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Article

Prognostic Value of Lactate, Standard Base Excess, and Alactic Base Excess in Sepsis and Septic Shock: A Retrospective Cohort Study

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Abstract: Sepsis and septic shock are severe medical conditions associated with high morbidity and mortality, necessitating accurate risk stratification to optimize clinical management. Lactate is a well-established biomarker for sepsis prognosis, reflecting tissue hypoxia and anaerobic metabolism. Standard Base Excess (SBE) is commonly used to assess metabolic acidosis, but its prognostic value in sepsis remains uncertain, particularly in the presence of elevated lactate levels. Alactic Base Excess (aBE), calculated as SBE + lactate, has been proposed as an alternative marker to differentiate non-lactate metabolic derangements. This retrospective cohort study aimed to compare the prognostic accuracy of lactate, SBE, and aBE in predicting mortality among patients with sepsis and septic shock. A total of 218 patients admitted to the intensive care unit (ICU) were analyzed, including 90 survivors and 128 non-survivors. Elevated lactate levels were significantly associated with mortality ($p < 0.001$) and remained an independent predictor in multivariate analysis (odds ratio [OR]: 1.40, 95% confidence interval [CI]: 1.11–1.77, $p = 0.005$). SBE showed a weak association with mortality in univariate analysis but lost significance in multivariate models. ABE did not demonstrate significant prognostic value. Receiver Operating Characteristic (ROC) analysis confirmed lactate as the most reliable predictor, with an area under the curve (AUC) of 0.742. These findings suggest that lactate remains the superior prognostic biomarker in sepsis and septic shock, whereas SBE and aBE do not provide additional predictive value. Further research is needed to explore the dynamic changes in these biomarkers over time to refine risk stratification in septic patients.

Keywords: sepsis; septic shock; lactate; standard base excess; alactic base excess; prognosis; mortality prediction

1. Introduction

Sepsis and septic shock are life-threatening conditions associated with high morbidity and mortality, requiring prompt diagnosis and appropriate management. Despite advancements in critical care medicine, risk stratification remains a significant challenge, as early identification of high-risk patients is crucial for timely interventions. Various prognostic biomarkers have been investigated to improve mortality prediction in sepsis, with arterial blood gas (ABG) parameters playing a central role in assessing metabolic derangements [1].

Lactate is a well-established biomarker in sepsis, reflecting tissue hypoxia, impaired oxygen utilization, and anaerobic metabolism [2]. Elevated lactate levels are consistently associated with poor clinical outcomes and have been incorporated into sepsis management guidelines. However, lactate alone may not provide a comprehensive assessment of metabolic disturbances in septic patients, particularly in the presence of concurrent acid-base imbalances [3].

Standard Base Excess (SBE) is widely used to evaluate metabolic acid-base disorders by quantifying the amount of acid or base required to return blood pH to 7.4 under standard conditions. However, its interpretation becomes complex in sepsis, where elevated lactate levels significantly influence metabolic acidosis. To address this limitation, Alactic Base Excess (aBE) has been proposed as an alternative parameter that accounts for non-lactate metabolic derangements by incorporating lactate into the base excess calculation ($aBE = SBE + \text{lactate}$). Theoretically, aBE could offer a more accurate assessment of metabolic imbalances unrelated to lactate, such as hyperchloremic acidosis or renal dysfunction [4].

Although lactate, SBE, and aBE are all relevant biomarkers in critically ill patients, their comparative prognostic value in sepsis and septic shock remains inadequately explored. Previous studies have primarily focused on lactate as an isolated predictor of mortality, while the role of SBE and aBE in risk stratification is less well-defined. Moreover, existing research has yielded conflicting results regarding the clinical utility of these parameters in sepsis [5,6]. Therefore, further investigation is needed to determine whether SBE and aBE provide additional prognostic value beyond lactate in critically ill septic patients.

This study aimed to compare the effectiveness of lactate, SBE, and aBE in predicting mortality in sepsis and septic shock and to explore the relationships between these biomarkers.

2. Materials and methods

2.1. Study Design and Participants

This retrospective cohort study was conducted in the medical intensive care unit of Ondokuz Mayıs University Faculty of Medicine between 2022 and 2023. Patients diagnosed with sepsis or septic shock according to the Sepsis-3 criteria were included. Inclusion criteria were age ≥ 18 years and availability of arterial blood gas analysis at ICU admission. Exclusion criteria included terminal-stage palliative care patients, those who died within the first 24 hours of ICU admission, and cases with incomplete or missing medical records.

2.2. Data Collection

All patient data, including demographic characteristics, severity scores, and laboratory findings, were retrospectively collected from electronic medical records. The severity of illness was assessed using the Acute Physiology and Chronic Health Evaluation II (APACHE II) score, which estimates disease severity based on physiological parameters, and the Sequential Organ Failure Assessment (SOFA) score, which evaluates the extent of organ dysfunction in critically ill patients. Patients were categorized into survivors and non-survivors, and the following variables were analyzed: age, sex, APACHE II, SOFA, and Charlson Comorbidity Index scores; serum creatinine, sodium, and chloride levels; as well as arterial blood gas parameters, including pH, bicarbonate, standard base excess (SBE), lactate, and alactic base excess (aBE), which was calculated as $SBE + \text{lactate}$.

Supportive therapies, including the use of vasopressors, invasive mechanical ventilation, and hemodialysis, were also recorded. The length of ICU stay was measured in days. To address missing data, variables with more than 5% missing values were excluded from the analysis, while those with less than 5% missing data were handled using multiple imputation techniques.

2.3. Statistical Analysis

Statistical analysis was performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed variables were compared using the independent t-test, and results were reported as mean \pm standard deviation. Non-normally distributed variables were analyzed using the Mann-Whitney U test, with results presented as median (interquartile range). Categorical variables were compared using the chi-square test and reported as frequency (percentage).

To evaluate the prognostic value of lactate, SBE, and aBE in predicting mortality, receiver operating characteristic curve analysis was conducted. The area under the curve was calculated, and the optimal cut-off value was determined using the Youden Index. Independent predictors of mortality were identified using multivariate logistic regression analysis. Variables with $p < 0.10$ in univariate analysis were included in the model. Results were reported as odds ratios with 95% confidence intervals. A p -value < 0.05 was considered statistically significant.

3. Results

A total of 218 patients diagnosed with sepsis or septic shock were included in the study, with 90 survivors and 128 non-survivors. There was no significant difference in age ($p = 0.990$) or sex distribution ($p = 0.074$) between survivors and non-survivors. However, non-survivors had significantly higher disease severity scores, including APACHE II (28 [23–33] vs. 23 [18–27], $p < 0.001$) and SOFA (12 [9–14] vs. 9 [7–12], $p < 0.001$), compared to survivors (Table 1).

Table 1. Demographic and clinical characteristics between survivors and non-survivors in patients with sepsis and septic shock.

Variables ^a	Survivors N=90	Nonsurvivors N=128	P value univariate
Age	71 (60-80)	71 (59-79)	0.990
Male	41 (46)	74 (58)	0.074
APACHE II	23 (18-27)	28 (23-33)	<0.001
SOFA	9 (7-12)	12 (9-14)	<0.001
Charlson Comorbidity Index	3 (2-5)	3 (2-5)	0.978
Creatinin	2.33 (0.96-3.79)	1.9 (1.3-2.9)	0.401
Serum sodium	134 (138-144)	139 (134-144)	0.704
Serum chloride	101 (96-107)	101 (94-106)	0.472
PH	7.38 (7.30-7.46)	7.38 (7.27-7.46)	0.717

Standart HCO ₃	22 (18-25)	20 (16-24)	0.030
SBE	-2.8 (-7.6 - +0.9)	-5.1 (-9.8 - -1)	0.046
Lactate	1.2 (1.8-2.9)	2.9 (2.3 - 4.1)	<0.001
ABE	-0.5 (-4.8- +2.2)	-1.6 (-5.6 - +2.2)	0.551
Vasopressor use	64 (71.1)	124 (97)	<0.001
IMV	34 (38)	110 (86)	<0.001
Hemodialysis	32 (36)	73 (57)	0.002
Length of ICU days	7 (4-13)	8 (4-16)	0.166

^a Data are presented as median (interquartile range) for continuous variables and n (%) for categorical variables. **Abbreviations:** APACHE II, Acute Physiology and Chronic Health Evaluation II; SOFA, Sequential Organ Failure Assessment; HCO₃, bicarbonate; SBE, standard base excess; ABE, alactic base excess; IMV, invasive mechanical ventilation; ICU, intensive care unit.

Regarding biochemical markers, standard bicarbonate levels were significantly lower in non-survivors (20 [16–24] vs. 22 [18–25], $p = 0.030$). Similarly, SBE values were significantly more negative in non-survivors (-5.1 [-9.8 - -1] vs. -2.8 [-7.6 - +0.9], $p = 0.046$). The most striking difference was observed in lactate levels, which were significantly higher in non-survivors (2.9 [2.3–4.1] vs. 1.2 [1.8–2.9], $p < 0.001$). However, aBE values did not significantly differ between groups (-1.6 [-5.6 - +2.2] vs. -0.5 [-4.8 - +2.2], $p = 0.551$) (Table 1). Non-survivors required significantly more vasopressor support (97% vs. 71.1%, $p < 0.001$), invasive mechanical ventilation (86% vs. 38%, $p < 0.001$), and hemodialysis (57% vs. 36%, $p = 0.002$) compared to survivors. The median ICU stay did not significantly differ between groups (8 [4–16] vs. 7 [4–13] days, $p = 0.166$).

In the logistic regression analysis, higher APACHE II scores were significantly associated with increased mortality risk (OR: 1.08, 95% CI: 1.02–1.15, $p = 0.013$). Similarly, elevated lactate levels were found to be a significant predictor of mortality (OR: 1.40, 95% CI: 1.11–1.77, $p = 0.005$). Vasopressor use was also associated with a higher risk of mortality (OR: 1.32, 95% CI: 1.05–1.68, $p = 0.028$). Additionally, invasive mechanical ventilation was identified as a significant factor linked to increased mortality (OR: 1.45, 95% CI: 1.10–1.92, $p = 0.012$) (Table 2).

Table 2. Logistic Regression Analysis of Factors Associated with Mortality.

Variables	OR	%95 CI	P value
APACHE II score	1.08	1.02-1.15	0.013
Lactate level	1.40	1.11-1.77	0.005
Vasopressore use	1.32	1.05-1.68	0.028
IMV	1.45	1.10-1.92	0.012

Abbreviations: APACHE II; Acute Physiology and Chronic Health Evaluation II, OR; Odds Ratio, CI; Confidence Interval, IMV; Invasive Mechanical Ventilation.

Although both lactate and SBE showed a significant association with mortality in univariate analysis (Table 1), only lactate remained an independent predictor in multivariate logistic regression analysis (Table 2). aBE was not associated with mortality (Table 1). Therefore, ROC curve analysis for aBE was not performed, and the predictive value of SBE was limited.

The receiver operating characteristic curve analysis for lactate in patients with sepsis and septic shock showed an area under the curve (AUC) of 0.742 (95% CI: 0.672–0.812, $p < 0.001$), indicating good predictive ability for mortality. In contrast, the AUC for SBE was 0.421 (95% CI: 0.343–0.498, $p = 0.046$), suggesting poor diagnostic performance (Figure 1). The optimal cutoff value for lactate was determined as 2.0 mmol/L, with a sensitivity of 86.7% and a specificity of 60.0%.

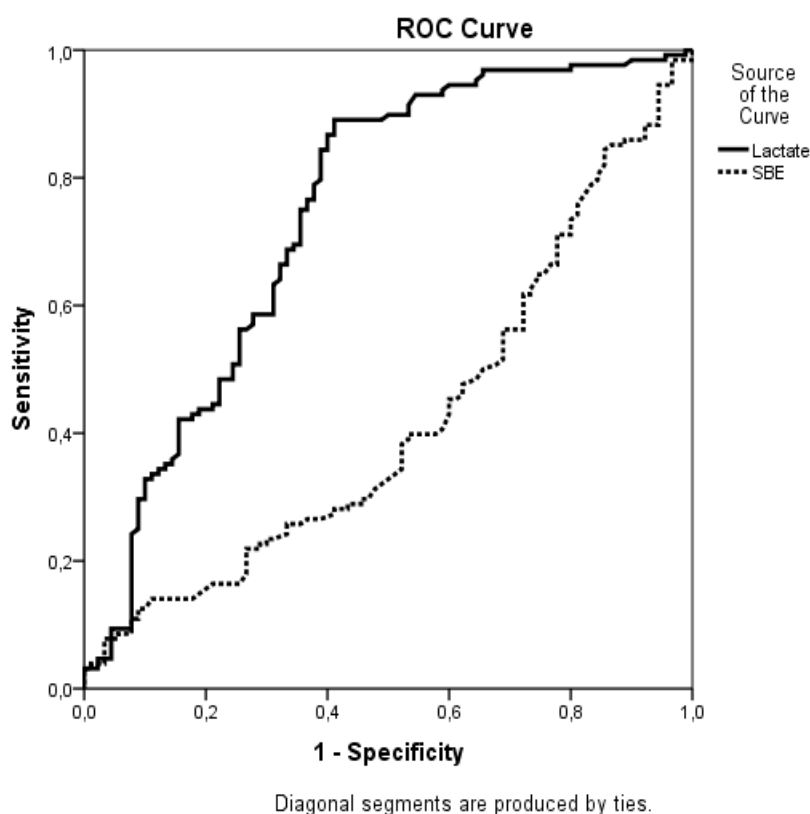


Figure 1. Receiver Operating Characteristic (ROC) Curve for lactate and SBE in patients with sepsis and septic shock. The area under the curve (AUC) for lactate is 0.742 (95% CI: 0.672–0.812; $p < 0.001$). The AUC for SBE is 0.421 (95% CI: 0.343–0.498; $p = 0.046$).

4. Discussion

In this study, we investigated the prognostic value of lactate, standard base excess (SBE), and alactic base excess (aBE) in predicting mortality among patients with sepsis and septic shock. Our findings indicate that lactate is a strong and independent predictor of mortality, whereas SBE and aBE do not provide additional prognostic value. These results have significant implications for risk stratification in sepsis and the use of metabolic biomarkers in clinical practice.

Unlike previous studies, our findings align with existing evidence supporting lactate as a critical biomarker in sepsis prognosis [8–10]. Research has shown that lactate levels above 2 mmol/L are associated with significantly increased hospital mortality, even in hemodynamically stable sepsis patients [11]. Additionally, meta-analyses have suggested that changes in lactate levels over time may be more predictive of mortality than a single measurement [12,13]. However, unlike studies evaluating lactate kinetics, this analysis focuses solely on the initial lactate value at ICU admission without assessing variations over time.

Lactate is a well-known biomarker of impaired oxygen utilization, mitochondrial dysfunction, and anaerobic metabolism [14]. Among the patients analyzed, non-survivors had significantly higher lactate levels than survivors, with this difference being statistically significant. The receiver operating characteristic analysis confirmed the strong predictive performance of lactate, with an optimal cutoff of 2.0 mmol/L, providing high sensitivity (86.7%) and acceptable specificity (60.0%). These findings reinforce the importance of early lactate monitoring in sepsis management and suggest that dynamic changes in lactate levels over time may further improve risk assessment.

SBE quantifies the amount of acid or base required to return blood pH to 7.4 under standard conditions, making it a widely used parameter for evaluating metabolic acidosis and overall acid-base balance [4,15]. Several studies have highlighted the clinical relevance of SBE in critically ill patients, particularly in sepsis and trauma, where metabolic acidosis often plays a crucial role in disease progression and outcomes [16,17].

In our analysis, while SBE was significantly associated with mortality in univariate analysis, this association was not maintained in multivariate models, suggesting that its prognostic value is largely overshadowed by lactate. This finding aligns with previous research indicating that lactate, as a marker of impaired tissue oxygenation and anaerobic metabolism, often outperforms SBE in predicting mortality in critically ill patients [8,10]. The stronger predictive role of lactate may stem from its direct reflection of tissue hypoxia and mitochondrial dysfunction, whereas SBE represents a more generalized measure of acid-base balance influenced by multiple metabolic factors.

Additionally, we analyzed aBE, which theoretically isolates non-lactate metabolic disturbances by excluding lactate from the calculation. However, aBE did not show a significant association with mortality. This finding is consistent with prior studies suggesting that while lactate-independent metabolic acidosis contributes to disease severity, its prognostic impact is less pronounced when compared to lactate-driven acidosis [18,19]. Given these results, lactate remains the dominant metabolic marker for risk stratification in critically ill patients, reinforcing the importance of dynamic lactate monitoring in clinical practice.

Several factors may explain the limited prognostic utility of SBE and aBE in sepsis. First, metabolic acidosis in sepsis is primarily driven by lactate accumulation. Since lactate is already a strong prognostic marker, SBE, which inherently incorporates lactate-driven acidosis, may not provide additional independent prognostic value. aBE, which aims to differentiate non-lactate acidosis, may be less relevant in sepsis, where lactate is the dominant driver of metabolic acidosis.

Another possible explanation for the lack of prognostic value in SBE and aBE is the influence of various compensatory mechanisms. Unlike lactate, which directly reflects tissue perfusion abnormalities and anaerobic metabolism, SBE and aBE are affected by multiple physiological adaptations, including renal bicarbonate regulation, respiratory compensation, and electrolyte disturbances [20]. For example, patients with sepsis-induced metabolic acidosis may develop compensatory respiratory alkalosis through hyperventilation, normalizing SBE values despite the severity of their condition. Similarly, renal compensation may buffer metabolic acidosis over time, modifying SBE and aBE values independently of sepsis severity [21]. These physiological adaptations

likely dilute the prognostic significance of SBE and aBE, explaining their weaker association with mortality compared to lactate.

Previous research on the prognostic value of SBE and aBE has yielded inconsistent results [22–25]. Some findings suggest that more negative SBE values, indicating severe metabolic acidosis, are associated with increased mortality in critically ill patients. However, differences in patient populations across studies complicate direct comparisons. While some analyses have identified SBE as a predictor of mortality in general ICU settings, its prognostic significance appears to diminish in sepsis-specific cases [22]. Similarly, base excess has shown relevance in trauma-related outcomes but lacks the same predictive strength in sepsis [25]. The present analysis contributes to this discussion by suggesting that SBE and aBE may not serve as reliable independent prognostic markers in sepsis, where lactate remains the dominant indicator. Further investigation is required to determine whether these parameters hold predictive value in specific subgroups, such as patients with concurrent renal failure or electrolyte imbalances.

One of the key strengths of our study is its focused comparison of three metabolic biomarkers within a well-defined cohort of sepsis and septic shock patients. The statistical analyses were robust, and the study design allowed for meaningful comparisons between survivors and non-survivors. However, our study also has several limitations.

First, its retrospective and single-center design may limit the generalizability of our findings. Additionally, we only assessed biomarker levels at ICU admission, without considering dynamic changes over time. Sepsis is a rapidly evolving condition, and serial biomarker measurements may provide better prognostic insights than a single baseline value. Future studies should investigate how trends in lactate, SBE, and aBE correlate with patient outcomes. Furthermore, we did not analyze the impact of sepsis etiology or coexisting conditions on biomarker performance. Since different infection sources and comorbidities can influence metabolic responses, future research should explore whether certain patient subgroups may benefit from SBE- or aBE-based prognostic assessment.

5. Conclusion

Our study confirms that lactate remains the strongest and most reliable predictor of mortality in sepsis and septic shock, while SBE and aBE do not provide additional prognostic value. These findings reinforce the importance of lactate-based risk stratification and suggest that SBE and aBE should be interpreted cautiously in sepsis patients. Future research should focus on incorporating serial biomarker measurements and exploring novel metabolic markers to enhance sepsis prognostication.

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References

1. Mukherjee S, Das S, Mukherjee S, Ghosh PS, Bhattacharya S. Arterial blood gas as a prognostic indicator in patients with sepsis. *Indian J Med Microbiol.* 2020;38[3-4]:457-460. doi:10.4103/ijmm.IJMM_19_478

2. De Backer D, Vincent JL. Understanding hyperlactatemia in human sepsis: Are we making progress? *Am J Respir Crit Care Med.* 2019;200[8]:1070-1071. doi:10.1164/rccm.201905-1024LE
3. Yang K, Fan M, Wang X, et al. Lactate induces vascular permeability via disruption of VE-cadherin in endothelial cells during sepsis. *Sci Adv.* 2022;8[17]:eabm8965. doi:10.1126/sciadv.abm8965
4. Gattinoni L, Vasques F, Camporota L, et al. Understanding lactatemia in human sepsis: Potential impact for early management. *Am J Respir Crit Care Med.* 2019;200[5]:582-589. doi:10.1164/rccm.201812-2342OC
5. Cantos J, Huespe IA, Sinner JF, et al. Alactic base excess is an independent predictor of death in sepsis: A propensity score analysis. *J Crit Care.* 2023;74:154248. doi:10.1016/j.jcrc.2022.154248
6. Smuszkiewicz P, Jawień N, Szrama J, Lubarska M, Kusza K, Guzik P. Admission lactate concentration, base excess, and alactic base excess predict the 28-day inward mortality in shock patients. *J Clin Med.* 2022;11[20]:6125. doi:10.3390/jcm11206125
7. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock [Sepsis-3]. *JAMA.* 2016;315[8]:801-810. doi:10.1001/jama.2016.0287
8. Jansen TC, van Bommel J, Schoonderbeek FJ, et al. Early lactate-guided therapy in intensive care unit patients: a multicenter, open-label, randomized controlled trial. *Am J Respir Crit Care Med.* 2010;182[6]:752-761. doi:10.1164/rccm.200912-1918OC
9. Shapiro NI, Howell MD, Talmor D, et al. Serum lactate as a predictor of mortality in emergency department patients with infection. *Ann Emerg Med.* 2005;45[5]:524-528. doi:10.1016/j.annemergmed.2004.12.006
10. Mikkelsen ME, Miltiades AN, Gaieski DF, et al. Serum lactate is associated with mortality in severe sepsis independent of organ failure and shock. *Crit Care Med.* 2009;37[5]:1670-1677. doi:10.1097/CCM.0b013e31819fcf68
11. Vincent JL, Quintairos e Silva A, Couto L, et al. The value of blood lactate kinetics in critically ill patients: a systematic review. *Crit Care.* 2016;20:257. doi:10.1186/s13054-016-1403-5
12. Zhang Z, Xu X. Lactate clearance is a useful biomarker for the prediction of all-cause mortality in critically ill patients: a systematic review and meta-analysis. *Crit Care Med.* 2014;42[9]:2118-2125. doi:10.1097/CCM.0000000000000405
13. Haas SA, Lange T, Saugel B, et al. Severe hyperlactatemia, lactate clearance and mortality in unselected critically ill patients. *Intensive Care Med.* 2016;42[2]:202-210. doi:10.1007/s00134-015-4127-0
14. Gladden LB. Lactate metabolism: a new paradigm for the third millennium. *J Physiol.* 2004;558[1]:5-30. doi:10.1113/jphysiol.2003.058701
15. Kellum JA. Determinants of blood pH in health and disease. *Crit Care.* 2000;4[1]:6-14. doi:10.1186/cc644
16. Noritomi DT, Soriano FG, Kellum JA, et al. Metabolic acidosis in patients with severe sepsis and septic shock: a longitudinal quantitative study. *Crit Care Med.* 2009;37[10]:2733-2739. doi:10.1097/ccm.0b013e3181a59165
17. Vincent JL, Moreno R. Clinical review: scoring systems in the critically ill. *Crit Care.* 2010;14[2]:207. doi:10.1186/cc8204
18. Andersen LW, Mackenhauer J, Roberts JC, et al. Etiology and therapeutic approach to elevated lactate levels. *Mayo Clin Proc.* 2013;88[10]:1127-1140. doi:10.1016/j.mayocp.2013.06.012
19. Al-Jaghbeer M, Kellum JA. Acid-base disturbances in intensive care patients: etiology, pathophysiology, and treatment. *Nephrol Dial Transplant.* 2015;30[7]:1104-1111. doi:10.1093/ndt/gfu289
20. Seifter JL, Chang HY. Disorders of acid-base balance: new perspectives. *Kidney Dis [Basel].* 2017;2[4]:170-186. doi:10.1159/000453028
21. Dubin A, Meneses MM, Masevicius FD, et al. Comparison of three different methods of evaluation of metabolic acid-base disorders. *Crit Care Med.* 2007;35[5]:1264-1270. doi:10.1097/01.CCM.0000259536.11943.90
22. Cusack RJ, Rhodes A, Lochhead P, et al. The strong ion gap does not have prognostic value in critically ill patients in a mixed medical/surgical adult ICU. *Intensive Care Med.* 2002;28[7]:864-869. doi:10.1007/s00134-002-1318-2
23. Park M, Maciel AT, Noritomi DT, et al. Effect of PaCO₂ variation on standard base excess value in critically ill patients. *J Crit Care.* 2009;24[4]:484-491. doi:10.1016/j.jcrc.2008.12.018

24. Park M, Taniguchi LU, Noritomi DT, et al. Clinical utility of standard base excess in the diagnosis and interpretation of metabolic acidosis in critically ill patients. *Braz J Med Biol Res.* 2008;41[3]:241-249. doi:10.1590/s0100-879x2006005000199
25. Diao MY, Wang T, Cui YL, Lin ZF. *Zhonghua Wei Zhong Bing Ji Jiu Yi Xue.* 2013;25[4]:211-214. doi:10.3760/cma.j.issn.2095-4352.2013.04.008

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