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ORIGINAL RESEARCH

# Dynamic arterial elastance for predicting mean arterial pressure response to fluid administration in hypotensive critically ill surgical patients: Prospective observational study

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## ABSTRACT:

**Background:** Postoperative hypotension frequently occurs in high-risk surgical patients and is associated with elevated risks of adverse cardiovascular outcomes. Dynamic arterial elastance ($Ea_{dyn}$), calculated as the ratio of pulse pressure variation (PPV) to stroke volume variation (SVV), has been proposed as a functional parameter to guide fluid resuscitation by reflecting arterial load.

**Method:** This prospective observational study was conducted in a single tertiary care center and enrolled postoperative patients who developed hypotension requiring fluid resuscitation. Hemodynamic variables were measured immediately before and after fluid administration. PPV was recorded through an invasive arterial catheter, while SVV was assessed using an internally calibrated pulse contour analysis device. The primary objective was to determine $Ea_{dyn}$'s predictive performance in predicting mean arterial pressure (MAP) responsiveness following fluid loading. Secondary analyses compared $Ea_{dyn}$'s predictive performance with systemic vascular resistance (SVR) and the diastolic shock index (DSI).

**Result:** Fifty-one patients were included, and 73 hypotensive interventions were analyzed, of which 55 were classified as MAP responders. An $Ea_{dyn}$ threshold of ≥ 1.02 yielded an area under the receiver operating characteristic curve (AUC) of 0.802 (95% CI, 0.703-0.901), with a sensitivity of 56.4% and a specificity of 100%. In contrast, SVR and DSI demonstrated poor discriminatory ability, with AUCs of 0.548 and 0.565, respectively. $Ea_{dyn}$ significantly outperformed both indices (p = 0.003).

**Conclusion:** $Ea_{dyn}$ is demonstrated as a potential reliable hemodynamic index for predicting MAP responsiveness following fluid administration in mechanically ventilated postoperative patients with fluid-responsive hypotension.

**Trial registration:** TCTR20240330001

**Keywords:** Dynamic arterial elastance; Mean arterial pressure; Hemodynamic monitoring; Fluid responsiveness; Postoperative hypotension

## INTRODUCTION

Postoperative hypotension has been identified as a significant factor associated with an increased risk of myocardial infarction and mortality [1]. This risk can be mitigated through optimal preoperative conditioning and adequate resuscitation [2]. Postoperative hypotension can result from multiple factors, including hypovolemia or severe inflammatory response [3].

According to Ohm's law, the relationship of hemodynamic parameters can be described in the MAP - CVP = CO x SVR equation. Both cardiac output (CO) and SVR, which determine the afterload in this equation, can contribute to the change in MAP. This relationship is one of the explanations that abnormal vasomotor tone results in an inappropriate increase of MAP despite adequate CO [4]. Although most studies represented afterload as SVR, this parameter only accounts for static afterload in peripheral arterioles. Another component of afterload is dynamic arterial afterload from the aorta [5]. Arterial afterload measurement can be conducted based on the principle of effective arterial elastance [6] (Ea), which represents the line connecting the left ventricular end-diastolic pressure-volume point through the end-systolic pressure-volume point of the pressure-volume loop [7] .

Ea is calculated as end-systolic pressure divided by stroke volume. However, its direct measurement may be challenging in clinical settings. $Ea_{dyn}$ is defined as PPV divided by SVV, which represents the dynamic interaction of pressure and volume change in the arterial system [8]. $Ea_{dyn}$ can help predict the change in arterial pressure after fluid administration in fluid-responsive patients.

From the study by Bar et al., $Ea_{dyn}$ was used as a guide when reducing the dose of norepinephrine (NE) after stable hemodynamics, which has a beneficial outcome on decreased duration of NE and ICU length of stay [9]. Furthermore, a systematic review and meta-analysis by Zhou et al. analyzed the effectiveness of $Ea_{dyn}$ in predicting pressure responsiveness after fluid administration, which guides the decision to increase fluid bolus or initiate vasopressor as NE [4]. These studies included septic shock, hypovolemic shock in medical ICUs, and perioperative hypotension during surgical procedures [10]. The threshold of $Ea_{dyn}$ in this study varied from 0.65 to 0.89, with different sensitivity and specificity.

Furthermore, the study by Ospina Tascón et al. found that DSI, defined as heart rate (HR) divided by diastolic blood pressure (DBP) [11], can also be a good parameter to determine the severity of vasoplegic shock and determine the initiation of vasopressor [12]. Our study aims to assess the ability of $Ea_{dyn}$ to predict MAP response after fluid administration of postoperative hypotension in critically ill surgical patients and to compare the predictive capabilities of various afterload evaluation modalities, including $Ea_{dyn}$, SVR, and DSI, in assessing pressure responsiveness.

### KEY MESSAGES:

- $Ea_{dyn}$ effectively predicted MAP responsiveness to fluid bolus in fluid-responsive postoperative mechanically ventilated patients with hypotension
- The $Ea_{dyn}$ cut-off value in hypotensive postoperative mechanically ventilated patients exhibits a high positive predictive value, supporting its utility as a diagnostic criterion for ruling patients in this study.
- SVR and DSI demonstrated lower predictive ability in distinguishing MAP responsiveness from non-responsiveness in this population.

## MATERIALS AND METHODS

### Study design

Prospective observational study

### Study setting

Surgical intensive care unit (ICU) of King Chulalongkorn Memorial Hospital.

### Eligibility criteria

### Inclusion criteria

1. Age 18 years or older
2. High-risk surgical patient (as defined by The European Society of Cardiology Guidelines for the assessment and management of cardiovascular patients undergoing non-cardiac surgery in 2022 [13])
3. Postoperative hypotension: defined as MAP ≤ 65 mmHg (patient may receive prior resuscitation with norepinephrine dose ≤ 0.05 mcg/kg/min)
4. Controlled mechanical ventilation (TV ≥ 8 ml/kg predicted body weight)
5. The predictor of fluid responsiveness is defined as pulse pressure variation (PPV) ≥ 12%

### Exclusion criteria

1. Cardiac arrhythmia
2. Ratio of heart rate (HR) to respiratory rate (RR) < 3.6
3. HR < 60 beats per minute (bpm)
4. The patient underwent cardiothoracic surgery.
5. Increase intraabdominal pressure
6. Acute respiratory distress syndrome
7. Right heart failure
8. Valvular heart disease
9. Significant myocardial disease
10. Pulmonary edema
11. Pregnancy

### Recruitment and consent

The investigator informed and obtained consent from patients who underwent high-risk surgery on the day of admission before the procedure. Standard treatment was provided regardless of whether the patient consented to participate in the trial or declined. In emergency surgery cases, the investigator informed and obtained consent from the legally authorized representative.

### Intervention

After the patient or the legally authorized representative consented, the patient underwent surgery and received standard care. The intervention took place upon the patient's arrival in the ICU and the development of postoperative hypotension. The hemodynamic parameters were recorded before and after fluid administration.

### Research procedure

1. High-risk surgical patients who underwent noncardiac surgery and were admitted to the surgical ICU were continuously monitored for hemodynamic parameters using a central venous catheter and an arterial catheter as part of standard treatment. These catheters were connected to an internally-calibrated cardiac output monitoring system (FloTrac sensor on EV1000 monitor, Edwards Lifesciences, Irvine, CA, USA) and an Intellivue MX700 bedside vital sign monitor (Philips, Petaluma, CA, USA). Hemodynamic parameters were measured realistically and recorded for 48–72 hours.

2. When patients developed postoperative hypotension within 48 hours after surgery, initial resuscitation was performed using norepinephrine at a dose of ≤ 0.05 μg/kg/min as part of standard care. Norepinephrine was initiated within the first hour of resuscitation after adequate fluid resuscitation had been performed, particularly in patients who were fluid non-responsive or showed signs of fluid overload.

3. Pulse pressure variation (PPV) was used to predict fluid responsiveness. A positive result was defined as PPV ≥12%. PPV was selected for this study due to its availability as a routine bedside monitoring parameter and its high diagnostic accuracy, with a reported sensitivity of 88% and specificity of 89%, as demonstrated in a review by Monnet et al. [14].

4. Hemodynamic parameters were recorded before fluid administration, including PPV, SVV, SBP, DBP, MAP, HR, SVR, SVI and SVRI

5. Within 15 minutes, a 250 ml infusion of 5% albumin was administered. The hemodynamic parameters were recorded immediately after fluid administration and every 1 minute until 5 minutes after fluid administration, as shown in the flow diagram (Figure 1).

6. Cardiac output was evaluated by transthoracic echocardiography by measuring the velocity-time integral (LVOT VTI) and the left ventricular outflow tract diameter (LVOT diameter) before and after fluid administration.

7. Five critical care fellows participated in the study. All fellows underwent training for standardized measurement, and their measurements were verified by a board-certified cardiac intensivist before data collection. Furthermore, all images and measurements taken during data collection were recorded for subsequent review by the cardiac intensivist.

8. MAP responsiveness was defined as an increase in MAP of ≥15% following fluid administration, accompanied by an increase in cardiac output of ≥10%. MAP non-responsiveness was defined as an increase in the maximum MAP within 5 minutes post-fluid administration of <15%, despite an increase in cardiac output of ≥10%

9. $Ea_{dyn}$ and DSI were calculated by reviewing the record of hemodynamic parameters at a similar time.

### Outcome measurement

### Primary objective

Determine the ability of $Ea_{dyn}$ to predict the response of MAP after fluid administration of postoperative hypotension in critically ill, mechanically ventilated surgical patients.

### Secondary objective

Compare the ability of $Ea_{dyn}$, SVR, and DSI to predict MAP responsiveness after fluid administration. Another secondary outcome is to analyze the total amount of fluid resuscitation in postoperative hypotension patients who receive $Ea_{dyn}$ monitoring.

### Statistical analysis

The sample size was determined using Kappa-based estimation instead of conventional sensitivity and specificity calculations, as no prior studies have reported sensitivity or specificity in a comparable population of mechanically ventilated postoperative patients with hypotension. The Kappa in the range of intermediate to good agreement [15, 16] (Kappa 0.4-0.75) was applied. The sample size of 73 patients was calculated with a proportion of the outcome of 0.6 as in the previous study [15], a type I error of 0.05, a desired power of 0.8, and an expected dropout rate of 10%.

Data analysis was performed with Stata version 18 (StataCorp LLC, College Station, TX, USA). Categorical data were reported as percentages and numbers, while continuous data were presented as mean and standard deviation or median and interquartile range, depending on the data distribution. Comparisons of proportions between two groups were made using the Chi-square test or Fisher's exact test, as appropriate. $Ea_{dyn}$'s ability to predict MAP responsiveness was analyzed using AUC and ROC curve. Sensitivity and specificity were also examined to validate the $Ea_{dyn}$ threshold. The positive predictive value (PPV) and negative predictive value (NPV) were assessed to determine the efficacy of the $Ea_{dyn}$ threshold. This threshold was tested using the Kappa statistic to confirm the degree of reliability, with values ranging from 0.4 to 0.75, indicating intermediate to good agreement. The predictive ability of $Ea_{dyn}$, SVR, and DSI for MAP responsiveness was compared using the ROC comparison test.

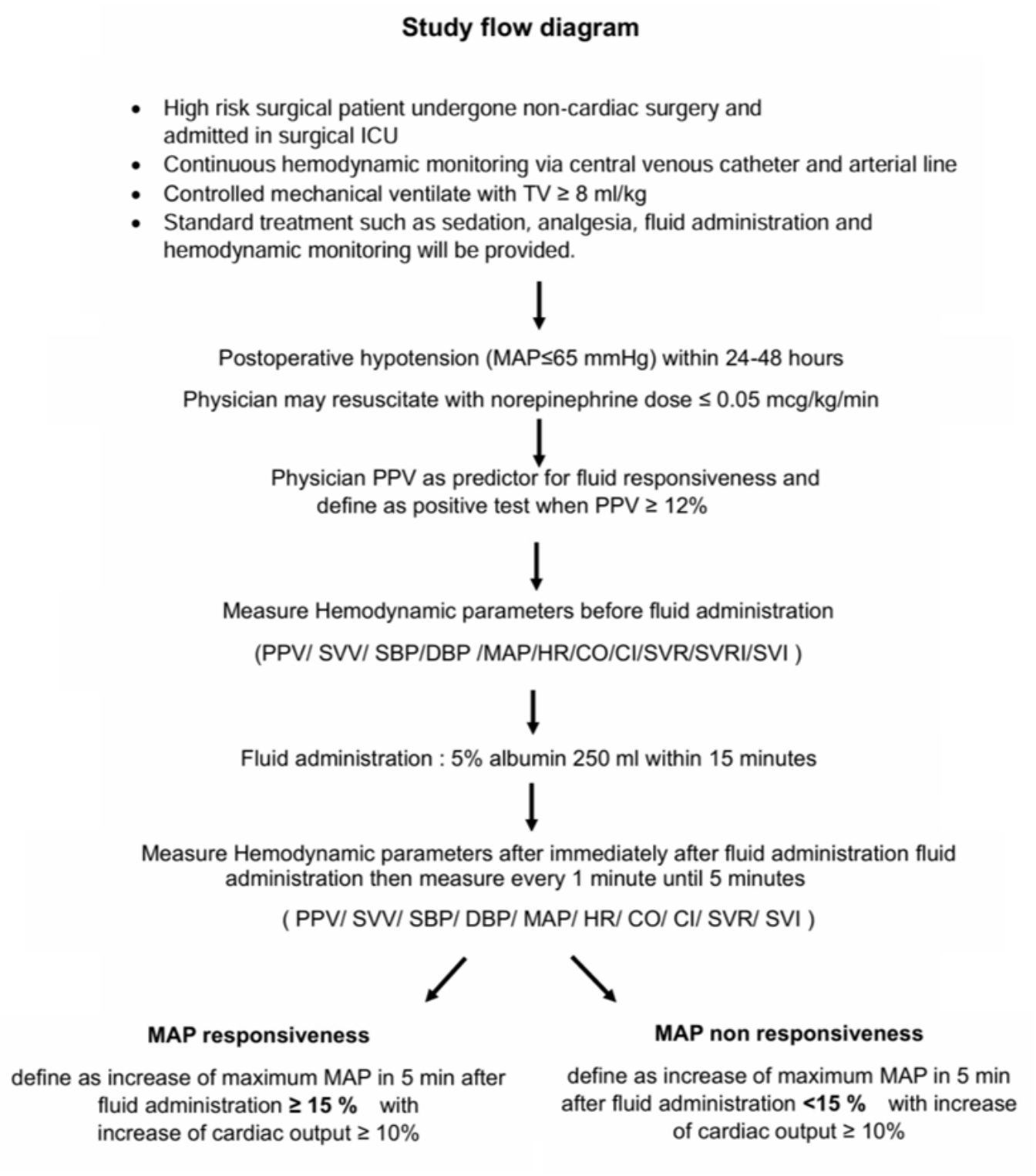



**Figure 1.** Study flow diagram

## RESULTS

Fifty-one patients were enrolled in the study, comprising 73 interventions. Of these, 55 interventions were classified as MAP-responsive following fluid administration, as illustrated in Figure 2. Baseline characteristics were not significantly different between the MAP-responsive and MAP-nonresponsive groups, as shown in Table 1. Except for $Ea_{dyn}$ (1.0 [1.0–1.1] vs. 0.9 [0.9–1.0]; $p < 0.001$) and PPV (15.7 [14.7–16.6]% vs. 13.2 [12.0–14.4]%; $p = 0.002$), no statistically significant differences were observed in baseline hemodynamic parameters before fluid administration, as presented in Table 2.

For the primary outcome, $Ea_{dyn}$ values ≥ 1.02 effectively differentiated MAP responders from non-responders, with of 0.802 (0.703–0.901), indicating acceptable discriminatory performance, as illustrated in Figure 3.The threshold $Ea_{dyn}$ value of ≥ 1.02 demonstrated a sensitivity of 56.4 % (42.3-69.7) and a specificity of 100 % (81.5-100), confirming its validity. The PPV was 100%,while the NPV was 42.9 %, indicating the efficacy of the threshold. The kappa statistic showed fair agreement, with a value of 0.389 (0.231 - 0.548).

The study identified a "grey zone" for $Ea_{dyn}$, defined as the range of 0.94–1.04, in which MAP responsiveness could not be reliably differentiated. In this study, 33 cases (45.2%) fell within this grey zone, as illustrated in Figure 4.

For the secondary outcomes, there were no statistically significant differences in SVR or the DSI between the MAP-responsive and MAP-non-responsive groups. The SVR in the MAP-responsive group was 1121.7 (1025.4-1218), compared to 1063.7 (952.9-1174.4) in the MAP-non-responsive group ($p = 0.39$). DSI was 1.9 (1.7-2) in the MAP-responsive group and 1.7 (1.6-1.7) in the MAP-non-responsive group ($p = 0.36$).

SVR and DSI exhibited limited predictive capacity, with AUCs of 0.548 (0.401–0.695) and 0.565 (0.434–0.697), respectively. While the overall predictive performance of $Ea_{dyn}$ was 0.802 (0.703-0.901), comparison of the ROC curves for $Ea_{dyn,}$ SVR, and DSI demonstrated statistically significant differences among the three parameters (overall p-value = 0.003), as illustrated in Figure 5.

We observed a trend of higher fluid administration in the MAP-non-responsive group, which may increase the risk of fluid overload. The volume of fluid administered was significantly higher in the MAP non-responsive group (319 ml, 250 (262.1-376.7) mL; $p< 0.001$), as detailed in Table 3.

The percentage increase in CO after fluid bolus, representing fluid responsiveness, did not differ significantly between the MAP-responsive and MAP-nonresponsive groups (19.6 [16.3–22.9]% vs. 14.0 [11.2–16.9]%; $p = 0.06$) as illustrated in Figure 6.The MAP-responsive group did not observe the highest MAP immediately following fluid administration. The peak MAP values were recorded 2 minutes (1-3) post-administration, as illustrated in Figure 6.

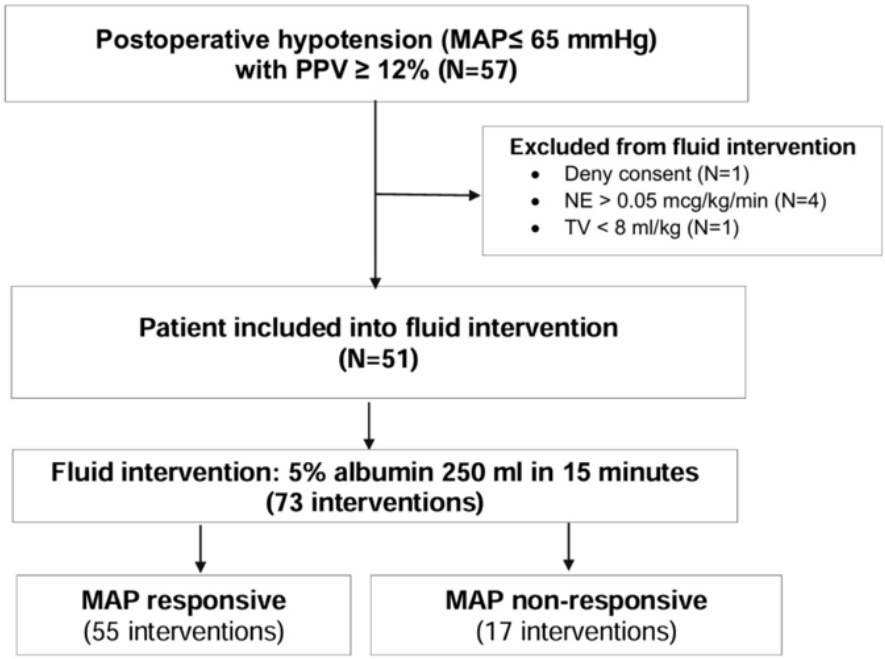



**Figure 2.** Flow chart of the included fluid intervention

**Table 1.** Baseline characteristics

| Variable | Total (N=51) | MAP response (N=43) | MAP non-response (N=8) | P-value |
|---|---|---|---|---|
| Age (yr), Mean ±SD | 65.3±13.5 | 65.9±13.5 | 62±14.3 | 0.46 |
| Sex, N (%) | | | | 0.86 |
| • Male | 24 (47.1) | 20 (46.5) | 4 (50) | |
| • Female | 27 (52.9) | 23 (53.5) | 4 (50) | |
| BMI (kg/m$^2$), Mean ±SD | 23.8±4.8 | 23.9±4.7 | 23.0±5.8 | 0.63 |
| APACHE II score, Mean ±SD | 16.6±5.1 | 16.9±5.1 | 15.3±4.9 | 0.41 |
| Comorbid, N (%) | | | | |
| • Hypertension | 33 (62.8) | 27 (62.8) | 6 (75) | 0.69 |
| • Dyslipidemia | 20 (39.2) | 16 (37.2) | 4 (50) | 0.70 |
| • Diabetes | 17 (33.3) | 13 (30.2) | 4 (50) | 0.42 |
| • Cancer | 15 (29.4) | 11 (25.6) | 4 (50) | 0.21 |
| • Ischemic heart disease | 4 (7.8) | 2 (4.7) | 2 (25) | 0.11 |
| • Stroke | 2 (3.9) | 2 (4.7) | 0 (0) | 0.53 |
| • Renal disease | 2 (3.9) | 2 (4.7) | 0 (0) | 0.53 |
| • Other | 5 (9.8) | 4 (9.3) | 1 (12.5) | 0.78 |
| • No co-morbidity | 7 (13.7) | 7 (16.3) | 0 (0) | 0.58 |
| Urine output (ml/kg/hour), Mean ±SD | 0.44±0.18 | 0.44±0.19 | 0.41±0.12 | 0.71 |
| Type of shock, N (%) | | | | |
| • Hypovolemic shock | 27 (54) | 22 (52.4) | 5 (62.5) | 0.71 |
| • Distributive shock | 23(46) | 20 (47.6) | 3 (37.5) | 0.71 |
| Receive vasopressor: Norepinephrine, N (%) | | | | |
| • Yes | 30 (58.8) | 27 (62.8) | 3 (37.5) | 0.25 |
| • No | 21 (41.2) | 16 (32.7) | 5 (62.5) | 0.25 |
| Type of surgical operation, N | | | | |
| • Gastrointestinal | 20 (39.2) | 15 (34.9) | 5 (62.5) | 0.14 |
| • Colorectal | 13 (25.5) | 12 (27.9) | 1 (12.5) | 0.36 |
| • Urology | 4 (7.8) | 4 (9.3) | 0 (0) | 0.40 |
| • Gynecology | 2 (3.9) | 2 (4.7) | 0 (0) | 0.52 |
| • Vascular | 5 (9.8) | 4 (9.3) | 1 (12.5) | 0.78 |
| • Orthopedic | 3 (5.8) | 3 (7.8) | 0 (0) | 0.44 |
| • Hepatobiliary | 2 (3.9) | 2 (4.7) | 0 (0) | 0.53 |
| Sedation, Mean ±SD | | | | |
| • Fentanyl (mcg/kg/hr) | 24.1 ±8.6 | 24.1±9.1 | 24.2 ±6.6 | 0.99 |
| • Propofol (mg/kg/hr) | 40.5±25.5 | 39.2 ±25 | 50 ±30 | 0.38 |
| • Dexmedetomidine | 0.005±0.001 | 0.005±0.001 | 0.005±0.001 | 0.53 |
| Ventilator setting | | | | |
| Mode, N (%) | 72 (98.7) | 54 (98.2) | 18 (100) | 0.57 |
| Respiratory rate, Mean ±SD | 13±2.3 | 13.1±2.4 | 12.3±0.7 | 0.31 |
| Inspiratory pressure, Mean ±SD | 13.6±1.4 | 13.6±1.5 | 13.6±1.1 | 0.99 |
| PEEP, Mean ±SD | 5.8±1.6 | 5.8±1.7 | 5.8±1.4 | 0.92 |
| $FiO_2$, Mean ±SD | 0.41±0.06 | 0.41±0.06 | 0.41±0.04 | 0.89 |

**Abbreviations:** MAP: mean arterial pressure

**Table 2.** Baseline hemodynamic parameters before fluid bolus.

| Hemodynamic parameters (Mean (95%CI)) | MAP response (N=55) | MAP non-response (N=18) | P-value |
|---|---|---|---|
| SBP : Systolic blood pressure (mmHg) | 87.6 (84-91.2) | 93.4 (87.1-99.8) | 0.02 |
| DBP : Diastolic blood pressure (mmHg) | 46 (44.1-47.9) | 46.6 (44.8-48.3) | 0.38 |
| HR: Heart rate (bpm) | 83.4 (78-88.7) | 77 (71.3-82.7) | 0.95 |
| DSI (Diastolic shock index) | 1.9 (1.7-2) | 1.7 (1.6-1.7) | 0.36 |
| CO: Cardiac output (L/min) | 4 (3.5-4.4) | 4.1 (3.6-4.5) | 0.73 |
| CI : Cardiac index (L/min/$m^2$) | 2.6 (2.2-2.9) | 2.8 (2.4-3.2) | 0.50 |
| SVV : Stroke volume variation (%) | 15.1 (14.2-15.9) | 14.2 (12.9-15.5) | 0.48 |
| PPV: Pulse pressure variation (%) | 15.7 (14.7-16.6) | 13.2 (12-14.4) | 0.002 |
| $Ea_{dyn}$: Dynamic arterial elastance | 1 (1-1.1) | 0.9 (0.9-1) | <0.001 |
| SVR : Systemic vascular resistance (dynes/sec/$cm^{-5}$) | 1121.7 (1025.4-1218) | 1063.7 (952.9-1174.4) | 0.39 |
| SVRI : Systemic vascular resistance index (dynes/sec/ $m^2$/$cm^{-5}$) | 1790.1 (1632.8-1947.4) | 1583.7 (1369.1-1798.3) | 0.19 |
| SVI: Stoke volume index (ml/ $m^2$) | 32.8 (28.6-37.1) | 36.9 (31-42.8) | 0.38 |

**Abbreviations:** MAP: mean arterial pressure

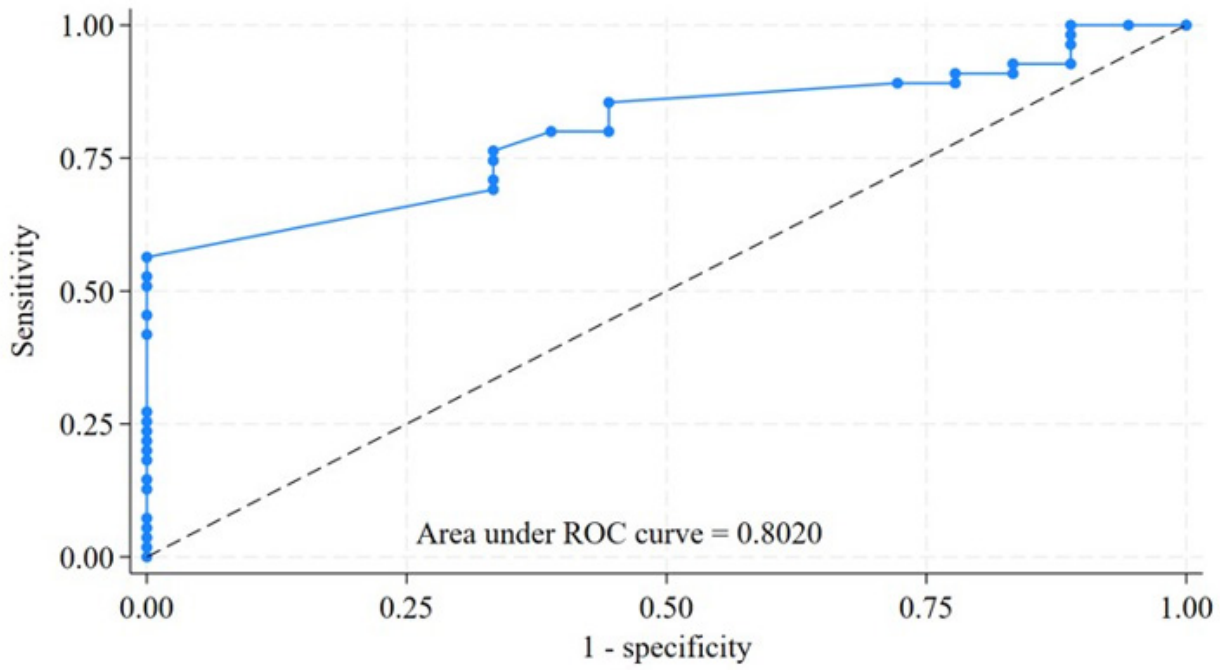
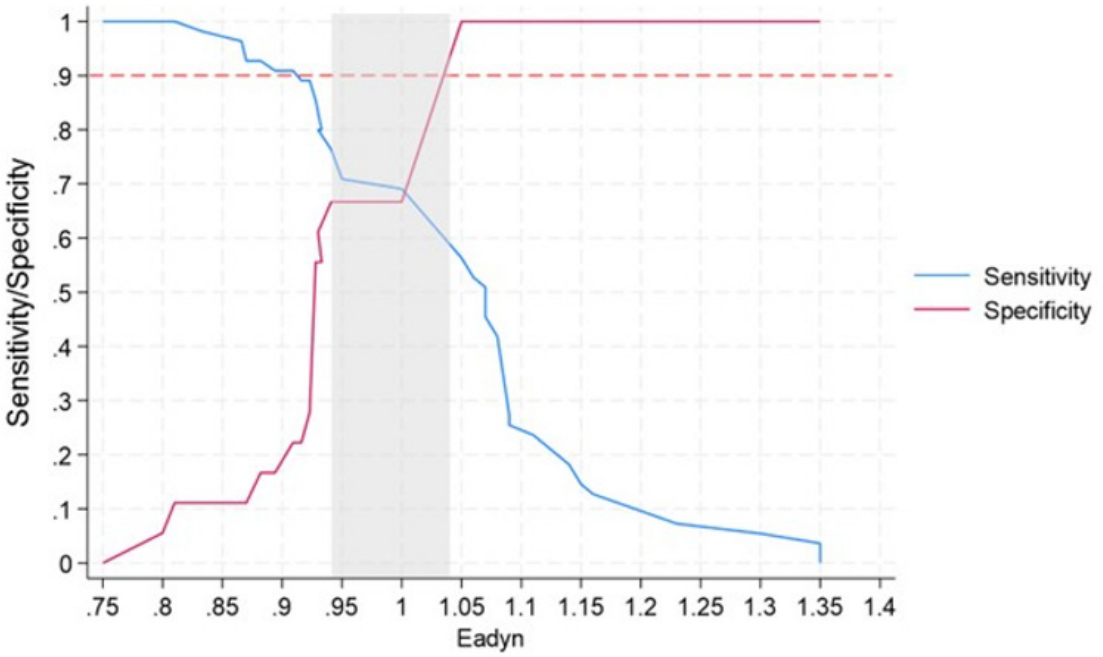


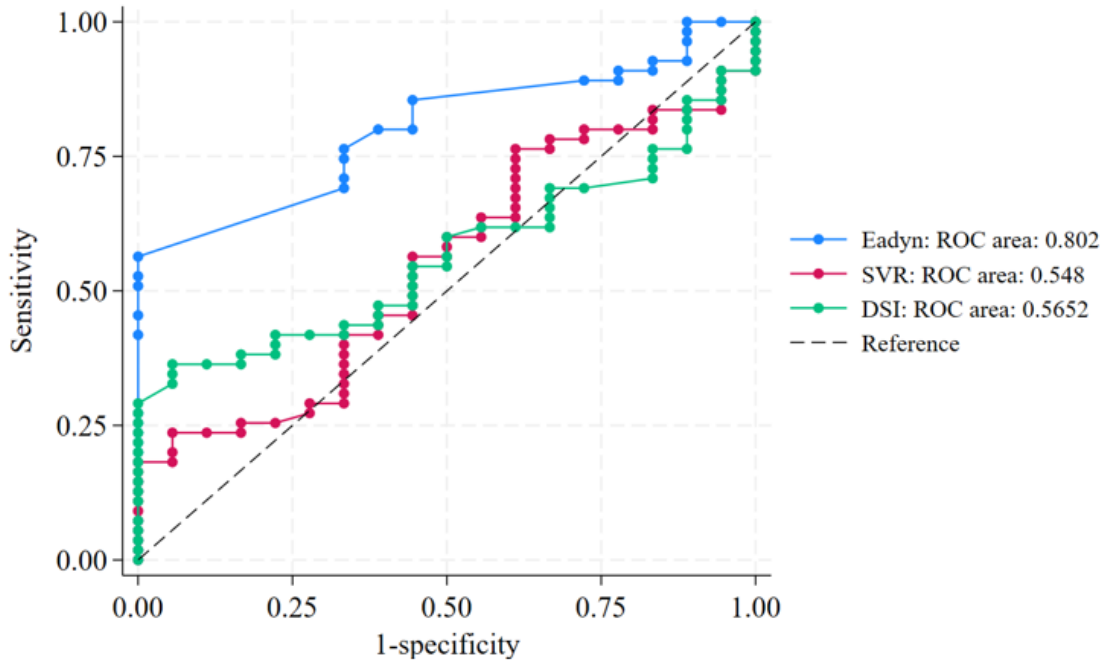

**Figure 3.** Receiver operating characteristic (ROC) analysis of dynamic arterial elastance ($Ea_{dyn}$).
Kappa (95% CI) = 0.389 (0.231 - 0.548) for $Ea_{dyn} \geq 1.02$
**Abbreviations:** AUC: Area under the curve; NPV: Negative Predictive Value; PPV: Positive Predictive Value




**The grey zone of $Ea_{dyn}$ was 0.94-1.04**

| | N | % |
|---|---|---|
| <0.94 | 9 | 12.3 |
| 0.94-1.04 | 33 | 45.2 |
| >1.04 | 31 | 42.47 |

**Figure 4.** Grey zone of dynamic arterial elastance ($Ea_{dyn}$).
The grey zone of $Ea_{dyn}$ was 0.94-1.04



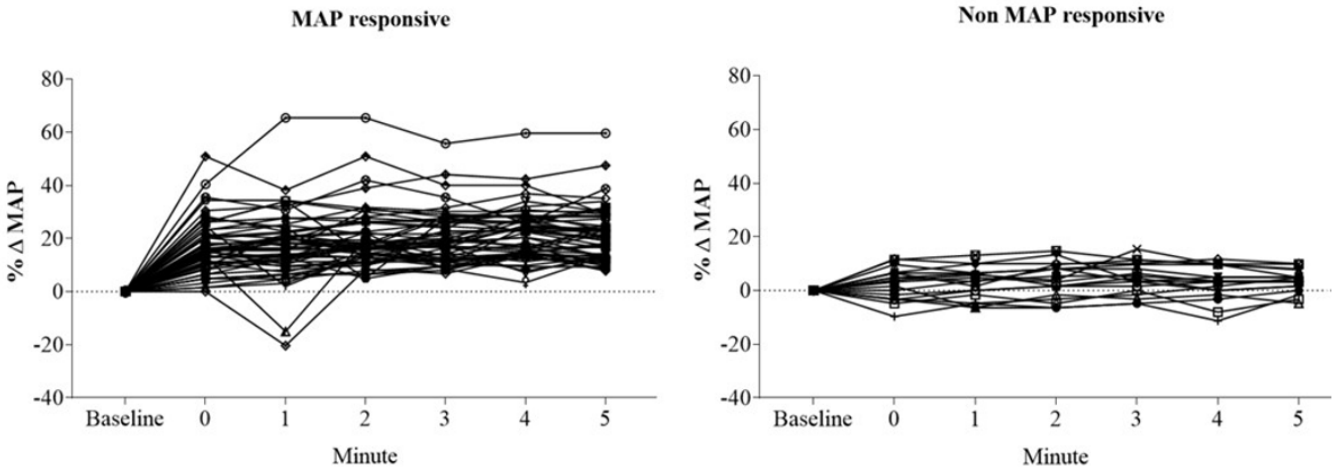

| | ROC area under the curve | 95%CI | P-value | Overall P-value |
|---|---|---|---|---|
| $Ea_{dyn}$ | 0.802 | 0.703-0.901 | ref | 0.003 |
| DSI | 0.565 | 0.434-0.697 | 0.008 | |
| SVR | 0.548 | 0.401-0.695 | 0.008 | |

**Figure 5.** Comparison of receiver operating characteristic (ROC) analysis between $Ea_{dyn}$, SVR, and DSI.
**Abbreviations:** $Ea_{dyn}$ : Dynamic Arterial Elastance; DSI: Diastolic Shock Index; SVR: Systemic Vascular Resistance

**Table 3.** Amount of fluid administration in MAP responsive group and MAP non-responsive group.

| | MAP non-response (N=18) | MAP response (N=55) | P-value |
|---|---|---|---|
| Amount of fluid (ml) | 319 .4 (262.1-376.7) | 250 (250-250) | < 0.001 |

**Abbreviations:** MAP: mean arterial pressure




| Hemodynamic parameters (Mean (95%CI)) | MAP response (N=55) | MAP non-response (N=18) | P-value |
|---|---|---|---|
| Duration of Δ Maximum MAP, median (IQR) (minutes) | 2 (1-3) | 3 (1-4) | 0.17 |
| % Δ Maximum MAP from baseline | 25.4 (22.7-28.1) | 6.7 (4.0-9.3) | <0.001 |
| CO at baseline (L/min) | 4 (3.5-4.4) | 4.1 (3.6-4.5) | 0.73 |
| % CO change from baseline | 19.6 (16.3-22.9) | 14.0 (11.2-16.9) | 0.06 |

**Figure 6.** MAP responsiveness after fluid administration.
**Abbreviations:** % ΔMaximum MAP: percentage of MAP change between before and after fluid bolus; % CO change: percent of cardiac output change between before and after the fluid bolus

## DISCUSSION

The main findings of our study indicate that an $Ea_{dyn}$ value ≥ 1.02 effectively differentiates MAP responsiveness from non-responsiveness in postoperative mechanically ventilated patients with hypotension, with an AUC of 0.802, a sensitivity of 56.4%, and a specificity of 100%. These results suggest that $Ea_{dyn}$ may be a useful diagnostic tool to determine the fluid bolus in fluid-responsive postoperative mechanically ventilated patients with hypotension.

The findings of our study are consistent with previous research by Monge García et al., which reports that the $Ea_{dyn}$ value >0.89 predicted an increase of 15% in MAP after fluid administration with a sensitivity of 93.75% and a specificity of 100% in mechanically ventilated hypotensive patients with acute circulatory failure in the medical and surgical ICU. Their study concluded that $Ea_{dyn}$ is a valuable predictor of MAP changes in response to fluid expansion. It also emphasized that initial hemodynamic resuscitation should optimize CO and MAP to ensure adequate perfusion pressure and organ perfusion [5].

However, our results did not align with the systematic review and meta-analysis of Zhou et al., which demonstrated a higher precision of $Ea_{dyn}$ in the medical ICU [4]. They assumed that the effectiveness of $Ea_{dyn}$ depends on arterial compliance. In the medical ICU, most patients receive vasopressors, whereas surgical patients are generally healthier, with normal cardiac and vascular function. Surgical patients typically present with hypovolemic shock and do not necessarily require vasopressors.

The high accuracy of $Ea_{dyn}$ observed in postoperative patients in our study may be attributed to including both hypovolemic and distributive shock cases. Additionally, a significant proportion of our study population (58%) received vasopressors, which may have influenced the accuracy of $Ea_{dyn}$.

These findings suggest that $Ea_{dyn}$ may be used in combination with other hemodynamic parameters to support clinical decision-making regarding fluid bolus administration in postoperative hypotensive patients, as highlighted in the systematic review and meta-analysis by Zhou et al.[4]

Our study also analyzed the grey zone of $Ea_{dyn}$ and identified that values between 0.94 and 1.04 were inconclusive in distinguishing between MAP responsiveness and non-responsiveness. Fewer than 50% of cases (45.2%) fell within this grey zone, supporting its diagnostic accuracy. The grey zone is defined as a range in which sensitivity and specific-

ity are below 90%, with a diagnostic tolerance of 10% [17]. Our findings are consistent with the study by Monge et al., which reported a grey zone for $Ea_{dyn}$ ranging from 0.72 to 0.88 in predicting MAP response after fluid administration in mechanically ventilated patients with acute circulatory failure [18]. The clinical application of $Ea_{dyn}$ when values fall within the grey zone provides valuable guidance for clinicians in interpreting this index in combination with other hemodynamic parameters in clinical practice and highlights the need for additional information when making clinical decisions[19].

SVR and DSI demonstrated limited predictive value for MAP responsiveness, with AUCs of 0.548 and 0.565, respectively. In contrast, ROC curve analysis revealed that $Ea_{dyn}$ exhibited the highest overall predictive performance, with an AUC of 0.802.

SVR and DSI are commonly used to predict the need for vasopressors in vasodilatory shock, particularly in septic shock. A study by Ospina-Tascón et al. demonstrated an association between increased DSI and a higher risk of mortality in septic shock patients. This relationship is based on the pathophysiology of DBP, which depends on vascular tone and the duration of the cardiac cycle. Therefore, the combination of DBP and HR may reflect the severity of circulatory dysfunction in vasodilatory states [20].

However, in critically ill surgical patients, circulatory instability is often multifactorial, involving not only vasodilation but also hypovolemia and the effects of anesthetic agents [3]. $Ea_{dyn}$ more accurately reflects the dynamic elastic properties of the vascular wall than SVR [21]. Monge et al. demonstrated that $Ea_{dyn}$ is a superior predictor of MAP response to fluid challenge in circulatory shock compared to SVR [18]. These additional factors may explain the limited predictive ability of SVR and DSI for mean MAP responsiveness in this population.

We also analyzed the duration of maximum MAP response and found that peak MAP values occurred 2 minutes post-administration in the MAP-responsive group. This finding is consistent with a study by Fischer et al., which demonstrated a gradual increase in MAP, reaching maximum responsiveness 4 minutes after a fluid bolus in fluid-responsive patients with hypotension during major vascular surgery [21]. This delay can be attributed to the gradual increase in stroke volume following fluid administration, leading to increased CO and MAP.

Furthermore, total fluid administration was significantly greater in the MAP-nonresponsive group, suggesting that the use of $Ea_{dyn}$ to guide fluid management may help prevent fluid overload. Our findings are consistent with those of Russo et al., who conducted a randomized open-label clinical trial in patients undergoing pancreatic surgery and demonstrated a significantly lower fluid balance in the group managed with a protocol incorporating $Ea_{dyn}$ for the treatment of hypotension [22]. These findings further support the role of $Ea_{dyn}$-guided fluid management in reducing the risk of fluid overload.

The strengths of our study include confirmation of $Ea_{dyn}$'s predictive ability for MAP responsiveness in patients with postoperative hypotension, as demonstrated by a high AUC, and the identification of a grey zone for cases with inconclusive results. In addition, our findings support the potential role of $Ea_{dyn}$-guided fluid management in reducing the risk of fluid overload. Furthermore, the study provides a comparative analysis of different afterload assessment modalities, including $Ea_{dyn}$, SVR, and DSI.

The limitations of our study include its observational design and relatively small sample size, which warrant validation in a larger randomized controlled trial. Another limitation is the potential for mathematical coupling, as both PPV and SVV are derived from the same arterial waveform. As described by Monge García et al., because SVV and PPV are obtained from pulse contour analysis, mathematical coupling cannot be excluded as a possible explanation for the observed findings [23].

However, this concern aligns with the findings of Magrelli et al., who conducted a prospective observational study on fluid responders undergoing aortic surgery. Their study compared $Ea_{dyn}$ calculated from the FloTrac system (internally calibrated pulse contour analysis) and Bioreactance, showing no statistically significant difference ($0.83 \pm 0.37$ vs $0.75 \pm 0.33$, $p = 0.16$). These results suggest that although mathematical coupling cannot be entirely excluded, the accuracy of $Ea_{dyn}$ remains comparable whether derived from the same arterial waveform or an independent method [24].

## CONCLUSION

$Ea_{dyn}$ is demonstrated as a potential reliable hemodynamic index for predicting MAP responsiveness to fluid bolus in fluid-responsive postoperative mechanically ventilated patients with hypotension. In contrast, SVR and DSI demonstrated lower predictive ability, distinguishing MAP responsiveness from non-responsiveness in this population. Further randomized controlled trials are required to validate $Ea_{dyn}$'s predictive ability in postoperative mechanically ventilated patients with hypotension.

## CONFIDENTIALITY

All participants will not be individually identified. Each patient is represented by a unique study code number. All data are recorded exclusively in the case record forms and stored securely on the investigator's personal computer with restricted access. Upon completion of the study, all data will be permanently deleted and destroyed in accordance with institutional data protection policies.

## ETHICS

The study has received ethical approval from the Chulalongkorn University Faculty of Medicine Institutional Review Board (IRB No. 0941/66) and is registered with the Thai Clinical Trials Registry (TCTR20240330001).

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## AUTHORS' CONTRIBUTIONS

(I) Conceptualization: Isaravadee Rakphuak, Sahadol Poonyathawon; (II) Data collection: Isaravadee Rakphuak, Warangkana Chaipatanakarn; (III)Formal analysis: Isaravadee Rakphuak; (IV) Funding acquisition: Isaravadee Rakphuak; (V) Methodology: Isaravadee Rakphuak, Sahadol Poonyathawon; (VI) Project administration: Isaravadee Rakphuak; (VII) Visualization: Isaravadee Rakphuak, Warangkana Chaipatanakarn; (VIII) Writing - Original draft: Isaravadee Rakphuak; (IX) Writing – review & editing: Thammasak Thawitsri, Pongpol Sirilaksanamanon, Chalermchai Komaenthammasophon, Sahadol Poonyathawon; (X) Echocardiogram training for CO measurement: Chalermchai Komaenthammasophon (Cardiac intensivist)

## REFERENCES

1. Khanna AK, Shaw AD, Stapelfeldt WH, Boero IJ, Chen Q, Stevens M, et al. Postoperative Hypotension and Adverse Clinical Outcomes in Patients Without Intraoperative Hypotension, After Noncardiac Surgery. Anesth Analg. 2021;132:1410–20.
2. Hoppe P, Kouz K, Saugel B. Perioperative hypotension: clinical impact, diagnosis, and therapeutic approaches. Journal of Emergency and Critical Care Medicine. 2019;4.
3. Bain C, Myles PS, Corcoran T, Dieleman JM. Postoperative systemic inflammatory dysregulation and corticosteroids: a narrative review. Anaesthesia. 2022;78(3):356-70.
4. Zhou X, Pan W, Chen B, Xu Z, Pan J. 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. Annals of intensive care. 2021;11(1):119.
5. Monge García MI, Gil Cano A, Gracia Romero M. Dynamic arterial elastance to predict arterial pressure response to volume loading in preload-dependent patients. Crit Care. 2011;15:R15.
6. Monge García MI, Santos A. Understanding ventriculo-arterial coupling. Ann Transl Med. 2020;8:795.
7. Asleh R, Resar JR. Utilization of Percutaneous Mechanical Circulatory Support Devices in Cardiogenic Shock Complicating Acute Myocardial Infarction and High-Risk Percutaneous Coronary Interventions. J Clin Med. 2019;8.
8. Monge García MI, Guijo González P, Gracia Romero M, Gil Cano A, Rhodes A, Grounds RM, et al. Effects of arterial load variations on dynamic arterial elastance: an experimental study. Br J Anaesth. 2017;118(6):938–46.
9. Bar S, Leviel F, Abou Arab O, Badoux L, Mahjoub Y, Dupont H, et al. Dynamic arterial elastance measured by uncalibrated pulse contour analysis predicts arterial-pressure response to a decrease in norepinephrine. Br J Anaesth. 2018;121:534-40.
10. Seo H, Kong YG, Jin SJ, Chin JH, Kim HY, Lee YK, et al. Dynamic Arterial Elastance in Predicting Arterial Pressure Increase After Fluid Challenge During Robot-Assisted Laparoscopic Prostatectomy: A Prospective Observational Study. Medicine. 2015; 94(41):e1794.
11. Ospina-Tascón GA, Teboul J-L, Hernandez G, Alvarez I, Sánchez-Ortiz AI, Calderón-Tapia LE, et al. Diastolic shock index and clinical outcomes in patients with septic shock. Annals of Intensive Care. 2020;10:41.
12. Hernández G, Ospina-Tascón GA, Damiani LP, Estenssoro E, Dubin A, Hurtado J, et al. Effect of a Resuscitation Strategy Targeting Peripheral Perfusion Status vs Serum Lactate Levels on 28-Day Mortality Among Patients With Septic Shock: The ANDROMEDA-SHOCK Randomized Clinical Trial. JAMA. 2019;321:654-64.
13. Halvorsen S, Mehilli J, Cassese S, Hall TS, Abdelhamid M, Barbato E, et al. 2022 ESC Guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery. Eur Heart J. 2022;43:3826-924.
14. Monnet X, Marik PE, Teboul JL. Prediction of fluid responsiveness: an update. Annals of intensive care. 2016;6(1):111.
15. Shoukri MM, Asyali MH, Donner A. Sample size requirements for the design of reliability study: review and new results. Stat Methods Med Res. 2004;13:1-21.
16. Donner A, Eliasziw M. A goodness-of-fit approach to inference procedures for the kappa statistic: Confidence interval construction, significance-testing and sample size estimation. Stat Med. 1992;11:1511-9.
17. Biais M, Ehrmann S, Mari A, Conte B, Mahjoub Y, Desebbe O, et al. Clinical relevance of pulse pressure variations for predicting fluid responsiveness in mechanically ventilated intensive care unit patients: the grey zone approach. Crit Care. 2014;18:587.
18. García MIM, Romero MG, Cano AG, Aya HD, Rhodes A, Grounds RM, et al. Dynamic arterial elastance as a predictor of arterial pressure response to fluid administration: a validation study. Critical Care. 2014;18:626.
19. Cannesson M. The "grey zone" or how to avoid the binary constraint of decision-making. Can J Anaesth. 2015;62:1139-42.
20. Ospina-Tascón GA, Hernández G, Bakker J. Diastolic shock index (DSI) works… and it could be a quite useful tool. Annals of Intensive Care. 2020;10:109.
21. Fischer A, Menger J, Mouhieddine M, Seidel M, Edlinger-Stanger M, Bevilacqua M, et al. Stroke Volume and Arterial Pressure Fluid Responsiveness in Patients With Elevated Stroke Volume Variation Undergoing Major Vascular Surgery: A Prospective Intervention Study. J Cardiothorac Vasc Anesth. 2023;37:407-14.
22. Russo A, Aceto P, Cascarano L, Menga LS, Romanò B, Carelli S, et al. A dynamic elastance-based protocol to guide intra-operative fluid management in major abdominal surgery: A randomised clinical trial. Eur J Anaesthesiol. 2025;42:727-36.
23. García MI, Romero MG, Cano AG, Aya HD, Rhodes A, Grounds RM, et al. Dynamic arterial elastance as a predictor of arterial pressure response to fluid administration: a validation study. Crit Care. 2014;18:626.
24. Abstracts of the ICARE 2023 77th SIAARTI National Congress. Journal of Anesthesia, Analgesia and Critical Care. 2023;3:43.