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ORIGINAL ARTICLE

Comparison of mean systemic pressure among patients with acute circulatory failure receiving passive leg raising vs. pneumatic leg compression

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ABSTRACT:

Background: Driving pressure of venous return (VR) is determined by a pressure gradient between mean systemic pressure (Pms) and central venous pressure (CVP). While passive leg raising (PLR) and pneumatic leg compression PC (PC) can increase VR, no study has explored the effects of these two procedures on Pms and VR-related hemodynamic variables.

Methods: Forty patients with acute circulatory failure were enrolled in this analysis. All patients obtained both PLR and PC, and were measured for Pms, CVP, mean arterial pressure (MAP), cardiac output (CO), VR resistance (RVR), and systemic vascular resistance (SVR) at baseline and immediately after procedures. To minimize carry over effect, the patients were divided in 2 groups based on procedure sequence which were 1) patients receiving PLR first then PC (PLR-first), and 2) patients receiving PC first then PLR (PC-first). Both groups waited for a washout period before performing the 2 second procedure. Primary outcome was difference in Pms between PLR and PC procedures. Secondary outcome were differences in CVP, MAP, CO, RVR, and SVR between PLR and PC procedures.

Results: No difference was found in baseline characteristics and no carry over effect was observed between the 2 groups of patients. Compared with baseline, both PLR and PC significantly increased Pms, CVP, MAP, and CO. PLR increased Pms (9.0±2.3 vs 4.8±1.7 mmHg, p<0.001), CVP (4.5±1.2 vs. 1.6±0.7 mmHg, p<0.001), MAP (22.5±5.6 vs. 14.4±5.0 mmHg, p<0.001), and CO (1.5±0.5 vs. 0.5±0.2 L/min, p<0.001) more than PC. However, PC, also significantly increased RVR (16 ± 27.2 dyn.s/cm^5, p=0.001) and SVR (78.4 ± 7.2 dyn.s/cm^5, p<0.001) but no difference in PLR group.

Conclusion: Among patients with acute circulatory failure, PLR increased Pms, CVP, MAP, and CO more than PC.

Keywords: Mean systemic pressure, Cardiac output, Venous return.

INTRODUCTION

According to the Guyton model of circulation, systemic venous return (VR) is determined by two components [1]. The first is the pressure gradient between the mean systemic pressure (Pms) and the right atrial pressure, which attributes to promote VR. The second is the resistance to VR (RVR), which tends to impede VR. Pms is a quantitative measure of intravascular filling pressure, theoretically defined as the pressure that exists when stop-flow circumstance equilibrates all cardiovascular compartment and was first described by Bayliss and Starling in a dog model during cardiac arrest [2]. The Pms depends basically on two variables: the "stressed volume", which is the blood stretches the blood vessel causing intravascular pressure, and the compliance of the cardiovascular system. Under steady conditions, the cardiac output (CO) and VR are equal, and any parameter determining VR will therefore also determine CO. Fundamentally, Guyton stated that VR is defined by three parameters: the Pms, the right atrial pressure (RAP) and the RVR. The difference between the Pms and RAP or central venous pressure (CVP) is the pressure gradient of VR (dVR) where Pms is the pressure promoting the return of blood to the heart. Under steady resistances, the VR is approximately proportional to this dVR. While the classic study by Guyton noticed that an increase in blood volume increases Pms and also decreases RVR because of distension of vessels wall [2], recent studies have found that vasopressor increases RVR and restores mean arterial pressure (MAP) [3].

Passive leg raising (PLR) has been developed as a test to predict fluid responsiveness [4]. This maneuver is supposed to transfer a significant volume of venous blood toward the intrathoracic compartment. However, it has been suggested that PLR could have nonsignificant effects on cardiac preload, particularly in case of intra-abdominal hypertension. This would result in a false negative PLR test in spite of an actual fluid responsiveness. Estimating Pms and RVR are not practically at bedside as it requires, as it requires measurement of intravascular systemic pressure during cardiac arrest. Recently, an elegant method has been proposed by Maas and co-workers to estimate Pms and RVR at the bedside, based on recording several pairs of CO and CVPcic pressure [5]. Currently, intermittent pneumatic compression (PC) comprises an alternative method to prevent venous thromboembolism (VTE) among patients in the intensive care unit (ICU) [6]. Its mechanisms increase VR [7]. Related studies demonstrated that PC could increase VR up to 106 mL among patient with varicose veins [8]. While both PLR and PC can increase VR, no study has explored the effects of these two procedures on Pms and VR-related hemodynamic variables, leading us to conduct this study investigating the effects of these procedures on Pms and RVR.

MATERIALS AND METHODS

This study aimed to compare changes in Pms and VR-related hemodynamic variables following PLR and PC among patients with shock. We performed a prospective randomized cohort and cross-over study. To randomize which patients started with PLR or PC, we used the block of four method to create a 1:1 ratio by variables in block size. Then with computer-generated sequence and allocation, single blinded by opaque envelopes (concealed with opaque envelopes). patients were classified to PLR-first and PC-first as described below.

KEY MESSAGES:

- Both PLR and PC increased Pms, CVP, MAP, and CO among patients with acute circulatory failure and PLR increased more than PC.

Participants

In all, 40 patients in the medical ICU, Phramongkutklao Hospital, Bangkok, Thailand from April 2020 to February 2021 were monitored for invasive arterial blood pressure, peripheral O_2 saturation (SpO_2), and electrocardiogram. Sedative and paralysis agents were given, keeping Richmond Agitation Sedation Scale (RASS) less than -3 and Bispectral index (BIS) 40 to 60 while monitoring based on bedside physician judgement including fentanyl, propofol, midazolam and cisatracurium. Patients were mechanically ventilated using a volume-control ventilation mode, and tidal volume 8 ml/kg. The respiratory rate was adjusted to maintain normocapnia, inspired oxygen fraction was adjusted to maintain SpO_2 above 94%, and inspiratory/expiratory ratio was 1:2. The providing physician consulted a radiologist or vascular surgeon to screen deep vein thrombosis (DVT) among all patients enrolled in this study using bedside ultrasound. All patients were measured for Pms, CVP, MAP, CO, RVR, and SVR at baseline and immediately after each procedure. To minimize carry over effect, patients were divided in 2 groups based on procedure sequence which included 1) patients receiving PLR first then PC (PLR-first), and 2) patients receiving PC first then PLR (PC-first). Both groups waited during a washout period of 15 minutes before performing the second procedure. The inclusion criteria included age more than 18 years, septic shock (sepsis-3 criteria) or acute circulatory failure defined as persistent MAP less than 65 mmHg at least 15 min despite adequate volume resuscitation (perform dynamic parameters shows fluid nonresponsive) or required vasopressor to maintain MAP more than 65 mmHg. The exclusion criteria included patients having contraindication for esophageal Doppler catheter placement, intolerance to esophageal Doppler probe insertion, coarctation of the aorta, bleeding tendency, patients who have received advanced mechanical hemodynamic support, valvular heart disease, contraindication for fluid challenge, contraindications for PC and PLR such as limb amputation or bed ridden more than 1 month before enrollment due high risk for DVT, peripheral arterial disease (ankle-brachial index <0.9), pregnancy, intra-abdominal pressure more than 16 mmHg, acute respiratory distress syndrome, muscle atrophy of the leg, refractory shock (norepinephrine dose ≥0.5 μg/kg/min), increased intracranial pressure, pneumothorax or cardiac arrhythmia. Withdrawal criteria included patients presenting complications from esophageal Doppler insertion, hypoxemia (O_2 saturation <92%), increased vasopressor use, cardiac arrhythmia, pain when performing PLR and PC, and intra-abdominal pressure more than 16 mmHg during procedure. For the high PEEP during Pms measurement among patients with shock, when their hypotension worsened or they experienced any complications during the procedure such as arrhythmia, the patients were excluded from our study.

Hemodynamic Monitoring

Radial arterial catheter and central venous catheter were connected to a bedside monitor on one side and to a specific transducer (Philips Intellivue Philips MX600, USA) for blood pressure and CVP monitoring. The value of CO, SV, and SVR were estimated using the aortic flow from an esophageal Doppler (CardioQ, Deltex medical, UK). Pms was measured using the 4-inspiratory hold maneuver method suggested by Maas et al [5]. Briefly, the patients received the 4-inspiratory hold maneuver with PEEP of 5, 15, 25, and 35 cmH_2O and recorded pairs of CO and CVP to generate a VR Guyton's curve (Fig. 1). The Pms was estimated as the pressure corresponding to the x-intercept of the regression line.

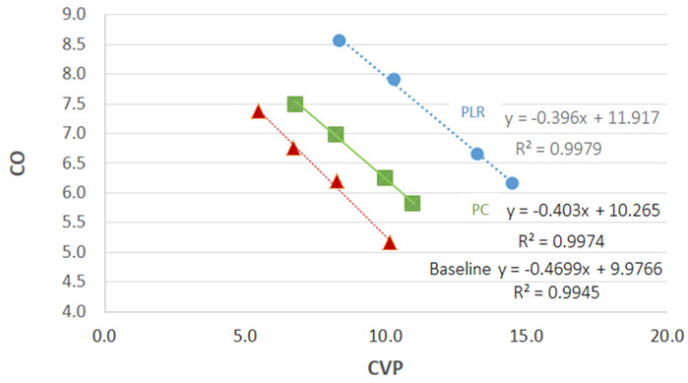

Figure 1. Venous return Guyton's curve generated from the 4-inspiratory hold maneuver method

Interventions

In the PLR-first group, the patient started in semi recumbent with head elevation 45 degree position. Then the upper body was placed in a horizontal position and patients passively raised their legs at 45 degrees and maintained the maximal effect at 30 seconds during the 4-inspiratory hold maneuver method. In the PC-first group, the sleeves inflated pressure at 40 mmHg for 2 minutes after immediately fully inflating at 2 minutes to perform the 4-inspiratory hold maneuver method and then were deflated.

Statistical Analysis

The primary outcomes were differences in Pms between PLR and PC procedures and secondary outcomes were differences in CVP, MAP, CO, RVR, and SVR between PLR and PC procedures. In a related pilot study of PLR and volume expansion on Pms and VR [3], sample size estimation showed that at least 26 patients were required to evaluate ability and to compare difference in Pms between PLR and PC procedures. Results were expressed as mean ± SD when data were normally distributed or median and interquartile range (IQR) when not. Hemodynamic parameters were compared at baseline between PLR and PC procedures using the independent-t test, paired t-test, Fisher's exact test, Pearson's correlation, and repeated measure ANOVA test. The effects of volume expansion on hemodynamic parameters were analyzed using Friedman nonparametric repeated measures comparisons. A p-value less than 0.05 was considered statistically significant. Statistical analysis was performed using SPSS, Version 23.0.

Ethics Approval and Consent to Participate

The Institutional Review Board, Royal Thai Army Medical Department Ethics Committee approved this study March 4, 2020. Research no. R177h/62 following the Council for International Organization of Medical Science (CIOMS) Guidelines 2012 and Good Clinical Practice of International Conference on Harmonization statement no. IRBRTA 292/2563.

RESULTS

Patient Characteristics

All 40 patients with acute circulatory failure were enrolled in our analysis and obtained both PLR and PC classified by two groups. Hemodynamics parameters were measured for Pms, CVP, MAP, CO, RVR, and SVR at baseline, and immediately after procedures. Most patients were female (52%) with average age of 68 years. The most frequent coexisting disease was hypertension, and the most common etiology of shock was septic shock (Table 1). Regarding baseline characteristics between the two groups of patients, there was no difference of observed variables in baseline characteristic between the two groups

Differences in Pms and VR-related Variables Compared between PLR and PC

No carry over effect was observed between the two groups and for the crossover study, period effect analysis and sequence effect analysis results are shown in Table 3. No change was observed regarding p-value between the two groups. Compared with baseline, both PLR and PC significantly increased Pms, CVP, MAP, and CO (Table 3), while both PC and PLR significantly increased Pms (Fig. 2). PLR increased Pms (9.0±2.3 vs 4.8±1.7mmHg, p<0.001) (Fig 3A), MAP (22.5±5.6 vs. 14.4±5.0mmHg, p<0.001) (Fig. 3B), CO (1.5±0.5 vs. 0.5±0.2 L/min, p<0.001) (Fig. 3C), and CVP (4.5±1.2 vs. 1.6±0.mmHg, p<0.001) (Fig. 3D), dVR (4.4+2.6 vs. 3.2+1.7, p<0.001) more than PC (Fig. 4A). PC. However, PC not PLR, significantly increased RVR (16±27.2 dyn.s/cm^5, p=0.001) (Fig. 4B) and SVR (78.4±7.2 dyn.s/cm^5, p<0.001) (Fig. 4C). Additionally, we explored whether NE affected Pms and RVR and found a fair correlation between NE dosage and RVR in PLR procedure (r= 0.35, p=0.027) but not in PC procedure.

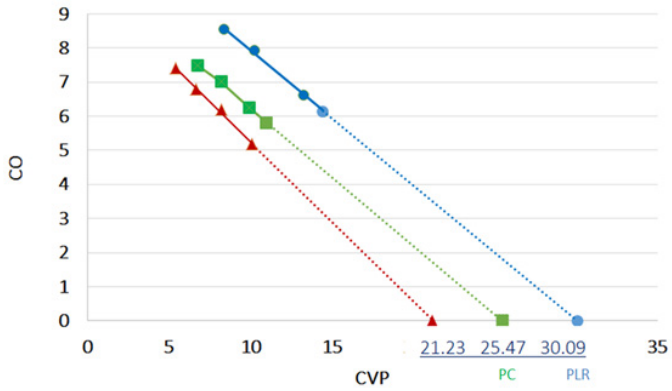

Figure 2. Guyton's VR curves and Pms plotted among patients receiving PC and PLR

Table 1. Guyton's VR curves and Pms among patients receiving PC and PLR

Variables	N = 40
Male, n (%)	19 (47.5)
Age (yr)	68.25 ± 17.23
Body weight (kg)	58.25 ± 5.38
Co-morbidity, n (%)	
Hypertension	29 (72.5)
Dyslipidemia	18 (45)
Diabetes mellitus	18 (45)
Chronic kidney disease	10 (25)
Chronic liver disease	9 (22.5)
Coronary artery disease	4 (10)
Other diseases	21 (52.5)
IV fluid (mL)	1725 ± 521
Type of shock, n (%)	
Septic	33 (82.5)
Cardiogenic	4 (10)
Hypovolemic	3 (7.5)
Variables	**Mean ± SD.**
Norepinephrine dosage (μg/kg/min)	0.35 ± 0.06
APACHE II score	12.68 ± 2.07
MAP on admission (mmHg)	80.25 ± 7.39
Blood lactate on admission (mmo/L)	9.23 ± 5.8
Pulse pressure variation on admission (%)	15.35 ± 0.92

Table 2. Baseline characteristics compared between the two groups

Variable	PLR →PC (n=20)	PC→ PLR (n=20)	P-value
Male, n (%)	9 (45)	10 (50)	1.0
Age (yr)	66.1 ± 18.5	70.4 ± 16.04	0.437
Body weight (kg)	58.5 ± 5.4	58 ± 5.48	0.773
Coexisting diseases, n (%)			
HT	16 (80)	13 (65)	0.480
DLP	10 (50)	8 (40)	0.751
DM	10 (50)	8 (40)	0.751
CKD	8 (40)	2 (10)	0.065
Chronic liver disease	4 (20)	5 (25)	1.0
CAD	4 (20)	0 (0)	0.106
APACHE II Score	12.7 ± 1.95	12.65 ± 2.23	0.940
Received IV fluid (mL)	1651.5 ± 470.15	1798 ± 570.53	0.381
Fever	6 (30%)	8 (40%)	0.741
Sedation drug			
Fentanyl	17 (85%)	16 (80%)	1
Propofol	6 (30%)	8 (40%)	0.741
Dormicum	4 (20%)	2 (10%)	0.661
Type of Shock			
Septic	17 (85)	16 (80)	1
Cardiogenic	2 (10)	2 (10)	0.605
Hypovolemic	2 (10)	1 (5)	0.231
NE dosage (μg/kg/min)	0.34 ± 0.07	0.35 ± 0.06	0.486

Value presented as mean ± SD or n (%), P-value corresponds to Independent-t test and Fisher's exact test.

Table 3. Changes in hemodynamic variables from baseline, compared between PLR and PC maneuvers

Variables	PLR				PC				Difference between PLR vs. PC	P-value
	Baseline	Post-PLR	Mean change	p-value	Baseline	Post- PC	Mean change	p-value		
MAP (mmH)	74.95 ± 4.68	97.43 ± 7.78	22.48 ± 5.6	<0.001*	74.95 ± 4.68	89.3 ± 6.5	14.35 ± 5	<0.001*	8.13 ± 3.47	<0.001#
Pms (mmHg)	25.73 ± 7.05	34.65 ± 7.45	8.93 ± 2.34	<0.001*	25.73 ± 7.05	30.5 ± 6.94	4.78 ± 1.72	<0.001*	4.15 ± 1.08	<0.001#
CO (L/min)	6.49 ± 1.27	7.97 ± 1.14	1.48 ± 0.47	<0.001*	6.49 ± 1.27	7.01 ± 1.28	0.52 ± 0.17	<0.001*	0.97 ± 0.38	<0.001#
CVP (mmHg)	7.68 ± 1.49	12.2 ± 1.18	4.53 ± 1.22	<0.001*	7.68 ± 1.49	9.28 ± 1.13	1.6 ± 0.71	<0.001*	2.93 ± 1.05	<0.001#
RVR (dyn.s/cm^5, Wood units)	245.6 ± 144.8	236 ± 107.2	-9.6 ± 54.4	0.260	245.6 ± 144.8	261.6 ± 130.4	16 ± 27.2	0.001*	-25.6 ± 34.4	<0.001#
Pms-CVP (mmHg)	18.05 ± 7.61	22.45 ± 7.99	4.4 ± 2.61	<0.001*	18.05 ± 7.61	21.23 ± 7.38	3.18 ± 1.72	<0.001*	1.23 ± 1.59	<0.001#
SVR (dyn.s/cm^5, Wood units)	877.6 ± 264	876.8 ± 176.8	-0.8 ± 124	0.959	877.6 ± 264	956 ± 252.8	78.4 ± 7.2	<0.001*	-80 ± 91.2	<0.001#

Value presented as mean±SD, and mean change. * depicts p <0.05 and compared between baseline vs. each intervention, # depicts p<0.05 and compared between two interventions. P-value analyzed using the paired t-test.

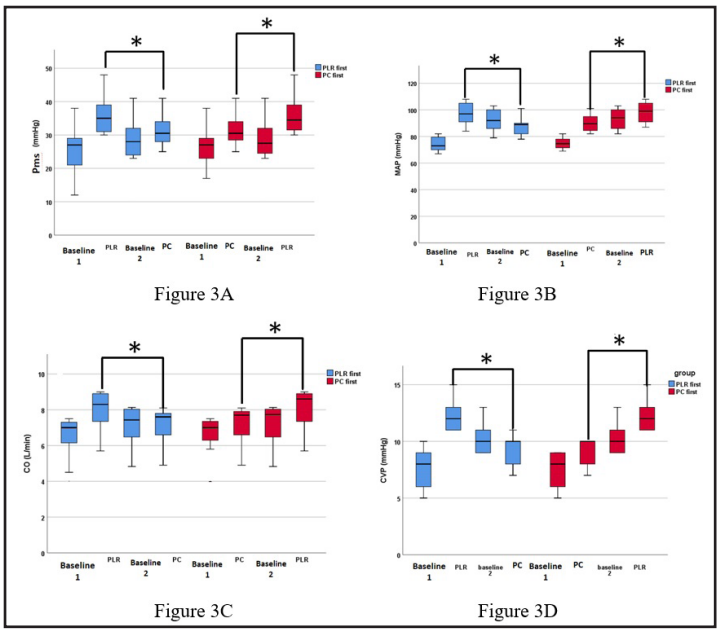

Figure 3A Figure 3B

Figure 3C Figure 3D

Figure 3A Comparing Pms between PLR-first and PC-First
Figure 3B Comparing MAP between PLR-first and PC-First
Figure 3C Comparing CO between PLR-first and PC-First
Figure 3D Comparing CVP between PLR-first and PC-First
p-value corresponds to Repeated ANOVA test.

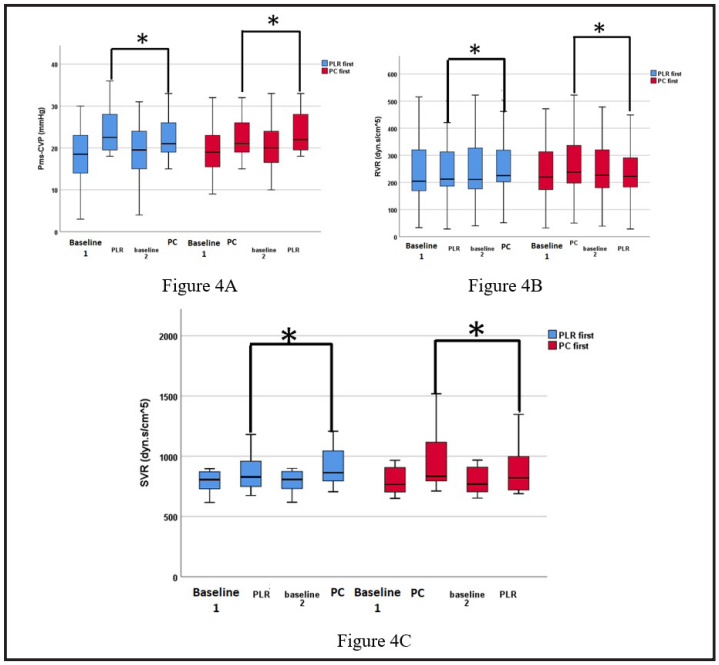

Figure 4A Figure 4B

Figure 4C

Figure 4A Comparing Pms-CVP between PLR-first and PC-First
Figure 4B Comparing RVR between PLR-first and PC-First
Figure 4C Comparing SVR between PLR-first and PC-First
p-value corresponds to Repeated ANOVA test.

DISCUSSION

In this study, we evaluated the effects of PC and PLR on Pms and VR-related parameters among patients with acute circulatory failure. We found that both PLR and PC significantly increased Pms, CVP, MAP, and CO from baseline. We also found that PLR significantly increased Pms, CVP, MAP, and CO higher than PC. Regarding venous and arterial systems, PC, not PLR, significantly increased RVR and SVR. Presently, among the critically ill patients with sepsis, PC is widely used as alternative method for DVT prophylaxis as recommended by Surviving Sepsis Campaign Guidelines 2018 and the European Society of Intensive Care Medicine [10]. However, even this study using PC in patients with septic shock, it seemed to have similar physiologic effects as the pneumatic anti-shcok garment (PASG) used in hemorrhagic shock [11]. Basically, it improves hemodynamics by increased Pms, and then augments VR increasing MAP [11]. Related studies [9], evaluating the effects of PC on augmented flow velocity and volume flow among patients with varicose veins found that PC significantly increased VR volume up to 106 ml per cycle. In addition, a related study among healthy volunteers found increased venous volume following thigh cuff pressure [7]. In this study we not only demonstrated that PC increased VR by increasing Pms but we also demonstrated that PC increased MAP among patients with acute circulatory failure. In contrast to PLR, we found that PC significantly increased RVR and SVR, which may have been because of the direct effect of vasoconstriction by PC in both venous and arterial systems of the thigh. We also found that PLR increased Pms, CVP, MAP, and CO more than PC.

This study encountered several limitations. First, CO was monitored by esophageal doppler ultrasound that is operator-dependent as well as the angle of the probe must be steadily maintained for accurate measurement. Second, this prospective cohort study and cross-over trial were conducted among patients receiving mechanical ventilation, so our findings cannot be extrapolated among patients with spontaneous breathing. Third, PC may have stimulated a sympathetic tone and interfered with hemodynamic interpretation in our study. However, using the changes of heart rate as a surrogate of sympathetic stimulation, we found no difference in heart rate at any time point. Therefore, we speculated that the effect of sympathetic stimulation during interventions may have been minimal. Finally, our study was conducted in single center and confined only to patients with acute circulatory failure. Although many exclusion criteria were used, our findings may not generalizable among general critically ill patients not requiring circulatory support.

CONCLUSION

Among patients with acute circulatory failure, both PLR and PC increased Pms, CVP, MAP, and CO. Compared to PC, these parameters increased more with PLR. PC, but not PLR, increased RVR and SVR. Our findings supported Guyton's theory regarding venous return physiology. PLR demonstrated significantly higher hemodynamics effects than PC in Pms, CVP, MAP and CO. Increased RVR and SVR were noted after PC application but not in PLR.

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AUTHORS' CONTRIBUTIONS

Conceptualization: P. Boontoterm, P. Wacharasint, P. Feungfoo, Data curation: P. Boontoterm, P. Wacharasint, Formal analysis: P. Boontoterm, P. Wacharasint, D. Panpanich Funding acquisition: P. Wacharasint, P. Feungfoo, Method: P. Boontoterm, P. Wacharasint, P. Feungfoo, Project administration: P. Wacharasint, P. Feungfoo, Visualization: P. Wacharasint, P. Feungfoo, Writing - original draft: P. Boontoterm, P. Wacharasint, Writing – review & editing: P. Boontoterm, P. Wacharasint.

SUPPLEMENTARY MATERIALS

none

ABBREVIATIONS

APACHE, Acute Physiology and Chronic Health Evaluation; AUC, Area under the curve; BMI, Body mass index; BP, Blood pressure; bpm, Beat per minute; CAD, Coronary artery disease; CO, Cardiac output; CI, Cardiac index; CKD, Chronic kidney disease; Cv, Vascular compliance; CVP, Central venous pressure; DLP, Dyslipidemia; DM, Diabetes mellitus; DVT, Deep venous thrombosis; ECMO, Extracorporeal membranous oxygenation; g, Gram; HR, Heart rate; HT, Hypertension; IAH, Intra-abdominal hypertension; IABP, Intra-aortic balloon pump; ICU, Intensive care unit; IV, Intravenous; IQR, Interquartile Range; kg, Kilogram; LV, Left ventricle; NE, Norepinephrine; PC, Pneumatic leg compression; PCWP, Pulmonary capillary wedge pressure; PLR, Passive leg raising; Pms, Mean systemic pressure; PPV, Pulse pressure variation; ROC, Receiver operating characteristic; RVR, Venous return resistance; SCD, Sequential compression device; SD, Standard deviation; SVR, Systemic vascular resistance; SV, Stroke volume; SVV, Stroke volume variation; VR, Venous return; Vs, Stress volume; VTE, Venous thromboembolism.

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