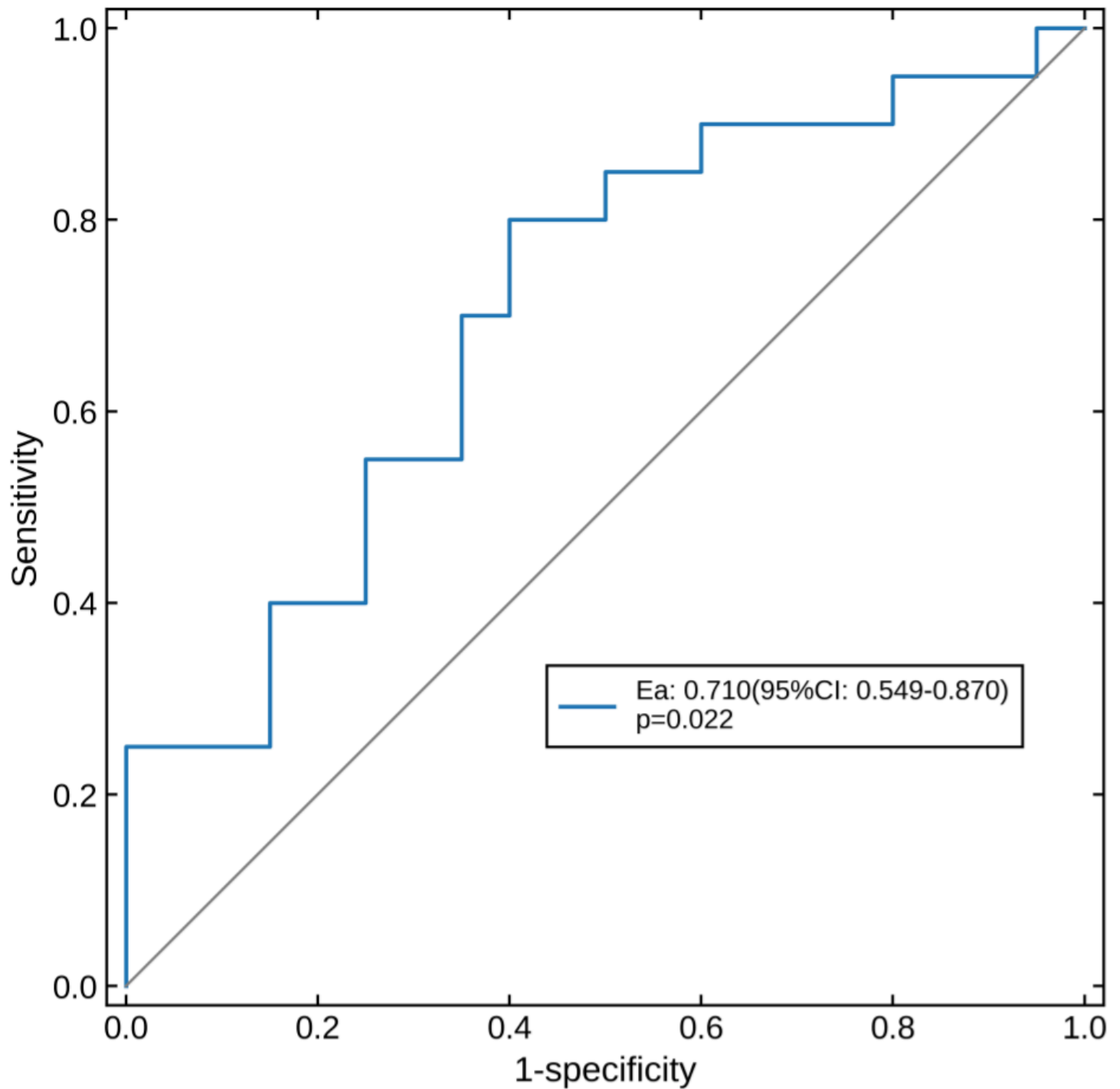


Figure 1

Receiver operating characteristic (ROC) curve of baseline effective arterial elastance (Ea) for predicting fluid responsiveness. The area under the curve (AUC) of Ea was 0.710 (95% confidence interval [CI]: 0.549–0.870, $p = 0.022$). The diagonal line represents the line of no discrimination (AUC = 0.5). Ea, effective arterial elastance; AUC, area under the curve; CI, confidence interval.

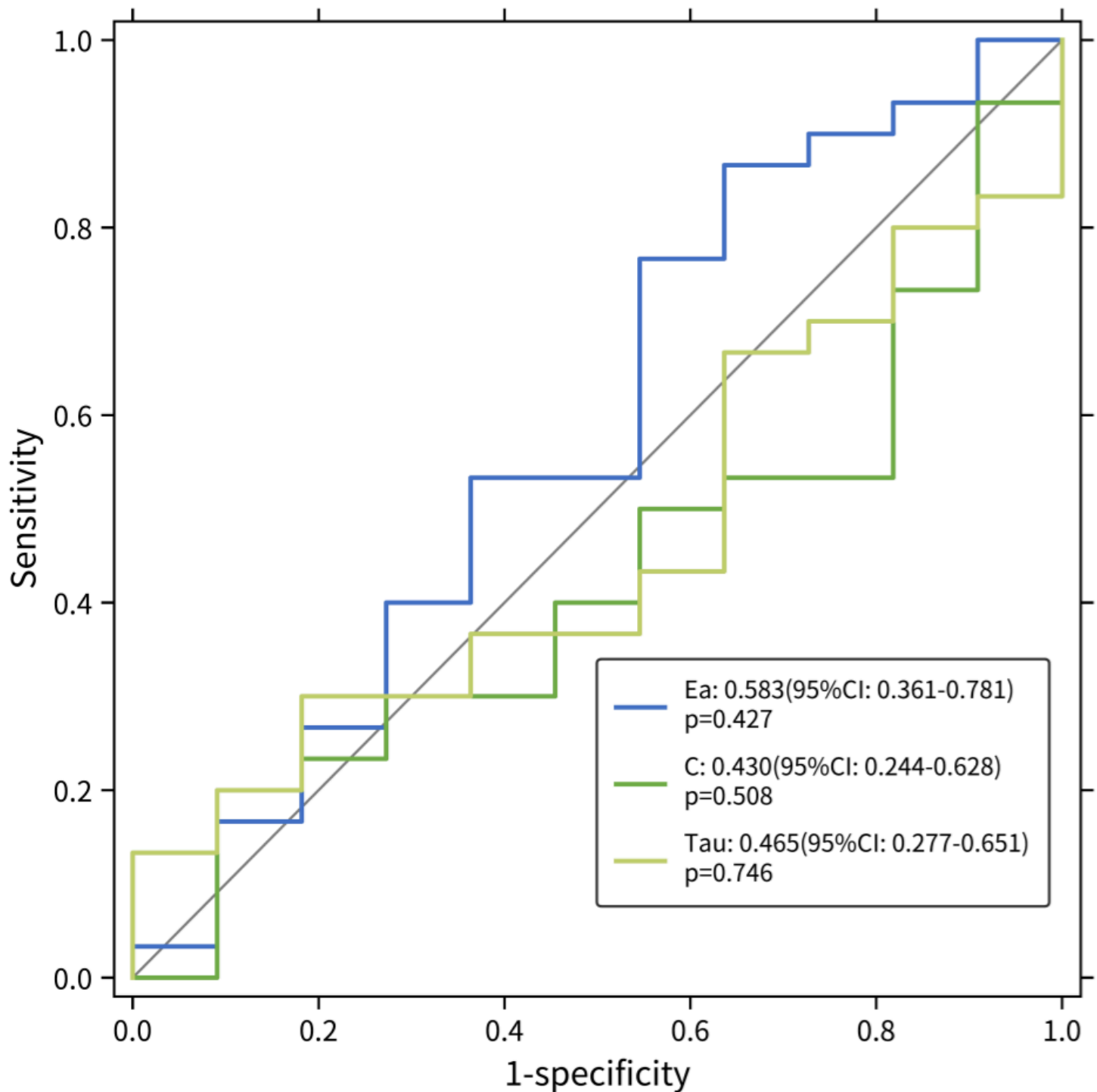


Figure 2

Receiver operating characteristic (ROC) curves of arterial load parameters for predicting arterial pressure responsiveness. The diagnostic performance of baseline effective arterial elastance (Ea), arterial compliance (C), and arterial time constant (Tau) was evaluated. The area under the curve (AUC) with 95% confidence interval (CI) and p value for each parameter is shown in the legend. The diagonal grey line represents the reference line of no discrimination (AUC = 0.5). None of the three indices demonstrated statistically significant predictive value (Ea: AUC = 0.583, 95% CI: 0.361–0.781, p = 0.427;

C: AUC = 0.430, 95% CI: 0.244–0.628, $p = 0.508$; Tau: AUC = 0.465, 95% CI: 0.277–0.651, $p = 0.746$). Ea, effective arterial elastance; C, arterial compliance; Tau, arterial time constant; AUC, area under the curve; CI, confidence interval.

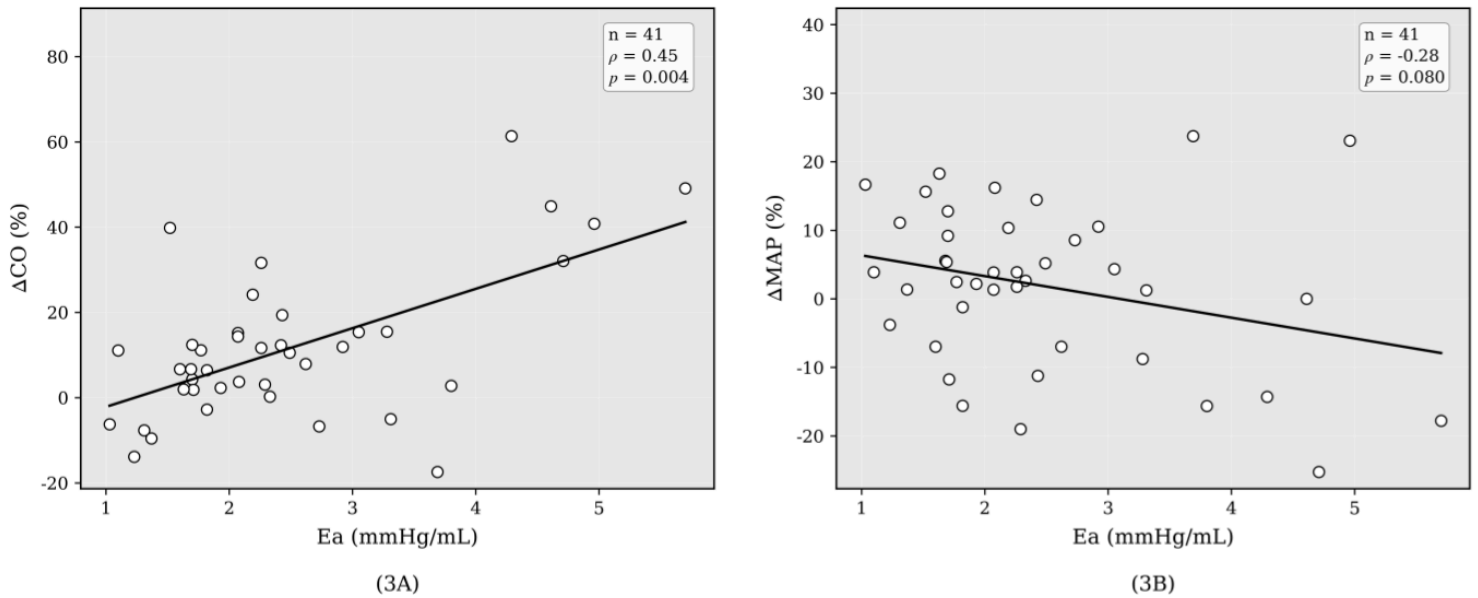


Figure 3

Correlation between baseline effective arterial elastance (Ea) and hemodynamic changes after the fluid challenge in septic shock. (3A) Scatter plot showing the correlation between baseline Ea and percentage change in cardiac output ($\Delta CO\%$) after the fluid challenge. Spearman's rank correlation analysis showed a moderate positive correlation ($\rho = 0.45$, $p = 0.004$, $n = 41$). (3B) Scatter plot showing the correlation between baseline Ea and percentage change in mean arterial pressure ($\Delta MAP\%$) after the fluid challenge. A weak negative correlation was observed but did not reach statistical significance ($\rho = -0.28$, $p = 0.080$, $n = 41$). Ea, effective arterial elastance; $\Delta CO\%$, percentage change in cardiac output; $\Delta MAP\%$, percentage change in mean arterial pressure.

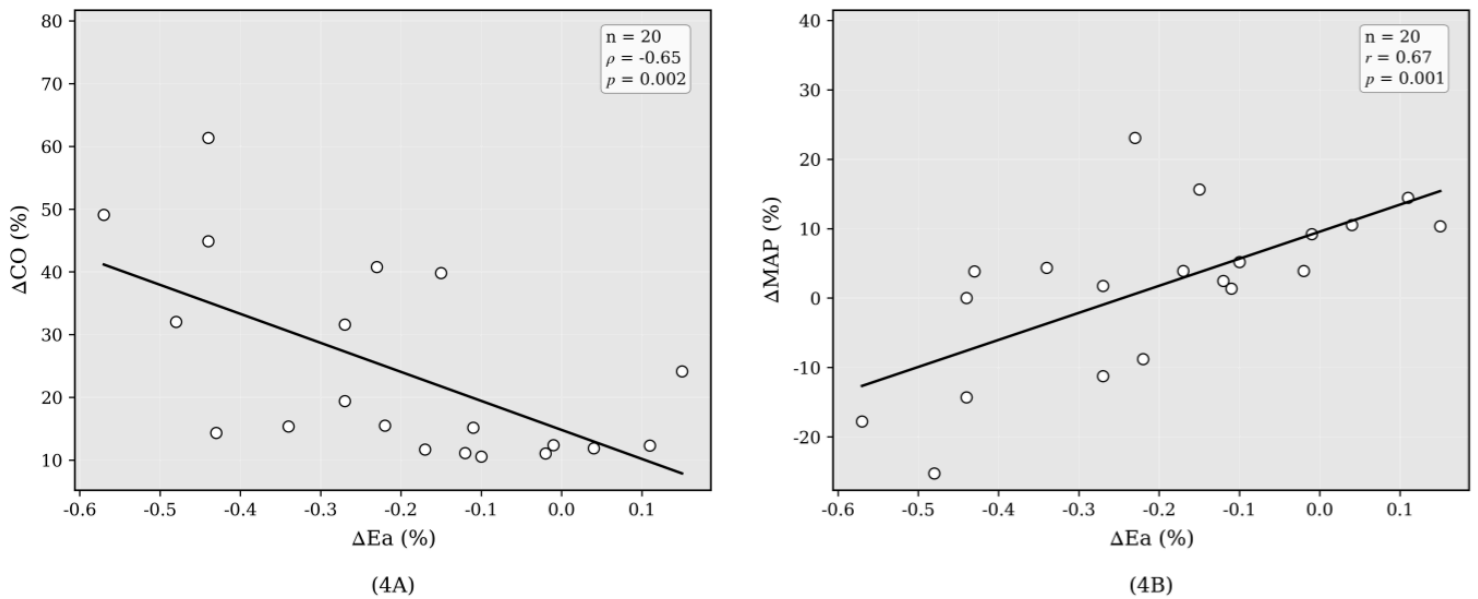


Figure 4

Correlation between percentage change in effective arterial elastance (ΔEa %) and hemodynamic changes after the fluid challenge in fluid responders. (4A) Scatter plot showing the correlation between ΔEa % and percentage change in cardiac output (ΔCO %) after the fluid challenge. Spearman's rank correlation analysis showed a strong negative correlation ($\rho = -0.65$, $p = 0.002$, $n = 20$). (4B) Scatter plot showing the correlation between ΔEa % and percentage change in mean arterial pressure (ΔMAP %) after the fluid challenge. Pearson's correlation analysis showed a strong positive correlation ($r = 0.67$, $p = 0.001$, $n = 20$). ΔEa %, percentage change in effective arterial elastance; ΔCO %, percentage change in cardiac output; ΔMAP %, percentage change in mean arterial pressure.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Tables.docx](#)