

# High Central Venous Oxygen Saturation and Elevated Serum Lactate in Septic Shock: A Marker of Impaired Oxygen Utilization.

*by* Jana Publication & Research

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## <sup>29</sup>High Central Venous Oxygen Saturation and Elevated Serum Lactate in Septic Shock: A Marker <sup>of</sup> Impaired Oxygen Utilization.

### <sup>4</sup>ABSTRACT

**Background:** Septic shock is a life-threatening condition characterized by circulatory and metabolic abnormalities, where timely recognition of impaired tissue oxygenation is essential for effective resuscitation. <sup>28</sup>Central venous oxygen saturation (ScvO<sub>2</sub>) is commonly used as a surrogate marker of oxygen balance; however, its reliability as a standalone indicator in sepsis remains controversial. Lactate levels are often used to detect occult hypoperfusion, and the role of arterial oxygen saturation (SaO<sub>2</sub>) in this context is unclear.

**Objectives:** <sup>19</sup>To evaluate the correlation between ScvO<sub>2</sub>, SaO<sub>2</sub>, and serum lactate levels in patients with septic shock, and assess the utility of combined monitoring for guiding resuscitation.

**Methods and Methodology:** <sup>2</sup>This prospective observational study was conducted over three months in the intensive care unit of Bapuji Hospital, Davanagere enrolling 50 adult <sup>31</sup>patients with septic shock as defined by Sepsis-3 criteria. ScvO<sub>2</sub>, SaO<sub>2</sub>, and serum <sup>21</sup>lactate levels were measured simultaneously at 1hr, 6hr and 12 or 24 hours after admission with diagnosis of shock. Patients were categorized based on ScvO<sub>2</sub> levels ( $\leq 85\%$  and  $>85\%$ ), and statistical analyses included group comparisons and correlation analysis using Pearson or Spearman coefficients.

**Results:** This study involved 50 participants, where patients with ScvO<sub>2</sub>  $>85\%$  had significantly higher median serum <sup>40</sup>lactate levels (4.1 mmol/L) compared to those with ScvO<sub>2</sub>  $\leq 85\%$  <sup>17</sup>(2.1 mmol/L;  $P < 0.001$ ), despite similar demographic and clinical profiles. SaO<sub>2</sub> was comparable between groups ( $P = 0.21$ ). Correlation analysis revealed a moderate positive

relationship between ScvO<sub>2</sub> and serum lactate ( $r = 0.56$ ,  $P < 0.001$ ), while no significant correlation was found between ScvO<sub>2</sub> and SaO<sub>2</sub> ( $r = 0.14$ ,  $P = 0.32$ ).

**Conclusion:** Our findings demonstrated that high ScvO<sub>2</sub> values in septic shock may reflect impaired oxygen extraction rather than adequate perfusion, as indicated by associated elevated serum lactate levels. SaO<sub>2</sub> does not correlate with tissue perfusion markers. These findings support the use of combined ScvO<sub>2</sub> and serum lactate monitoring for better evaluation of oxygenation and guide resuscitation in septic shock.

**Keywords:** *Septic shock, Central venous oxygen saturation, Lactate, Arterial oxygen saturation, Tissue perfusion, Resuscitation, Sepsis monitoring.*

## **INTRODUCTION**

<sup>7</sup> Life-threatening organ dysfunction due to dysregulated host response to infection is known as sepsis. An acute change in the total quick sequential organ failure assessment score of  $\geq 2$  as a result of infection is considered organ dysfunction. A kind of <sup>11</sup> sepsis known as septic shock occurs when underlying circulatory and cellular/metabolic abnormalities are severe enough to significantly raise mortality <sup>(1)</sup>. Between 56 to 91 cases of sepsis and septic shock occur annually in adults per 100,000 people <sup>(2)</sup>. In individuals with septic shock, the short-term death rate can exceed 50% and is typically between 20% and 30% <sup>(3)</sup>.

<sup>13</sup> A sensitive but nonspecific marker of metabolic stress is the amount of serum lactate. Lactate, a byproduct <sup>14</sup> of anaerobic glycolysis, is elevated in hypoxia, stress, and a number of serious disorders. Higher lactate levels are positively correlated with higher mortality, according to recent research, <sup>13</sup> the higher the lactate level, the more adverse the outcome <sup>(4-7)</sup>.

Lactate has been demonstrated to be a useful indicator of tissue hypoxia in studies with critically ill patients <sup>(8)</sup>. An additional indicator is central venous oxygen saturation (ScvO<sub>2</sub>), which serves as a stand-in for venous return oxygenation and, consequently, oxygen

supply and tissue consumption<sup>(9)</sup>. This study aims to evaluate<sup>3</sup> the correlation between ScvO<sub>2</sub>, SaO<sub>2</sub>, and serum lactate levels in patients with septic shock, with the goal of refining monitoring strategies and optimizing resuscitation.

### <sup>35</sup>**OBJECTIVE OF THE STUDY**

This study was conducted to analyze<sup>3</sup> the correlation between ScvO<sub>2</sub>, SaO<sub>2</sub>, and serum lactate levels in patients with septic shock at different stages of resuscitation.

### **METHODOLOGY**

<sup>2</sup>This prospective, observational study was conducted in the intensive care unit (ICU) of Bapuji Hospital, Davanagere over a three-month period from April to June 2025. The study included fifty adult patients admitted with a diagnosis<sup>33</sup> of septic shock as defined by the Sepsis-3 criteria. All enrolled patients required central venous catheterization for hemodynamic monitoring and clinical management as part of their standard care.

<sup>10</sup>The study protocol was reviewed and approved by the Institutional Review Board and written informed consent was obtained from all patients prior to participation. Informed consent of participating individuals was obtained.<sup>30</sup> Inclusion criteria were: age 18 years or older, confirmed or suspected infection, persistent hypotension requiring vasopressor support after adequate fluid resuscitation, and<sup>44</sup> the placement of a central venous catheter within one hour of shock recognition.<sup>37</sup> Patients were excluded if they had pre-existing chronic renal or hepatic failure, were experiencing active hemorrhage or had undergone recent major surgery, had a do-not-resuscitate (DNR) status at the time of enrollment.

Upon inclusion, demographic details and baseline clinical data were recorded, including<sup>15</sup> age, sex, comorbidities, Acute Physiology and Chronic Health Evaluation II (APACHE II) score, and qSequential Organ Failure Assessment (qSOFA) score. Blood sampling for central venous oxygen saturation (ScvO<sub>2</sub>), arterial oxygen saturation (SaO<sub>2</sub>), and serum lactate levels was performed simultaneously at three standardized time points: 1 hour,

6 hours, and 12 or 24 hours after the diagnosis of septic shock. ScvO<sub>2</sub> samples were obtained from the central venous catheter, whereas SaO<sub>2</sub> and lactate measurements were taken from arterial blood samples. All patients received standard treatment in accordance with contemporary sepsis management guidelines, including fluid resuscitation, vasopressor support, and empiric antimicrobial therapy.

<sup>5</sup> The data was collected and compiled in MS Excel. Descriptive statistics has been used to present the data. To analyse the data SPSS (Version 26.0) was used. Correlation coefficients were calculated using Pearson or Spearman methods, <sup>8</sup> as appropriate, and a P-value of less than 0.05 was considered statistically significant. Additionally, patients were stratified into two subgroups based on ScvO<sub>2</sub> levels ( $\leq 85\%$  and  $>85\%$ ) to further explore clinical and laboratory associations.

## **RESULTS**

<sup>39</sup> The study included 50 patients diagnosed with septic shock, stratified into two groups based on central venous oxygen saturation (ScvO<sub>2</sub>) levels: ScvO<sub>2</sub>  $\leq 85\%$  (n=35) and ScvO<sub>2</sub>  $>85\%$  (n=15). Demographic characteristics were comparable between the two groups as depicted in Table 1. The median age was similar (63 vs. 60 years,  $P = 0.68$ ), with a slightly higher proportion of males in the ScvO<sub>2</sub>  $\leq 85\%$  group (60% vs. 53%,  $P = 0.63$ ), though <sup>49</sup> this difference was not statistically significant. <sup>32</sup> The severity of illness, as reflected by the APACHE II and qSOFA scores, was also similar between groups. Median APACHE II scores were 23 vs. 26 ( $P = 0.18$ ), and qSOFA scores were 10 vs. 11 ( $P = 0.45$ ).

As seen in Table 1, the ScvO<sub>2</sub> values were <sup>26</sup> significantly different between the two groups, with the high ScvO<sub>2</sub> group demonstrating a median saturation of 88% <sup>16</sup> compared to 76% in the lower ScvO<sub>2</sub> group ( $P < 0.001$ ). Interestingly, the SaO<sub>2</sub> values remained consistently high across both groups (median 96% vs. 97%,  $P = 0.21$ ), indicating that arterial oxygen saturation was not significantly affected by ScvO<sub>2</sub> level and was not a distinguishing

parameter in this septic cohort. A notable and clinically significant finding was the marked difference in serum lactate levels between the two groups. Patients with ScvO<sub>2</sub> >85% had a median lactate of 4.1 mmol/L<sup>38</sup>, significantly higher than the 2.1 mmol/L observed in the group with lower ScvO<sub>2</sub><sup>24</sup> ( $P < 0.001$ ).

**Table 1: Characteristics of the study participants (n=50)**

Variable	All Patients (n=50)	ScvO <sub>2</sub> ≤85% (n=35)	ScvO <sub>2</sub> >85% (n=15)	P-value
Age (years)	62 (52–75)	63 (54–74)	60 (51–78)	0.68
Male (%)	29 (58%)	21 (60%)	8 (53%)	0.63
APACHE II score	24 (18–29)	23 (17–28)	26 (19–31)	0.18

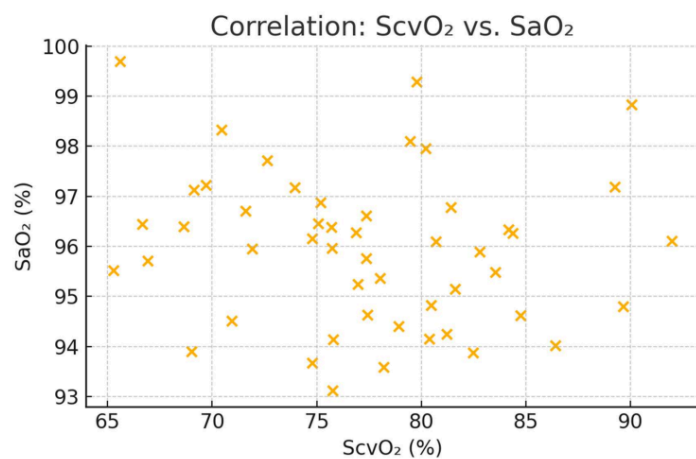
<b>SOFA score</b>	10 (8–13)	10 (8–13)	11 (8–14)	0.45
<b>ScvO<sub>2</sub> (%)</b>	79 (72–87)	76 (71–82)	88 (86–90)	<b>&lt;0.001</b>
<b>SaO<sub>2</sub> (%)</b>	96 (95–98)	96 (95–98)	97 (96–98)	0.21
<b>Lactate (mmol/L)</b>	2.5 (1.7–3.6)	2.1 (1.5–2.8)	4.1 (3.1–5.3)	<b>&lt;0.001</b>

Table 2 shows the relationships among oxygenation and metabolic markers, where correlation coefficients were calculated. ScvO<sub>2</sub> and SaO<sub>2</sub> demonstrated a weak, non-significant correlation (r = 0.14, P = 0.32; Figure 1) and a moderate positive correlation was observed between ScvO<sub>2</sub> and serum lactate levels (r = 0.56, P < 0.001; Figure 2).

**Table 2: Correlation Analysis (n=50)**

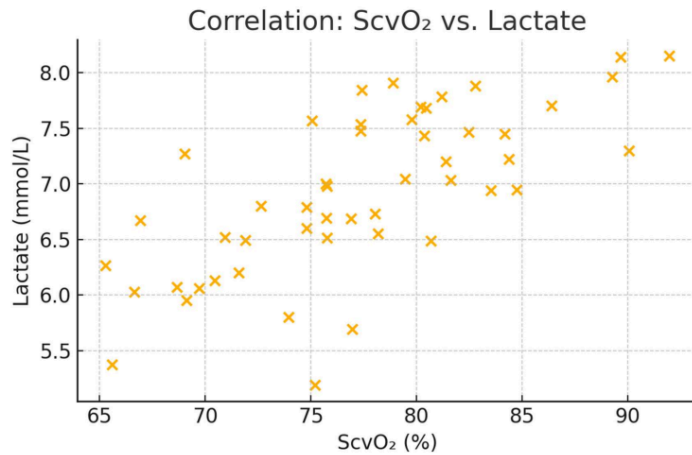
Variable Pairs	Correlation (r)	P-value
<b>ScvO<sub>2</sub> vs. SaO<sub>2</sub></b>	0.14	0.32
<b>ScvO<sub>2</sub> vs. Lactate</b>	0.56	<b>&lt;0.001</b>

**Figure 1: Correlation of ScvO<sub>2</sub> vs. SaO<sub>2</sub> (n=50)**



**Figure 2: Correlation of ScvO<sub>2</sub> vs. Lactate (n=50)**





## **DISCUSSION**

As both the number of sepsis-related deaths and the incidence of sepsis are rising. The onset of multiple organ failure syndrome often complicates <sup>23</sup> severe sepsis and septic shock. Multiple organ dysfunction syndrome[MODS] is thought to emerge as a result of global tissue hypoxia <sup>(10-12)</sup>.

<sup>41</sup> Our study provides valuable insights into the complex interplay between <sup>18</sup> central venous oxygen saturation (ScvO<sub>2</sub>), arterial oxygen saturation (SaO<sub>2</sub>), <sup>3</sup> and serum lactate levels in patients with septic shock. A key finding in our study was the paradoxical association between elevated ScvO<sub>2</sub> and increased serum lactate concentrations, suggesting that high arterial oxygen saturation may not reliably indicate adequate tissue oxygenation. High <sup>47</sup> Arterial Oxygen Saturation and elevated Central Venous Oxygen Saturation may reflect impaired oxygen extraction and underlying metabolic dysfunction, which are hallmarks of advanced septic shock.

Although ScvO<sub>2</sub> has traditionally <sup>34</sup> been used as a surrogate for the balance between oxygen delivery and consumption, our results challenge its interpretation when used in isolation. Our study reported a median ScvO<sub>2</sub> of 79% (IQR: 72–87%) and a median lactate level of <sup>27</sup> 2.5 mmol/L (IQR: 1.7–3.6) among 50 patients with septic shock. <sup>1</sup> In the present study, patients with ScvO<sub>2</sub> >85% demonstrated significantly higher median serum lactate levels compared to those with ScvO<sub>2</sub> ≤85% (<sup>17</sup> 4.1 vs. 2.1 mmol/L, P < 0.001), despite comparable <sup>1</sup> baseline characteristics such as age, sex, and illness severity scores (APACHE II and qSOFA). This suggests that elevated ScvO<sub>2</sub> values may, in some cases, reflect cellular or microcirculatory dysfunction rather than effective perfusion, as oxygen is not being adequately utilized at the tissue level.

Similarly, Permpikul et al. <sup>(17)</sup> also found that patients with ScvO<sub>2</sub> >85% had the highest lactate concentrations among all saturation groups, reinforcing the concept that high ScvO<sub>2</sub> is not necessarily reassuring in septic shock. Their study noted <sup>43</sup> no significant correlation between ScvO<sub>2</sub> and lactate at any time point, highlighting a decoupling between systemic oxygen delivery and metabolic recovery. Moreover, despite no significant changes in ScvO<sub>2</sub> values over time, serum lactate levels gradually decreased, with a calculated 15.4% clearance by the third time point—underscoring the usefulness of lactate as a dynamic and responsive marker.

In comparison, the Sneha et al. <sup>(13)</sup> study, which included a cohort of 130 patients, reported a mean ScvO<sub>2</sub> of 62.38% (SD: 27.86%) and a markedly higher baseline mean lactate level of 8.01 mmol/L (SD: 2.56). This suggests that their cohort may have presented with more severe metabolic disturbances at admission. The wider variability in ScvO<sub>2</sub> in their study could also reflect the inclusion of patients earlier in the shock trajectory or differences in the timing of measurements.

Also, Shi et al.<sup>(12)</sup><sup>(18)</sup> conducted a study in 37 patients with sepsis or septic shock, stratified by shock status (n=20), and outcomes (survivors n=26 vs. non-survivors n=11) and the overall Pearson correlation between ScvO<sub>2</sub> and serum lactate was positive ( $r = 0.37$ ,  $P < 0.001$ ), indicating a moderate inverse linear relationship in their study and they found no significant difference in ScvO<sub>2</sub> levels between sepsis and shock groups ( $P > 0.05$ ), but lactate was significantly higher in the shock group ( $P < 0.05$ ).

The moderate positive correlation between ScvO<sub>2</sub> and serum lactate ( $r = 0.56$ ,  $P < 0.001$ ) in the current study<sup>(48)</sup> indicating that higher ScvO<sub>2</sub> values were associated with higher lactate levels, a paradoxical but increasingly recognized feature in septic shock due to impaired cellular oxygen extraction. However, in Sneha et al.<sup>(13)</sup> study, a significant negative correlation was consistently observed between ScvO<sub>2</sub> and serum lactate across multiple time points, with r values ranging from -0.481 at baseline to -0.504 after achieving CVP targets, and -0.356 after achieving MAP targets.

At the same time, in the intensive care unit of a tertiary hospital, Park et al.<sup>(14)</sup> carried out a retrospective analysis in which they observed 65 consecutive patients with septic shock and severe sepsis without any medical intervention. After the resuscitation period, they found that both groups' ScvO<sub>2</sub> levels were higher than 70%, with the exception of the nonsurvivors group's second day (69.8%). Following the second day, the survivors' group's ScvO<sub>2</sub> was considerably greater ( $P < 0.001$ ). Both groups' lactate levels were comparable throughout their evolution and they came to the conclusion that while blood lactate levels are still used as outcome indicators for patients who have been resuscitated from severe sepsis and septic shock.

Similar to our study, a prospective observational cohort study was conducted by Mahajan et al.<sup>(15)</sup> in the high dependency and medical and surgical intensive care units of a tertiary care hospital in India over the course of eight months, where they came to the

conclusion that there was no proof that the ScvO<sub>2</sub> values had changed over time ( $P = 0.063$ ) and over time, serum lactate considerably dropped ( $P < 0.001$ ), with survivors experiencing a more marked decline than nonsurvivors ( $P < 0.001$ ). Also, serum Lactate and AG had a substantial link with one another in septic shock patients, while serum lactate and BE exhibited a moderate correlation, according to the Pongmancee and Vattanavanit<sup>(16)</sup> research.

In the present study, arterial oxygen saturation (SaO<sub>2</sub>) remained uniformly high across both groups, with no statistically significant difference between them ( $P = 0.21$ ). Moreover, the correlation between ScvO<sub>2</sub> and SaO<sub>2</sub> was weak and non-significant ( $r = 0.14$ ,  $P = 0.32$ ) in our study, indicating that SaO<sub>2</sub> is not a useful indicator of tissue-level oxygen utilization in septic shock. This finding emphasizes that systemic oxygenation, as measured by arterial saturation, may not reflect the adequacy of oxygen delivery at the microcirculatory or cellular level—where dysfunction is often profound in sepsis.

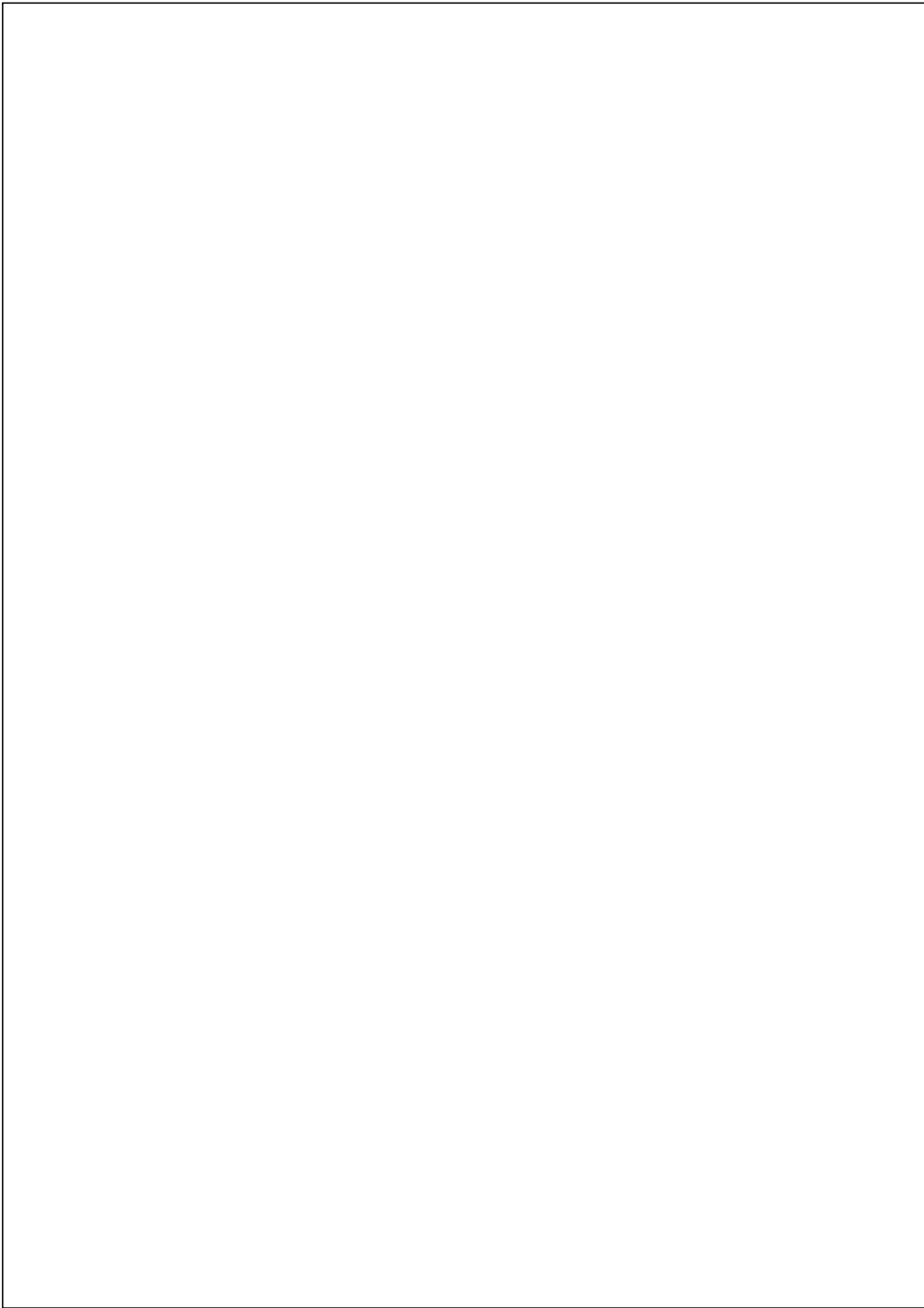
## **CONCLUSION**

In this prospective observational study of patients with septic shock, we found that elevated central venous oxygen saturation (ScvO<sub>2</sub> >85%) was significantly associated with higher serum lactate levels, suggesting impaired tissue oxygen utilization despite seemingly adequate oxygen delivery. Conversely, arterial oxygen saturation (SaO<sub>2</sub>) showed no meaningful correlation with either ScvO<sub>2</sub> or serum lactate, underscoring its limited value in assessing tissue perfusion in this setting. These findings highlight the limitations of relying on ScvO<sub>2</sub> alone as a marker of resuscitation adequacy in septic shock. Instead, a combined assessment of ScvO<sub>2</sub> and serum lactate offers a more accurate evaluation of oxygenation and metabolic status, helping to detect occult hypoperfusion and guide timely, targeted therapeutic interventions. Integrating both parameters into routine monitoring may enhance clinical decision-making and improve outcomes in critically ill septic patients.

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