PASSIVE LEG RAISING EFFECT AT RESUSCITATION AMONG PATIENTS WITH VASOPLEGIC STATE

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Abstract

Background: Several dynamic parameters have been used clinically to predict volume responsiveness and to guide fluid administration of which passive leg raising (PLR) is one of the most reliable techniques. PLR induces rising in cardiac output attributes through an unstressed volume mobilization from legs to heart causing autologous preload increment. Appropriate fluid resuscitation is essential and can be optimized by hemodynamic-based approach to vasodilatory hypotension.

Objective: This study aimed to evaluate the effectiveness and safety of PLR at early resuscitation among patients with vasoplegia.

Methods: We conducted a comparison study concerning an experimental design using a single blinded assessment of the outcomes that assigned patients with shock to be treated with PLR or flat position at early resuscitation. Forty patients with shock were included in this study. Twenty patients performed PLR at early resuscitation compared with the others that performed in the flat position and were measured for cardiac output (CO), mean arterial pressure (MAP), diastolic blood pressure (DBP), heart rate (HR), central venous pressure (CVP) and systemic vascular resistance (SVR) immediately after the procedures. The primary outcome was to evaluate the effect of early PLR on hemodynamic variables among hypotensive patients by comparing the difference in CO while the secondary outcomes were differences in MAP, DBP, HR, CVP, SVR, survival at hospital admission and the pulmonary complications of chest x-rays between the two groups.

Results: No difference was observed in baseline characteristics between the two groups of patients. Compared with the flat position, PLR at early resuscitation significantly increased CO (3.57 ± 0.27 vs. 2.2 ± 0.18 L/min, $p = 0.037$), MAP (22.48 ± 5.6 vs. 10.83 ± 4 mmHg, $p<0.001$), DBP (19 ± 0.20 vs. 1.23 ± 0.12 mmHg, $p=0.001$) and CVP (4.52 ± 0.19 vs. 2.18 ± 0.13 mmHg, $p=0.002$). However, no differences were observed in HR, SVR, pulmonary complications of chest X-rays [2 (10%) vs. 1 (5%), $p = 0.23$] as well as survival at hospital admission [16 (80%) vs. 13 (65%), $p = 0.48$] between the two groups.

Conclusion: Among patients with shock, PLR at early resuscitation significantly increased CO, MAP, DBP and CVP than that of those performing the flat position. No differences were found in HR, SVR, pulmonary complications; PLR did not improve survival to hospital admission.

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

J Southeast Asian Med Res 2021: 5(2):67-77
http://www.jseamed.org

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Received: 11 September 2021
Revised: 14 October 2021
Accepted: 20 October 2021

INTRODUCTION

Passive leg raising (PLR) is considered to be a maneuver that could promote venous return and increase systemic circulation.[1] PLR at early resuscitation mimics rapid volume expansion and is often used in intensive care units during the hemodynamic assessment of patients.[2] During vasoplegia or patients with shock, tissue perfusion is limited, leading to a low flow state. Increasing the venous and arterial bed resistances can improve myocardial and cerebral blood flow.[3] Early PLR stresses the volume of the venous reservoir, increasing the mean systemic pressure (Pms), which is the driving pressure of the venous return flow.[4] In a series of resuscitated pigs, PLR increased coronary perfusion pressure (CPP) compared with that in a control group. A retrograde volume loading of the aorta from early PLR may raise the intra-abdominal pressure and anterograde blood flow resistance, which increases the CPP gradient.[5] PLR has been developed as a test to predict fluid responsiveness.[6] This maneuver is supposed to transfer a significant volume of venous blood towards the intrathoracic compartment. However, it has been suggested that PLR could have nonsignificant effects on cardiac preload, in particular in the case of intra-abdominal hypertension. This would result in a negative PLR test result in spite of actual fluid responsiveness.

In recent years, concern has been growing about the safety of various interventions performed at early resuscitation. Fluid infusion at early resuscitation has led to worsened clinical outcomes.[7, 8] Another resuscitation body position, such as Trendelenburg, was associated with increased intracranial pressure. Data about the safety of PLR and the beneficial effect of early PLR remain limited. We hypothesized that early PLR could be a safe maneuver and could improve survival at discharge compared with that of patients treated using a standard protocol.

Methods

This study was reviewed and approved by the Ethics Committee of Institutional Review Board, Royal Thai Army Medical Department (IRBRTA 292/2563). The study was conducted in accordance with the Council for International Organization of Medical Science (CIOMS) Guidelines 2012 and Good Clinical Practices.

Sample size was calculated according to the comparison studies of PLR and the flat position in volume expansion.[4,15] At least 26 patients in total were required to compare differences between the two groups. Patients were enrolled on the scene at the initial shock and resuscitation assessment. In this study, patients with shock were included. A single blinded experiment was conducted by randomization of 1:1 ratio using variables of block size, computer-generated sequence and allocation and kept in opaque envelopes.

Participants

Informed consent was obtained from the enrolled patients or their legal representatives. From May 2020 to May 2021, 40 patients admitted in Medical Intensive Care Unit (ICU), Phramongkutklao Hospital, were monitored for invasive arterial blood pressure, peripheral O_2 saturation (SpO_2), and electrocardiogram.

Patients were assigned to PLR and the flat position groups. Of these, 20 patients used PLR which was performed within the first 5 min after initial shock and resuscitation assessment and was maintained until the end of resuscitation or until the patients presented stable hemodynamics. The angle of PLR was set at 45° following a related report.[4] To ensure that the legs were lifted at this angle, different assays were made. As a result, all ICU beds were equipped and adjusted to accurately measure this angle.

All patients were measured for CO, mean arterial pressure (MAP), diastolic blood pressure (DBP), heart rate (HR), central venous pressure (CVP) and systemic vascular resistance (SVR) immediately after PLR procedures. The inclusion criteria were patients aged more than 18 years old. The exclusion criteria included patients who had contraindications for PLR, e.g., limb amputation, traumatic patients with suspected pelvic or lower limb fracture, pregnancy, intraabdominal pressure more than 16 mmHg, increased intracranial pressure and pneumothorax. The primary outcome was to evaluate the effect of early PLR on hemodynamic variables among hypotensive

patients by comparing the difference of CO while the secondary outcomes were to compare differences in MAP, DBP, HR, CVP and SVR, survival at hospital admission and pulmonary complications in chest x-rays between the two groups.

Shock was defined as persistent MAP less than 65 mmHg at least 15 min despite adequate volume resuscitation (performed dynamic parameters shows fluid nonresponsive) or required vasopressors to maintain MAP more than 65 mmHg.

Septic shock was defined according to the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3) (persistence of infection, and required vasopressors to optimize MAP ≥65 mmHg, combined with a serum lactate level >2 mmoL/L (18mg/dL) despite adequate volume resuscitation.

Resuscitation was performed using early intravenous fluid resuscitation with balanced salt solution crystalloid or isotonic crystalloid at least 30 mL/kg according to Surviving Sepsis Campaign guidelines 2018, European Society of Intensive Care Medicine guidelines. Dynamic parameters used to assess adequate fluid resuscitation included pulse pressure variation (PPV), stroke volume variation (SVV) or mini-fluid challenge test when performed dynamic parameters showed fluid nonresponsive, and early use of vasopressor (norepinephrine) to maintain MAP more than 65 mmHg was applied.

General management in ICU

Patients were sedated with fentanyl and mechanically ventilated using pressure-controlled ventilation, aimed to maintain at Pplat <30 cm H_2O, using a tidal volume (V_T) of 6–8 mL/kg of predicted body weight. The faction of inspired oxygen (FiO_2) and Positive-End Respiratory Pressure (PEEP) were titrated to achieve peripheral saturation of oxygen (SpO_2) more than 94%, and RR was set to maintain arterial partial pressure of carbon dioxide ($PaCO_2$), and 35–45 mmHg end-tidal carbon dioxide ($ETCO_2$) was continuously measured.

Hemodynamic monitoring

Radial arterial catheter and central venous catheter were linked to a bedside monitor on one side and to a specific transducer (Philips Intellivue Philips MX600, USA) for blood pressure, DBP, HR and CVP monitoring. The values of CO and SVR were estimated from pulse contour analysis (EV1000 clinical platform, Edwards advanced hemodynamic monitoring tools for an integrated Edwards Critical Care System, USA).

Interventions

PLR was performed within the first 5 min after acute circulatory failure at the ICU and was maintained until the end of resuscitation. Procedures involved patients sitting in the 45 degrees, head up, semi-recumbent position, then lowering the patient's upper body to horizontal and passively raising the legs at 45 degrees up then maintaining the maximal effect occurring during resuscitation. To assess postresuscitation pulmonary complications, the report of the attending physician or radiologist on the first X-ray taken at the ICU was evaluated. Lung complications were considered when bilateral lung opacities, edema, pulmonary congestion or bilateral alveolar pattern and survival at hospital admission were described.

Statistical analysis

Results were expressed as mean ± SD when data were normally distributed or median and interquartile range (IQR) if not. Hemodynamic parameters were compared between PLR and the flat position during resuscitation 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 the Friedman nonparametric repeated measures comparisons. A *p*-value less than 0.05 was considered to be statistically significant. Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 23.0. (Armonk, NY, IBM Corp.)

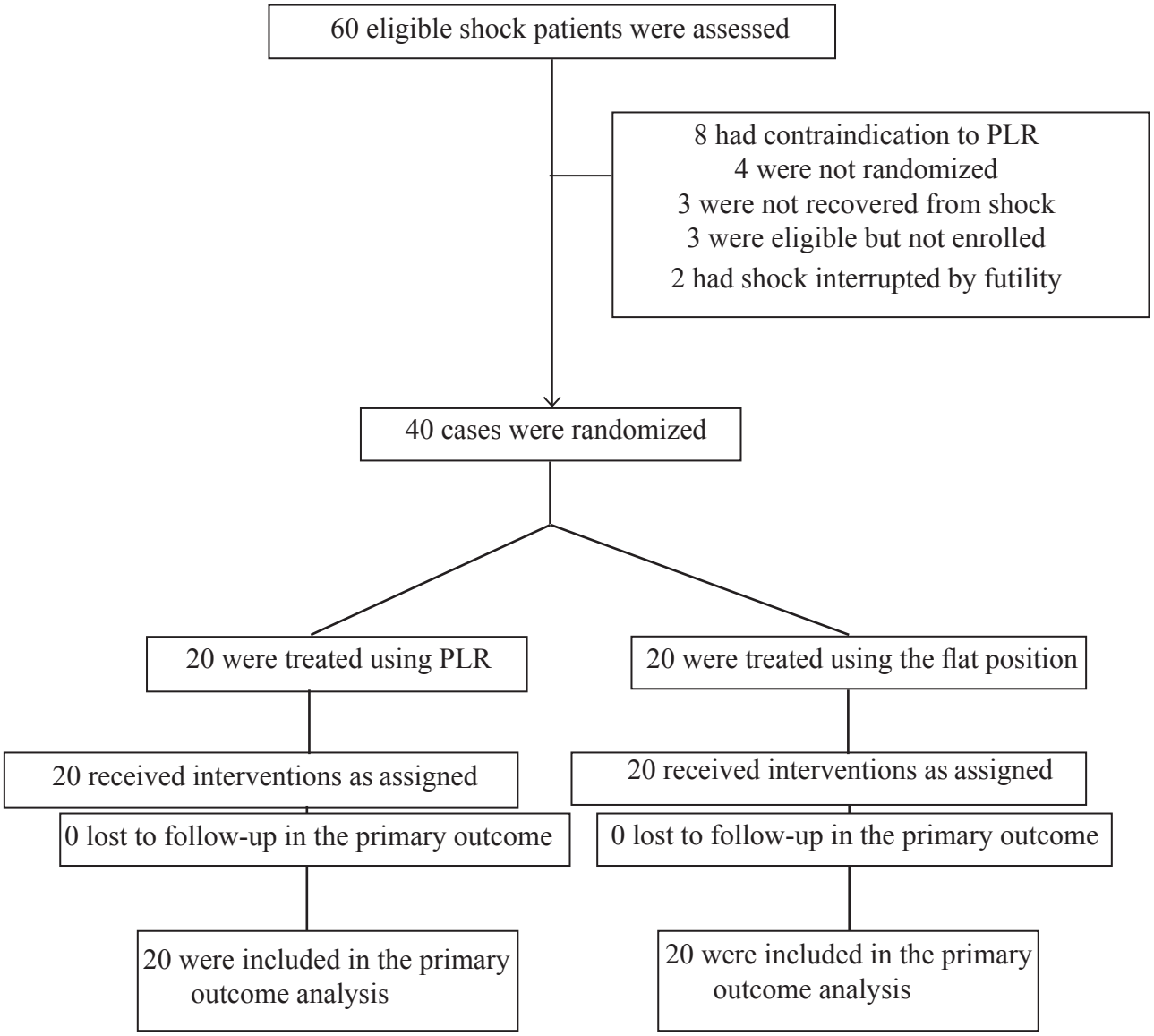

Figure 1. Flow chart of patient enrollment and analysis in the trial

Results

Patient characteristics

During the study period, 40 patients with acute circulatory failure were included. Twenty patients performed PLR. All patients were measured for CO, MAP, DBP, HR, CVP and SVR immediately after the procedures. Most patients were female (52%) with average age of 68 years. The most frequent coexisting disease was hypertension while the most frequent etiology of shock was septic shock (**Table 1**).

Regarding adverse effects, the incidence of pulmonary complications of the first chest X-rays were similar between the PLR and flat position groups, (10% vs. 5%, $p = 0.23$).

Clinical outcomes

No significant differences in survival at hospital admission were found [16 (80%) vs. 13 (65%), $p = 0.48$] between the two groups.

Table 1. Demographic data of 40 patients with acute circulatory failure

Demographic data	N = 40
Male, n (%)	19 (47.5)
Female, n (%)	21 (52.5)
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)

Characteristics between the two groups of patients

No significant difference of baseline characteristics was observed between the two groups of patients **(Table 2).**

Table 2. Comparison baseline characteristics between the two patient groups

Characteristics	**PLR (n=20)**	**Flat position (n=20)**	***p*-value**
Male, n (%)	9 (45)	10 (50)	1.0
Female, n (%)	11 (55%)	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 (%)			
Hypertension	16 (80)	13 (65)	0.480
Dyslipidemia	10 (50)	8 (40)	0.751
Diabetes mellitus	10 (50)	8 (40)	0.751
Chronic kidney disease	8 (40)	2 (10)	0.065
Chronic liver disease	4 (20)	5 (25)	1.0
Coronary artery disease	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			

Table 2. Comparison baseline characteristics between the two patient groups (ext.)

Characteristics	PLR (n=20)	Flat position (n=20)	*p*-value
Fentanyl	17 (85%)	16 (80%)	1
Heart rate (beat/min)	94 ± 23.25	93 ± 16.76	0.849
MAP on admission (mmHg)	54.95 ± 4.68	57.3 ± 6.5	0.67
DBP on admission (mmHg)	52 ± 5.55	54 ± 4.76	0.139
CVP on admission (mmHg)	7.68 ± 1.49	8.1 ± 1.8	0.73
CO on admission (L/min)	4.4 ± 1.27	4.8 ± 1.1	0.81
SVR on admission (dyn.s/cm^5)	870 ± 43.23	877 ± 45.41	0.576
Blood lactate on admission (mmol/L)	5.0 (4.0–6.9)	4.5 (3.0–7.5)	0.79
Type of Shock			
Septic	17 (85)	16 (80)	1
Cardiogenic	2 (10)	2 (10)	0.605
Hypovolemic	2 (10)	1 (5)	0.231
Dose NE (µg/kg/min)	0.34 ± 0.07	0.35 ± 0.06	0.486
Survival at hospital admission	16 (80)	13 (65)	0.480
Pulmonary complications	2 (10)	1 (5)	0.231
Days in ICU (mean)	5	6	0.63

Values presented as mean ± SD or n (%), *p*-values corresponded to independent-t test and Fisher's exact test.

*Acute Physiology and Chronic Health Evaluation

Differences of CO, MAP, DBP, HR, CVP and SVR compared between PLR and flat position groups

Changes in hemodynamic variables are shown in **Table 3**. PLR significantly increased CO (3.57 ± 0.27 vs. 2.2 ± 0.18 L/min, $p = 0.037$), MAP (22.48 ± 5.6 vs. 10.83 ± 4 mmHg, $p<0.001$), DBP (19 ± 0.20 vs. 1.23 ± 0.12 mmHg, $p=0.001$) and CVP (4.52 ± 0.19 vs. 2.18 ± 0.13 mmHg, $p=0.002$). PLR increased CO, MAP and CVP during resuscitation from baseline. Compared with the flat position, PLR increased CO, MAP and CVP more. No significant differences were found between the two groups regarding SVR [6 (-27.34, 15.34) vs. 1 (-46.83, 48.83) dyn.s/cm^5 (95%CI), $p = 0.704$] and HR [0.67 ± 0.15 vs. 0.2 ± 0.35 beat/min, $p = 0.98$].

Table 3. Changes in hemodynamic variables from baseline in PLR and the flat position during resuscitation.

Variables	Baseline before PLR (n=20)	PLR (n=20)	Baseline before flat position (n=20)	Flat position (n=20)	*p*-value
CO (L/min)	4.4 ± 1.27 Mean change from baseline	7.97 ± 1.54 3.57 ± 0.27 p<0.001*	4.8 ± 1.1 Mean change from baseline	7.0 ± 1.28 2.2 ± 0.18 *p*<0.001*	0.037#
MAP (mmHg)	54.95 ± 4.68 Mean change baseline	77.43 ± 7.78 22.48 ± 5.6 *p* <0.001*	57.3 ± 6.5 Mean change from baseline	68.13 ± 2.5 10.83 ± 4 *p* <0.001*	<0.001#

Table 3. Changes in hemodynamic variables from baseline in PLR and the flat position during resuscitation (ext.)

Variables	Baseline before PLR (n=20)	PLR (n=20)	Baseline before flat position (n=20)	Flat position (n=20)	*p*-value
DBP (mmHg)	52 ± 5.35 Mean change from baseline	71 ± 5.55 19 ± 0.20 *p* <0.001*	54 ± 4.76 Mean change from baseline	55.77 ± 4.88 1.23 ± 0.12 *p*=0.139	0.001#
HR (beat/min)	94 ± 23.25 Mean change from baseline	93.33 ± 23.4 0.67 ± 0.15 *p* =0.849	93 ± 16.76 Mean change from baseline	93.2 ± 16.41 0.2 ± 0.35 *p* =0.92	0.98
CVP (mmHg)	7.68 ± 1.49 Mean change from baseline	12.2 ± 1.68 4.52 ± 0.19 *p* <0.001*	8.1 ± 1.8 Mean change from baseline	10.28 ± 1.93 2.18 ± 0.13 *p* <0.001*	0.002#
SVR (dyn.s/cm^5)	870 ± 43.23 Mean change from baseline (95%CI)	876 ± 39.26 6 (-27.34,15.34) *p* =0.576	877 ± 45.41 Mean change from baseline (95%CI)	878 ± 45.05 1 (46.83,48.83) *p*= 0.98	0.53

Values presented as mean±SD and mean change presented as mean±SD and interquartile range,
*depicts *p* <0.05 and compared between baseline vs. each intervention
depicts *p*<0.05 and compared between two interventions
P-values were analyzed using the paired t-test and independent t-test.

Discussion

In this study, baseline characteristics of each group were comparable. We evaluated the effects of PLR on CO, MAP, DBP, HR, CVP and SVR among patients with acute circulatory failure during resuscitation. We found that PLR increased CO, MAP, DBP and CVP during resuscitation from baseline. Additionally, when compared with the flat position, PLR increased CO, MAP, DBP and CVP more. Significantly increased CO, MAP, DBP and CVP confirmed that PLR could actually represent a powerful preload challenge. The increase of venous return was attested to the increase of CO. Interestingly, PLR did not reduce venous return resistance (Rvr) while a decrease in Rvr due to a reduced sympathetic tone could have been expected from an improvement in CO. PLR resulted in a larger increase in Pms than in CVP. This increase in the pressure gradient for venous return was associated with an increase in CO. PLR effected increased venous return; thus, CO, MAP and CVP values increased when CO and MAP increased. This could improve tissue perfusion, tissue oxygenation and promote recovery of shock.

A Swedish research group reported that PLR was performed more often in cases involving a worsened clinical scenario and early PLR could improve its benefit on survival.[9] The idea of a transient effect of PLR over time has been described among patients with septic shock and is attributed to capillary leak.[10] During acute circulatory failure, maintained tissue perfusion which could favor the shortened effect of PLR on CO.[11] Optimizing venous return is the key to improve survival outcomes.

Experimental data support the distinct hemodynamic effect of PLR and volume load during resuscitation. Volume loading has been associated with decreased CPP due to the detrimental effect

of the increase in right atrial pressure (RAP) during the decompression phase.[12] However, PLR seemed not to alter RAP and has been associated with an increase in CPP. [13] It should be considered that the greatest change in CO due to PLR occurred after 1 min of the procedure.[14]

In the present study, we aimed to assess how hemodynamic variables changed during PLR among patients with shock. In particular, we aimed to investigate whether the absence of increased CO during PLR was due to an absence of increase in venous return, resulting in the absence of a significant increase in cardiac preload, or to a preload independence per se, that is, to an absence of increased CO to a significant increase in cardiac preload.

Laurent Guerin et al.[15] conducted a passive leg raising study among patients with shock and hemodynamic effects of PLR showing thatPLR increased cardiac index (CI) by 17 ± 20%. During PLR, CVP and CI significantly increased. PLR did not change the intra-abdominal pressure among the whole subjects (14 ± 6 mmHg before vs. 13 ± 5 mmHg during PLR, p = 0.26) or among patients with intra-abdominal hypertension at baseline (17 ± 4 mmHg before vs. 16 ± 4 mmHg during PLR, p = 0.14). Considering the whole subjects, the PLR-induced changes in CI predicted fluid responsiveness with an area under the receiver operator characteristic (ROC) curve of 0.98 ± 0.03. Our results confirmed the results to a related study showing 54% of increased CO. PLR could be used as a test for predicting fluid responsiveness. The test assumes that it increases the stressed blood volume by inducing the gravitational transfer of venous blood from the inferior limbs and the splanchnic compartment toward the cardiac cavities. [18] Nevertheless, the effects of PLR on the determinants of venous return have been investigated in only one study. [19] PLR test significantly shifts intravascular fluid from the legs to the abdomen, suggesting that this dynamic test may not be appropriate among patients with risk of intra-abdomenal or intrathoracic hypertension and also patients at risk of high intracranial pressure. Moreover, other studies have suggested that the PLR test would not be reliable in the case of intraabdominal hypertension because it would compress the inferior vena cava.[20, 21] However, in this study, we excluded patients with intra-abdominal hypertension.

In these regards, our study provides some interesting issues on the hemodynamic effects of PLR that PLR induced significantly increases in CO and CVP among all patients, confirming that it could actually represent a powerful preload challenge. These results agree with those of Keller et al.[19], who reported that PLR increased CVP from 4 to 6 mmHg. One of major interests of the study was to analyze the effects of PLR depending on the fluid responsiveness status. Increasing the pressure gradient for venous return was associated with an increase in CO. Physiologically, Pms depends on vascular compliance and on the volume of venous blood that is submitted to the strain of the venous reservoir walls, i.e., stressed blood volume.[22] As fluid infusion is assumed not to alter vascular compliance, our results suggested that fluid infusion increased Pms and cardiac preload by increasing the stressed blood volume, confirming the results by Keller et al.[19] Our results suggest that PLR also increased the stressed blood volume. CVP did not increase as much as Pms during PLR. This was probably related to the fact that in these fluid responsive patients, the heart was working on the steep part of the Frank-Starling curve. Therefore, a rightward shift on the venous return curve induced by the increase in Pms resulted in a smaller increase in CVP. Interestingly, PLR did not reduce venous return resistance (Rvr), while a decrease in Rvr due to a reduction in the sympathetic tone could have been expected from an improvement in CO.

Another physiological advantage was significantly higher diastolic blood pressure induced by PLR; the major determinant of coronary blood flow. One potential benefit is the combined increased diastolic blood pressure and steady heart rate theoretically allows more balance in myocardial oxygen demand and supply among patients with septic shock. The rapid effect of PLR as internal volume resuscitation has gained more attentions in its effectiveness and safety for out-of-hospital cardiopulmonary resuscitation (CPR) setting, however current evidence has not revealed clinical benefits.

A related study on PLR revealed significant sustained effects on cardiovascular parameters even at 10 min after the start of PLR.[23] Out of the parameters observed in this study, CO showed the largest and most stable sustained increase during the entire PLR time course. CO promptly returned to pre-PLR values at the end of PLR. This increase was believed to be primarily a passive response to the increase in preload due to PLR, which increases CVP, resulting in increased right ventricular CO and subsequently that of the left ventricular preload. This increase in CO presumably caused increases in the other parameters, including MAP, SBP and DBP during PLR. Wong et al. [24] reported that patients whose 500 mL blood was extracted before PLR showed a significantly greater increase in CO than that of the control. These results, together with our present observation, suggested that PLR could be an effective procedure for patients with relatively normal cardiac functions who are in hypovolemic shock and the vasoplegic state. HR tended to decrease in response to PLR. Among normal patients, strong homeostatic mechanisms work to maintain constant blood flow to vital organs. The increase in CO caused by PLR may have induced a blood pressure increase that was sensed by carotid or cardiopulmonary baroreceptors; and thus, decreased HR through a negative feedback system [25-27]. The decrease in HR during PLR was interpreted as the result of a compensatory reflex evoked by the increased CO in response to PLR. SVR is another important parameter of cardiovascular function. Although SVR gradually increased after a transient decrease at the start of PLR, no significant changes in SVR were observed at any time. This SVR response to PLR appeared to be transient and compensatory to the rapid increase in CO, which decayed in a relatively short time and caused no significant differences at any time.

Our study revealed the potential role of PLR in ICU that is probably not only the test of volume responsiveness but also an intervention for volume resuscitation among patients in the vasoplegic state even though the hemodynamic effect may last only a short period.

Study limitations are discussed below. Firstly, CO was monitored using the EV1000 clinical platform, performed by analyzing the radial artery pressure waveform. For rapid changing of intravascular volume or when using vasopressors, monitoring of femoral artery pressure has been recommended. Secondly, our study was conducted in a single center and confined only to patients with shock, so our findings could not be applied for other critically-ill patients without need of circulatory supports. Third, the time between shock with initial resuscitation and the PLR was not recorded which could be a potential confounder. Finally, PLR could have stimulated sympathetic tone and interfered with hemodynamic interpretation.

In this study, PLR was considered to be a maneuver that could promote venous return and improve hemodynamic variable among patients with shock. Some situations, i.e., CPR during cardiac arrest, can increase venous return and artificial circulation during chest compressions. During CPR, CO is limited; increasing the venous and arterial bed resistance that can improve myocardial and cerebral blood flow. PLR stresses the volume of the venous reservoir, increasing the mean systemic filling pressure, which is the driving pressure of the venous return flow. Use of coronary perfusion pressure (CPP) is a good predictor of the return of spontaneous circulation (ROSC) because a retrograde volume loading of the aorta from the PLR may occur, raising the intra-abdominal pressure and the anterograde blood flow resistance, which increases the CPP gradient. Further study about safety of various interventions performed by emergency teams during resuscitation would be very helpful. Data about the safety of PLR during CPR, and the beneficial effect of PLR performed during CPR remains very limited. It can be hypothesized that PLR performed at the beginning of out-of-hospital cardiac arrest treatment would be a safe maneuver and improve survival at discharge with good neurological outcomes compared with those of patients treated using a standard protocol.

Conclusion

Among patients with acute circulatory failure, PLR at early resuscitation significantly increased

CO, MAP, DBP and CVP more than those using the flat position. No differences in HR, SVR and pulmonary complications have been found; PLR did not improve survival to hospital admission.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Conflict of interests

No potential conflict of interest relevant to this article was reported.

Acknowledgements

We would like to thank Mrs Dollapas Panpanich for her assistance on data analysis, and also extend our appreciation to the staff of the Surgical and Medical Intensive Care Unit, Phramongkutklao Hospital for their assistance and resources used in conducting the study.

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