*Original Article*

# Correlation between Perfusion Index and Lactate Level in Critically Ill Patients

Jariya Sereeyotin MD[1], Nichcha Nutthirameth MD[2], Kanya Kumwilaisak MD[3]

[1] Division of Critical Care Medicine, Department of Anesthesiology, King Chulalongkorn Memorial Hospital and Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

[2] Department of Anesthesiology, King Chulalongkorn Memorial Hospital, Bangkok, Thailand

[3] Faculty of Medicine, Chulalongkorn University and Division of Critical Care Medicine, Department of Anesthesiology, King Chulalongkorn Memorial Hospital, Bangkok, Thailand

**Background**: During shock resuscitation, early detection of tissue hypoperfusion would guide physician to actively manage for the better outcome. Serum lactate is commonly used but it needs blood sampling and obtains intermittent data. Perfusion index (PI) analyzed from pulse-oximetry signal is a non-invasive and continuous monitoring for peripheral tissue perfusion. Previous studies have shown correlations between PI and microcirculation parameters in critically ill patients; however, limited data is available.

**Objective**: To investigate the correlation between PI and lactate level in critically ill patients.

**Materials and Methods**: The present study was a prospective observational study at the surgical intensive care unit of King Chulalongkorn Memorial Hospital (KCMH). All critically ill patients having tissue hypoperfusion and serum lactate of 2 mmol/L or greater were enrolled. PI was measured by MasimoRadical-7®PulseCO-Oximeter® and recorded values simultaneously with serum lactate at 0, 2, 6, and 24-hours during resuscitation.

**Results**: Of the 42 patients, the authors found a significant correlation between PI and lactate level at 0 and 2-hours (r=–0.397, p=0.009 and r=–0.311, p=0.045, respectively). The change in PI also significantly correlated with lactate clearance at first 6-hours of resuscitation (r=0.444, p=0.003 at 0 to 2-hour and r=0.370, p=0.017 at 2 to 6-hours). Twenty-four patients or 57% had lactate clearance of 10% or more within 2-hours, whereas 18 patients (42.8%) did not. The cut-off value of increasing in PI of less than 0.86 predicted patients who were not lactate clearance at 2-hours, (sensitivity 88.9%, specificity 54.2%, AUC 0.699, 95% CI 0.54 to 0.86, p=0.029).

**Conclusion**: PI may have value to adjunct continuous monitoring for peripheral perfusion during early resuscitation by using concurrently with serum lactate. Increase in PI of less than 0.86 within 2-hours should prompt the physician to manage it further.

**Keywords**: Lactate; Perfusion index; PI; Peripheral tissue perfusion; Microcirculatory monitoring

Received 30 May 2022 | Revised 10 October 2022 | Accepted 28 October 2022

**J Med Assoc Thai 2022;105(12):1246-53**

**Website:** http://www.jmatonline.com

The principles of shock resuscitation are maintaining hemodynamic variables and restoring microcirculation. Persistent impairment of tissue perfusion is associated with a poor survival outcome[1]. During circulatory shock, blood flow diverts from peripheral tissues to vital organs causing vasoconstriction and decreases perfusion to skin[2], so monitoring peripheral tissue perfusion would allow to recognize impaired organ perfusion early. Peripheral tissue is the last one to be reperfused after resuscitation.

At present, serum lactate is widely used to determine tissue perfusion since it was proved as a good predictor for mortality outcome[3,4]. Furthermore, surviving sepsis campaign guidelines 2016[5] suggest using it as a resuscitation guidance. However, this method requires blood sampling and provides only intermittent data.

Perfusion index (PI), an analysis of pulse oximetry signal, is a non-invasive continuous monitoring for peripheral tissue perfusion. It is the ratio of the pulsatile (arterial) blood flow to the non-pulsatile such as venous and capillaries, blood in peripheral tissues[6]. During circulatory shock, decrease in pulsatile blood flow while the constant non-pulsatile blood flow results in reduction of the ratio and PI will decrease. Alexandre et al[7] first showed that a cutoff PI value of 1.4 reflected the

**Correspondence to:**

Sereeyotin J.

Department of Anesthesiology, King Chulalongkorn Memorial Hospital, 1873 Henri Dunant Road, Pathumwan, Bangkok 10330, Thailand.

**Phone**: +66-2-2564000 ext. 81513, 66+81-1511599

**Email**: Jsereeyotin@gmail.com, Jariya.Se@chula.ac.th

**How to cite this article:**

Kumwilaisak K, Nutthirameth N, Sereeyotin J. Correlation between Perfusion Index and Lactate Level in Critically Ill Patients. J Med Assoc Thai 2022;105:1246-53.

**DOI**: 10.35755/jmedassocthai.2022.12.13718

abnormal peripheral tissue perfusion in critically ill patients and change in PI correlated with the change in core-to-toe temperature difference. Recent studies also revealed a correlation between PI and other microcirculation parameters in critically ill patients including serum lactate[8,9]; however, there is not much data and no definite cutoff value of PI relating to abnormal lactate was demonstrated. Therefore, the authors designed a study with the following objectives, 1) investigate a correlation between PI and serum lactate level, 2) correlation between change of PI and change of lactate level, 3) study whether vasopressor affects PI value or not.

## Materials and Methods

The present study was conducted over a 9-month period between May 2017 and January 2018 as a prospective observational study after being approved by the Ethics Committee, Faculty of Medicine, Chulalongkorn University (COA No.740/2017). Written informed consent was obtained from all patients or next of kin before commencement. The authors recruited all patients admitted to surgical intensive care unit (ICU) in King Chulalongkorn Memorial Hospital (KCMH) who showed signs of tissue hypoperfusion with at least one of the following, 1) mean arterial pressure (MAP) of less than 65 mmHg or decrease of systolic blood pressure (SBP) of at least 20% of baseline, 2) presence of skin mottling, 3) urine output of less than 0.5 mL/kg/hour, and hyperlactatemia or serum lactate of 2 mmol/L or more. Patients were excluded from the study if they were younger than 18 years old, pregnant, hypothermic with core temperature lower than 36°C, had conditions affecting PI measurement including peripheral arterial disease, limb ischemia, or burned all extremities, and had conditions confounding serum lactate level including severe liver disease, or being suspected of drug overdose from metformin, or salicylate.

### Patients' management

All patients received hemodynamic resuscitation based on standard clinical practice of KCMH by intensivists. Adequate pain control to postoperative patients and titrated sedation to all enrolled patients were provided throughout the study period. To minimize confounders of PI, the ambient temperature was controlled to be constant at about 23 to 25°C. The determination for either insertion of central venous catheter or monitoring global hemodynamic parameters such as cardiac output, stroke volume variation (SVV), and central venous oxygen saturation ($ScvO_2$), depended on intensivists' decision, case by case. However, in the event that central venous catheters were present, the authors obtained venous blood sampling for $ScvO_2$ and partial pressure of carbon dioxide in venous blood ($PvCO_2$) to determine the venous to arterial carbon dioxide difference ($Pv\text{-}aCO_2$). During the study period, the intensivists were blinded to the results of PI.

### Measurement

Peripheral PI was measured by the pulse oximeter Masimo SET Radical-7 (Masimo corp., Irvine, California) attached to the fingertip on the warmer side. Only one researcher installed the pulse oximetry in every patient and PI was monitored continuously for 24-hours after enrollment or earlier if discharged from the ICU. The PI value was recorded after signal stabilization. Blood for serum lactate was sampled from arterial catheter repeatedly in four time periods, at 0, 2, 4, and 24-hours, and measured by blood gas analyzer (Nova Biomedical CCX). Likewise, $ScvO_2$, $PvCO_2$, and $PaCO_2$ were measured at the same time and by the same analyzer as lactate.

### Data collection

The following data were collected at enrollment, baseline characteristics including gender and age, admission category, baseline SOFA score, global hemodynamic parameters as MAP, heart rate (HR), central venous pressure (CVP), and temperature. The authors recorded the PI value at 0, 2, 6, and 24-hours after enrollment, simultaneously with the serum lactate, $ScvO_2$ and $Pv\text{-}aCO_2$ measurement. Type and dose of vasopressor/inotropic agents were also recorded in each time period.

### Sample size calculation

Observational data from the previous study[9] reported correlation coefficient between PI and lactate –0.261. Allowing for 25% error of measurements, the total sample size required 142 measurements to provide 80% power at an $\alpha$ value of 0.05. Due to recording PI value in four time periods per patient, the final planned sample size was 36 patients.

### Statistical analysis

Data normality was tested by Kolmogorov-Smirnov test and was expressed as mean (standard deviation, SD) or as median (interquartile range, IQR) as appropriate. Demographic variables were presented by descriptive analysis. Pearson's correlation index

**Table 1.** The criteria for assessment of the Sequential Organ Failure Assessment (SOFA) score[20]

| SOFA score | 0 | 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|
| Respiratory system | | | | | |
| $PaO_2/FiO_2$ (mmHg) | <400 | <400 | <300 | <200 with respiratory support | <100 with respiratory support |
| Nervous system | | | | | |
| Glasgow Coma Scale | 15 | 13 to 14 | 10 to 12 | 6 to 9 | <6 |
| Cardiovascular system | | | | | |
| Mean arterial pressure (MAP) or administration of vasopressors required | MAP >70 mmHg | MAP <70 mmHg | Dopamine ≤5 µg/kg/minute or dobutamine (any dose) | Dopamine >5 µg/kg/minute or epinephrine ≤0.1 µg/kg/minute or norepinephrine ≤0.1 µg/kg/ minute | Dopamine >15 µg/kg/minute or epinephrine >0.1 µg/kg/minute or norepinephrine >0.1 µg/kg/minute |
| Liver | | | | | |
| Bilirubin (mg/dL) | <1.2 | 1.2 to 1.9 | 2.0 to 5.9 | 6.0 to 11.9 | >12.0 |
| Coagulation | | | | | |
| Platelets $\times 10^3$/mL | >150 | <150 | <100 | <50 | <20 |
| Kidneys | | | | | |
| Creatinine (mg/dL); urine output | <1.2 | 1.2 to 1.9 | 2.0 to 3.4 | 3.5 to 4.9 or urine output <500 mL/day | >5.0 or urine output <200 mL/day |

was used to evaluate the correlation between two continuous variables at each time, PI and lactate, and change in PI and change in lactate. In the meantime, partial correlation coefficient was used for correlation among three variables, PI, lactate, and vasopressor dose. The area under receiver operating characteristics curve (ROC curve) was calculated to generate the best cutoff PI value predicting lactate non-clearance with highest sensitivity and specificity (Youden index). To compare parameters between lactate clearance group and lactate non-clearance group, the independent t-test was used for normal distribution while the Mann-Whitney U was used for nonparametric variables. The authors considered p-value less than 0.05 to be statistically significant and used IBM SPSS Statistics, version 22.0 (IBM Corp., Armonk, NY, USA) to perform the statistical analysis.

## Results

Between May 1, 2017 and January 31, 2018, 42 critically ill patients who had tissue hypoperfusion were enrolled. One hundred fifty-nine recorded PI value and 159 lactate measurement were obtained in four time periods, with 42, 42, 41, and 34 measurements at 0, 2, 6, and 24-hours, respectively. PI value measurement at 6 and 24-hour were missed due to patients discharge from ICU. The median (IQR) SOFA score (see Table 1) at baseline was 3.5 (4 to 6). The present study patients were mainly in postoperative admission category with 36 of 42 patients, 85.71%. For the remaining six patients, three were sepsis (7.14%) and three were combined sepsis with postoperative care (7.14%). Baseline demographic data are shown in Table 2.

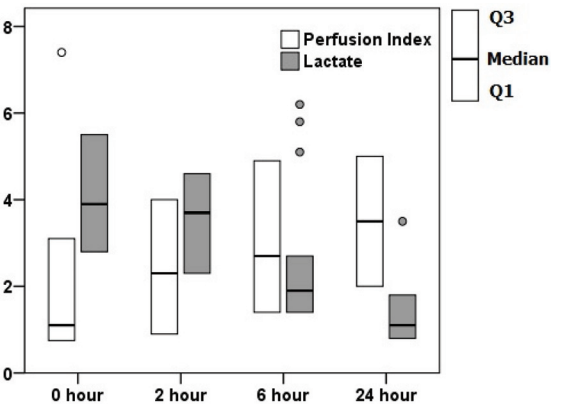


**Figure 1.** Change in perfusion index value and plasma lactate level during resuscitation periods.

### Correlation between perfusion index and lactate level

PI and lactate level altered in an inverse relationship overtime after resuscitation (Figure 1). The authors found a significant exponential correlation between PI and lactate level at 0 and 2-hours (r=–0.397, p=0.009 and r=–0.311, p=0.045, respectively) (Table 3). The change in PI also significantly correlated with lactate clearance at first 6-hours of resuscitation (r=0.444, p=0.003 at 0 to 2-hour and r=0.370, p=0.017 at 2 to 6-hours).

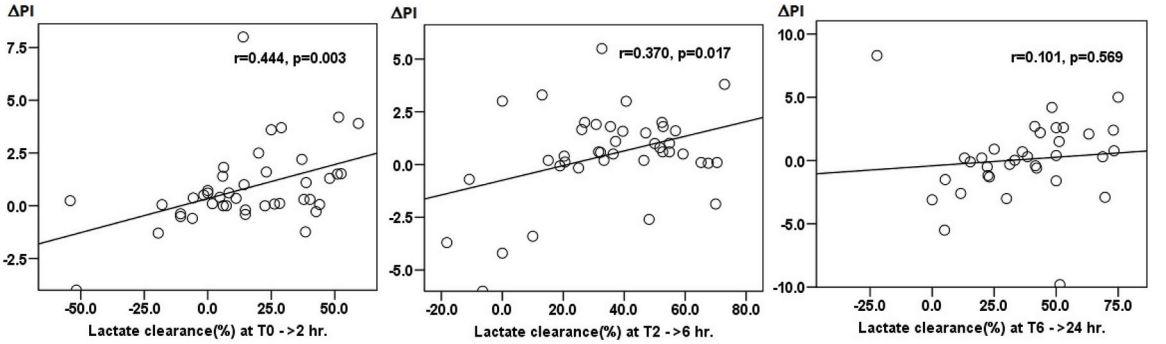


**Figure 2.** Relationship between change in PI and lactate clearance (%).

**Table 2.** Patient characteristics

| | |
|---|---|
| Total patients; n | 42 |
| Age (years); mean±SD | 67.07±11.52 |
| Sex (male/female); n (%) | 35 (83.30)/7 (16.67) |
| Underlying disease; n (%) | |
| Diabetes mellitus | 7 (16.67) |
| Hypertension | 19 (45.24) |
| Dyslipidemia | 10 (23.81) |
| Ischemic heart disease | 1 (2.38) |
| Old cerebrovascular disease | 3 (7.14) |
| Chronic kidney disease | 5 (11.90) |
| Others | 19 (45.24) |
| Baseline SOFA score; median (IQR) | 3.5 (2 to 6) |
| Body temperature (°C); mean±SD | 36.6±0.6 |
| Admission category; n (%) | |
| Postoperative care | 36 (85.71) |
| Sepsis | 3 (7.14) |
| Combined | 3 (7.14) |
| Vasopressor/dose (mcg/kg/minute); n/mean [SD] | |
| Norepinephrine | 14/0.14 [0.11] |
| Dobutamine | 2/9.3 |
| Mechanical ventilator (hours); median (IQR) | 17.5 (13.25 to 48.75) |
| ICU length of stay (day); median (IQR) | 2 (2 to 4) |
| Survived/non-survived; n (%) | |
| 7-day | 41 (97.62)/1 (2.38) |
| 30-day | 38 (90.48)/4 (9.52) |

SOFA=Sequential Organ Failure Assessment; ICU=intensive care unit; IQR=interquartile range; SD=standard deviation

**Table 3.** Correlation between PI and lactate level

| Correlation between | Time | Pearson correlation | p-value |
|---|---|---|---|
| PI and lactate (exponential relationship) | 0-hour | –0.372 | 0.025* |
| | 2-hour | –0.335 | 0.046* |
| | 6-hour | –0.201 | 0.193 |
| | 24-hour | –0.235 | 0.181 |
| Δ PI and Δ lactate | 0 to >2-hour | –0.279 | 0.073 |
| | 2 to >6-hour | –0.311 | 0.048* |
| | 6 to >24-hour | –0.039 | 0.827 |
| Δ PI and lactate clearance (%) | 0 to >2-hour | 0.466 | 0.004* |
| | 2 to >6-hour | 0.445 | 0.007* |
| | 6 to >24-hour | 0.101 | 0.569 |

PI=perfusion index

$\Delta PI = PI_{T2} - PI_{T1}$, $\Delta$ lactate = $lactate_{T2} - lactate_{T1}$,
Lactate clearance (%) = $(lactate_{T1} - lactate_{T2} / lactate_{T1}) \times 100\%$

* Statistically significant (p<0.05)

**Table 4.** Subgroup analysis of correlation between PI and lactate in postoperative patient category (36 of 42 patients)

| Correlation between | Time | Pearson correlation | p-value |
|---|---|---|---|
| PI and lactate (exponential relationship) | 0-hour | –0.372 | 0.025* |
| | 2-hour | –0.335 | 0.046* |
| | 6-hour | –0.114 | 0.515 |
| | 24-hour | –0.021 | 0.916 |
| Δ PI and lactate clearance (%) | 0 to >2-hour | 0.466 | 0.004* |
| | 2 to >6-hour | 0.445 | 0.007* |
| | 6 to >24-hour | 0.093 | 0.637 |

PI=perfusion index

$\Delta PI = PI_{T2} - PI_{T1}$,
Lactate clearance (%) = $(lactate_{T1} - lactate_{T2} / lactate_{T1}) \times 100\%$

* Statistically significant (p<0.05)

However, this relationship disappeared in the late phase of resuscitation at 6 to longer than 24-hour. These are shown in Table 3 and Figure 2.

In subgroup analysis, 36 of the 42 patients were of postoperative category. The correlation between PI and lactate was still statistically significant at 0 and 2-hour as whole sample size with r=–0.372, p=0.025; 0-hour and r=–0.335, p=0.046; 2-hour. Likewise, the change in PI also significantly correlated with lactate clearance (%) as well, r=0.466, p=0.004 at 0 to 2-hour and r=0.445, p=0.007 at 2 to 6-hour (Table 4). A subgroup of septic patients was too

**Table 5.** Correlation between PI and $ScvO_2$, PI and Pv-$aCO_2$

| Correlation between | Time | Pearson correlation | p-value |
|---|---|---|---|
| PI and $ScvO_2$ | 0-hour | 0.262 | 0.154 |
| | 2-hour | 0.437 | 0.014* |
| | 6-hour | 0.271 | 0.155 |
| | 24-hour | –0.74 | 0.732 |
| PI and Pv-$aCO_2$ | 0-hour | –0.232 | 0.218 |
| | 2-hour | –0.373 | 0.042* |
| | 6-hour | –0.027 | 0.888 |
| | 24-hour | –0.159 | 0.467 |

PI=perfusion index; $ScvO_2$=central venous oxygen saturation;
Pv-$aCO_2$=venous to arterial carbon dioxide partial pressure difference

Pv-$aCO_2$ = $PvCO_2$ – $PaCO_2$

* Statistically significant (p<0.05)

**Table 6.** Correlation between change in PI and % lactate clearance in the presence of vasopressor/inotropes

| Δ PI and lactate clearance (%) | No drug | | Norepinephrine | | Norepinephrine and dobutamine | |
|---|---|---|---|---|---|---|
| | r | p-value | r | p-value | r | p-value |
| 0 to >2-hour | 0.444 | 0.003* | 0.426 | 0.006* | 0.446 | 0.004* |
| 2 to >6-hour | 0.370 | 0.017* | 0.440 | 0.005* | 0.470 | 0.003* |
| 6 to >24-hour | 0.101 | 0.569 | 0.12 | 0.511 | 0.127 | 0.505 |

PI=perfusion index

Δ PI = $PI_{T2}$ – $PI_{T1}$,
Lactate clearance (%) = ($lactate_{T1}$ – $lactate_{T2}$ / $lactate_{T1}$) ×100%

r=Pearson correlation, * Statistically significant (p<0.05)

small to be analyzed so the correlation was not shown here.

### Correlation between perfusion index and other microcirculation parameters

PI was significantly correlated with $ScvO_2$, and Pv-$aCO_2$ only at 2-hour, while it was not in other periods (r=0.437, p=0.014 for $ScvO_2$, and r=–0.373, p=0.042 for Pv-$aCO_2$) (Table 5).

### Effect of vasopressor/inotropes dose to PI, PI, and lactate relationship

Seventeen of 42 patients with 47 of the 159 measurements, received norepinephrine during the study period with dose averaging 0.175 mcg/kg/minute (0.01 to 0.74 mcg/kg/minute, min-max dose). Six patients received dobutamine with a dose averaging 4.52 mcg/kg/minute (1.5 to 10 mcg/kg/minute, min-max dose). Neither correlation between PI and norepinephrine nor PI and dobutamine was found. Furthermore, the authors found norepinephrine dose had not affected the relationship between change in PI and lactate clearance (Table 6).

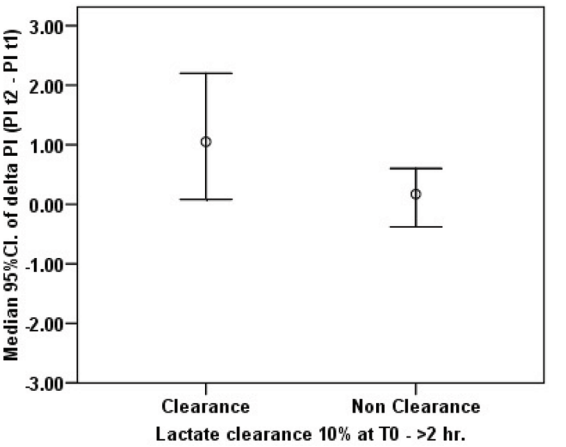


**Figure 3.** Comparison of the change in PI between lactate clearance group and lactate non-clearance group.

The authors defined the lactate clearance as decreasing of plasma lactate level at least 10% within two-hours and the present study report showed that 24 patients or 57.2% had lactate clearance, whereas 18 patients or 42.8% had not. The change in PI within 2-hour was significantly different between the two groups [median (IQR) 1.05 (0.07 to 2.35) in lactate clearance group versus 0.17 (–0.38 to 0.60) in lactate non-clearance group, p=0.029] (Figure 3). In contrast, the other variables were not different (Table 7). The cut-off value of increasing in PI less than 0.86 best predicted patients who were not lactate clearance at 2-hours with sensitivity of 88.9% and specificity of 54.2% (AUC 0.699, 95% confidence interval 0.54 to 0.86, p=0.029).

## Discussion

The present study aimed to clarify whether a PI correlates with plasma lactate level in order to use it instead of lactate for continuous monitoring during resuscitation. The main finding was that PI correlated with lactate level significantly at 0, 2-hours, and the correlation remained significant between change of PI and lactate clearance at first 6-hours of resuscitation. However, this relationship disappeared in the late phase of resuscitation.

The degree of correlation in the present study was consistent with the previous studies[8,9]. He et al performed a study in critically ill patients and showed the advantages of PI in terms of prediction for mortality and poor outcome after resuscitation. In addition, PI was correlated with the lactate (r=–0.261, p<0.0001), $ScvO_2$, and Pv-$aCO_2$ in all measurements during the 8-hour resuscitation. Furthermore, this

**Table 7.** Comparison of parameters between lactate clearance group and lactate non-clearance group

| Variables | Lactate clearance, n=24 (57%) | Lactate non-clearance, n=18 (42.8%) | p-value |
|---|---|---|---|
| Δ PI (0 to 2-hour); median (IQR) | 1.05 (0.07 to 2.35) | 0.17 (-0.38 to 0.60) | 0.029* |
| $ScvO_2$ at 2-hours (%); mean [SD] | 71.28 [8.55] | 66.85 [10.76] | 0.213 |
| Pv-$aCO_2$ at 2-hour (mmHg); median (IQR) | 6.1 (4.2 to 8.4) | 6.2 (4.0 to 11.2) | 0.806 |
| Baseline SOFA score; mean [SD] | 3.83[4.72] | 4.72 [3.64] | 0.384 |
| Mechanical ventilator (hour); median (IQR) | 16.5 (14 to 58) | 19 (12 to 63) | 0.731 |
| ICU length of stay (day); median (IQR) | 2 (2 to 7) | 2 (1 to 5) | 0.59 |
| Survival; n (%) | | | |
| 7-day | 24 (100) | 17 (94.44) | 0.243 |
| 30-day | 22 (91.67) | 16 (88.88) | 0.762 |

PI=perfusion index; $ScvO_2$=central venous oxygen saturation; Pv-$aCO_2$=venous to arterial carbon dioxide partial pressure difference; SOFA=Sequential Organ Failure Assessment; ICU=intensive care unit; IQR=interquartile range; SD=standard deviation

relationship was strengthened with abnormal PI (PI <0.4). Nevertheless, the present study found this correlation was just at 0 and 2-hour and even the correlation between change of PI and lactate clearance did not last longer than six-hours.

Based on physiology of vital organ preservation during circulatory failure, peripheral tissue perfusion in non-vital organs may deteriorate earlier and improve later than in vital organs so that PI is a sensitive marker of vital organ hypoperfusion. PI could detect early central hypovolemia in awake healthy volunteers in the study of van Genderen et al[10]. They showed the interesting finding that changes in PI preceded significant changes in HR and MAP. PI did not decrease further despite progression of hypovolemia. In this regard, PI reflected an alteration in peripheral vasomotor tone, and it decreased due to diversion of blood flow from peripheral tissue to vital organ[10,11]. On the other hand, it would also increase during a return of blood flow to it. And after adequate resuscitation, no more return of blood flow resulted in no further increase in PI. These may explain why the correlation between PI and lactate level was stronger in early phase and lessen over time.

The other reason to explain a loss of relationship in late phase is that PI reflects mainly as local tissue perfusion while plasma lactate reflects global tissue perfusion, similar to the $ScvO_2$ and Pv-$aCO_2$. Moreover, hyperlactatemia may persist even when there was no tissue hypoperfusion as it relies on both clearance and production mechanism, such as in a case of hepatic/renal dysfunction and high dose adrenergic drugs usage[12,13].

In clinical settings, PI was used in various aspects[14]. In septic shock patients, cutoff PI value at less than 0.3 could predict vasopressor requirement[15] and value below 0.2 could predict mortality after resuscitation[8]. However, the distribution of PI was highly skewed among patients in the present study, which was consistent with former studies[7]. Consequently, PI may be more valuable as a trend monitoring rather than a single value. The change in PI was also used to evaluate the depth of anesthesia[16,17] and served as an indicator of successful epidural[18,19] or peripheral nerve block.

To the best of the authors' knowledge, the present study is the first study demonstrating the correlation between change in PI and lactate clearance during early resuscitation in critically ill patients. It found the important finding that the cut-off value of increasing in PI less than 0.86 could predict patients who had no lactate clearance at 2-hours with sensitivity of 88.9% and specificity of 54.2%. The authors believed that using PI trend monitoring along with plasma lactate level will be a good screening tool alerting physician to perform further investigation in patients at risk of lactate non-clearance.

In terms of vasopressor effects on either PI value or correlation between PI and lactate level, the present study could not find any effects of vasopressor to PI although it should decrease the PI value due to the physiologic basics of vasoconstriction. This negative effect was similar to prior studies[7,8]. One of the studies performed in septic shock patients demonstrated that non-surviving group had significantly lower PI than survivors even though the dosage of norepinephrine in both groups was not different[8]. Therefore, low PI value would be truly related to tissue hypoperfusion. Vasomotor tone during septic shock is complicated. Vasodilation may be present during warm shock but there is heterogeneity among vascular bed at the same time and vasopressor effect would not be a simple manner. Further studies are warranted to explore this mechanism and effects.

The present study has limitations that should be acknowledged. Firstly, the authors would emphasize that PI reflects local tissue perfusion while plasma lactate reflects global tissue perfusion as already discussed. Moreover, multiple factors lead to hyperlactatemia even with the absence of tissue hypoperfusion. PI as a sole monitoring is still limited but it had a value as an adjunct to the lactate level or other parameter to determine requirement of further management and should be used as trend monitoring to follow up after resuscitation. Secondly, the majority of the present study subjects were postoperative patients who had hypovolemic condition, so the cutoff value of change in PI for predicting lactate non-clearance could not extrapolate to the sepsis group patients. Third, sample size in the present study was too small to explore the relationship between change in PI and poor outcome.

In conclusion, PI, which is a non-invasive continuous monitoring for tissue hypoperfusion based on analysis of pulse oximetry, correlated with plasma lactate level during early resuscitation period. It should be used as an adjunct continuous monitor along with lactate level to determine adequate resuscitation. Increase of PI less than 0.86 within two-hours after resuscitation prompts the physician to do further management with the patients. In the future, the study using change in PI for predicting lactate clearance to guide resuscitation must be confirmed for a potential to improve outcome.

### What is already known on this topic?

Few studies revealed a correlation between PI and lactate in critically ill patients, however, no definite cutoff value of PI relating to abnormal lactate was demonstrated.

### What this study adds?

The change in PI significantly correlates with lactate clearance at the first 6-hours of resuscitation The cut-off value of increasing in PI of less than 0.86 predicted the patients were not lactate clearance at 2-hours. Using PI in combination with lactate may help to predict which patients should receive further management.


### Acknowledgement

Masimo Corporation, the manufacturer of Masimo SET Radical-7 was not involved in the design and conduct of the study including providing equipment, analysis, or interpretation of data. No other funding resources had role in the present study. The study was supported by the surgical ICU, King Chulalongkorn Memorial Hospital. The authors express their gratitude to the nurses, physicians, and other health-care professionals as well as the patients and their families.


### Conflicts of interest

The authors declare no conflict of interest.

## References


1. Van Genderen ME, Lima A, Akkerhuis M, Bakker J, van Bommel J. Persistent peripheral and microcirculatory perfusion alterations after out-of-hospital cardiac arrest are associated with poor survival. Crit Care Med 2012;40:2287-94.
2. Bond RF. A review of the skin and muscle hemodynamics during hemorrhagic hypotension and shock. Adv Shock Res 1982;8:53-70.
3. Filho RR, Rocha LL, Corrêa TD, Pessoa CM, Colombo G, Assuncao MS. Blood lactate levels cutoff and mortality prediction in sepsis-time for a reappraisal? A retrospective cohort study. Shock 2016;46:480-5.
4. Trzeciak S, Dellinger RP, Chansky ME, Arnold RC, Schorr C, Milcarek B, et al. Serum lactate as a predictor of mortality in patients with infection. Intensive Care Med 2007;33:970-7.
5. Rhodes A, Evans LE, Alhazzani W, Levy MM, Antonelli M, Ferrer R, et al. Surviving sepsis campaign: International guidelines for management of sepsis and septic shock: 2016. Intensive Care Med 2017;43:304-77.
6. Masimo Corporation 40 Parker Irvine, California. Perfusion index [Internet]. 2007 [cited 2017 Jan 12]. Available from: https://www.masimo.co.uk/siteassets/uk/documents/pdf/clinical-evidence/whitepapers/lab3410f_whitepapers_perfusion_index.pdf.
7. Lima AP, Beelen P, Bakker J. Use of a peripheral perfusion index derived from the pulse oximetry signal as a noninvasive indicator of perfusion. Crit Care Med 2002;30:1210-3.
8. He HW, Liu DW, Long Y, Wang XT. The peripheral perfusion index and transcutaneous oxygen challenge test are predictive of mortality in septic patients after resuscitation. Crit Care 2013;17:R116.
9. He H, Long Y, Liu D, Wang X, Zhou X. Clinical classification of tissue perfusion based on the central venous oxygen saturation and the peripheral perfusion index. Crit Care 2015;19:330.
10. Van Genderen ME, Bartels SA, Lima A, Bezemer R, Ince C, Bakker J, et al. Peripheral perfusion index as an early predictor for central hypovolemia in awake healthy volunteers. Anesth Analg 2013;116:351-6.
11. Choi SB, Park JS, Chung JW, Kim SW, Kim DW. Prediction of ATLS hypovolemic shock class in rats using the perfusion index and lactate concentration. Shock 2015;43:361-8.

12. Hernandez G, Bellomo R, Bakker J. The ten pitfalls of lactate clearance in sepsis. Intensive Care Med 2019;45:82-5.
13. Vincent JL, Quintairos ESA, Couto L Jr, Taccone FS. The value of blood lactate kinetics in critically ill patients: a systematic review. Crit Care 2016;20:257.
14. Hasanin A, Mukhtar A, Nassar H. Perfusion indices revisited. J Intensive Care 2017;5:24.
15. Rasmy I, Mohamed H, Nabil N, Abdalah S, Hasanin A, Eladawy A, et al. Evaluation of perfusion index as a predictor of vasopressor requirement in patients with severe sepsis. Shock 2015;44:554-9.
16. Hager H, Reddy D, Kurz A. Perfusion index-a valuable tool to assess changes in peripheral perfusion caused by sevoflurane? Anesthesiology 2003;99:A593.
17. Hagar H, Church S, Mandadi G, Pulley D, Kurz A. The perfusion index measured by a pulse oximeter indicates pain stimuli in anesthetized volunteers. Anesthesiology 2004;101:A514.
18. Kakazu CZ, Chen BJ, Kwan WF. Masimo set technology using perfusion index is a sensitive indicator for epidural onset. Anesthesiology 2005;103:A576.
19. Ginosar Y, Weiniger CF, Meroz Y, Kurz V, Bdolah-Abram T, Babchenko A, et al. Pulse oximeter perfusion index as an early indicator of sympathectomy after epidural anesthesia. Acta Anaesthesiol Scand 2009;53:1018-26.
20. Lambden S, Laterre PF, Levy MM, Francois B. The SOFA score-development, utility and challenges of accurate assessment in clinical trials. Crit Care 2019;23:374.