

Evaluation of arterial blood gases of patients with type 1-2 respiratory failure diagnosed in intensive care using the quantitative Stewart method

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ABSTRACT

Introduction: Arterial blood gases are tests that provide reliable information about the metabolic and respiratory status of patients. In traditional methods, arterial blood gases are evaluated by calculating the bicarbonate concentration and anion gap in the plasma. Since ICU patients almost always have protein and electrolyte disorders, a different method has been suggested instead of the traditional method that neglects electrolyte and protein in the evaluation. In the mathematical model described by Stewart and modified by Figge et al., 4 types of respiratory failure were defined according to their pathophysiology: hypoxemic (type 1), hypercapnic (type 2), perioperative (type 3), and hypoperfusion-induced respiratory failure in shock patients (type 4). The study aims to evaluate the arterial blood gases of intensive care patients with type 1 and type 2 respiratory failure with the Stewart method and compare them with the traditional method.

Material and Method: In the study, serum BUN (blood urea nitrogen), creatinine, glucose, sodium, chlorine, serum BUN (blood urea nitrogen) of 106 patients diagnosed with type 1-2 respiratory failure. Together with potassium, magnesium, albumin values, pH, pCO₂, pO₂, HCO₃, base deficit, and lactate values studied in arterial blood gas sample were determined and evaluated according to the Stewart method.

Results: The length of stay of the patients in the intensive care unit was determined as 17.48±10.58 (minimum 6-maximum 68) days. It was determined that 74 (69.8%) of the patients were discharged and 32 (30.2%) died. When the blood gases and laboratory values of the patients were compared according to the clinical outcomes of the patients, no statistically significant correlation was found between the patients' mean pH, pCO₂, HCO₃, base deficit, pO₂, albumin, lactate, creatinine, glucose, potassium, and chlorine values and the patients' discharge or death. BUN values of patients who died were found to be higher than those who were discharged. A statistically significant difference was found between the development of hypoalbuminemia and the length of stay in the intensive care unit. It has been determined that the treatment costs of patients with severe hypoalbuminemia are higher than other patients. The length of stay in the intensive care unit of the patients who did not develop acidosis was found to be statistically significantly lower than the patients who developed metabolic and respiratory acidosis.

Conclusion: While a significant relationship was found between albumin levels, which has an important place in the Stewart method, and the length of stay in the intensive care unit, no relationship was found between albumin levels or lactate levels and mortality. However, increased BUN values were associated with mortality. As the albumin value decreases in patients with hypoalbuminemia, treatment costs increase. There is a need for larger multicenter studies with a larger sample group that will evaluate metabolic status with the Stewart method and investigate its relationship with mortality.

Keywords: Arterial blood gas, respiratory failure, Stewart method

INTRODUCTION

Respiratory failure is a syndrome characterized by the deterioration in the ability of the respiratory system to maintain its functions. and the inability of the tissues to provide oxygen (O₂) or to remove carbon dioxide (CO₂) to meet their needs (1). Many diseases can cause respiratory failure with different physio pathological mechanisms. Most of the patients who develop

respiratory failure require treatment in the intensive care unit (2,3).

Units. where unstable patients who require intensive treatment and observation are accepted. are intensive care units. Most of the patients followed in the intensive care unit (ICU) have respiratory failure due to infection or inflammation such as pneumonia, acute respiratory

distress syndrome (ARDS) (2,3). In most cases of respiratory failure, hypercapnia, hypoxia, apnea, and related complications are observed. Therefore, respiratory support treatments are needed and used (4).

Data on the incidence of respiratory failure in the ICU are insufficient. According to the results of studies conducted in Europe, the frequency of patients treated with mechanical ventilation for more than 24 hours in ICUs is estimated to be 77.6-88.6/100000 years (5). In the USA, the prevalence of respiratory failure was found to be 137/100000 years, and its incidence was 360000/year (6).

Respiratory failures can be classified under three headings according to their clinic, onset time, and pathophysiology. According to his clinic; It can be subdivided into hypoxemic, hypercapnic, and combined respiratory failure. According to the onset time, acute respiratory failure can be grouped as acute, chronic, and chronic. According to its pathophysiology, four types of respiratory failure can be defined, Type 1; hypoxemic respiratory failure, Type 2; hypercapnic respiratory failure, Type 3; perioperative respiratory failure, and Type 4 is defined as respiratory failure due to hypoperfusion developing in patients in shock (1).

Arterial blood gas is one of the important laboratory methods that provide reliable information about the metabolic and respiratory status of patients. Arterial blood gas analysis is performed using blood gas measurement devices. With these devices, pH, partial carbon dioxide pressure, partial oxygen pressure is measured using electrodes; bicarbonate, oxygen saturation, and base deficit are calculated. Metabolic acidosis is one of the acid-base disorders that we see frequently in intensive care units (7).

Patients with severe sepsis or septic shock admitted to the intensive care unit present a wide spectrum of acid-base disturbances, the most common being metabolic acidosis. The presence of metabolic acidosis is associated with higher morbidity and mortality in the intensive care unit (8).

In general, acid-base disturbances are analyzed according to conventional methods that include the determination of base excess, bicarbonate concentration in plasma, and anion gap. When an only simple acid-base disturbance is present. the conventional method is sufficient. However, it does not give detailed information about the source of the problem. The calculation of base deficiency is a calculated figure that assumes normal plasma protein and electrolyte contents and ignores the role of non-bicarbonate buffers in the blood. Therefore, the conventional interpretation can be clinically misleading, as electrolyte or protein abnormalities are almost always present in ICU patients. An alternative to

this traditional model is the mathematical model based on physicochemical principles described by Stewart and modified by Figge et al. (8,9).

This study aims to create a different perspective in the follow-up and treatment of patients by evaluating acid-base disorders with Stewart analysis in the patient population with respiratory failure hospitalized in the intensive care unit.

MATERIAL AND METHOD

Ethical approved this study by Ethics Committee of Atatürk Sanatoryum Training and Research Hospital (Date: 13.09.2019, Decision No: 1953). All procedures were performed adhered to the ethical rules and principles of the Helsinki Declaration. This study was conducted by evaluating the arterial blood gases of 106 patients diagnosed with type 1-2 respiratory failure between January 2019 and August 2019. Serum BUN, creatinine, glucose, sodium, chlorine, potassium, magnesium, albumin values of the patients as well as pH, pCO₂, pO₂, HCO₃, base deficit, and lactate levels studied in arterial blood samples were evaluated. ABG evaluation was made using the Stewart method on the website www.acidbase.org, and an interface that was standardized and turned into a 'calculator' was used.

In our study, patients' arterial blood gases, lactate, BUN, creatinine, glucose, sodium, chlorine, potassium, magnesium, albumin values, additional diseases, length of stay in the intensive care unit, treatment costs were recorded retrospectively, and those who were discharged from the intensive care unit and those who died while in the intensive care unit, was also recorded. The relationship between arterial blood gases evaluated by the Stewart method and other parameters was evaluated.

Inclusion criteria in the study;

- Intensive care patients diagnosed with type 1-2 respiratory failure
- Patients with respiratory failure hospitalized directly from the emergency department
- Patients who were transferred to chest intensive care from any service or a higher-level intensive care unit

Exclusion criteria from the study;

- Patients with missing data
- Intensive care patients other than respiratory failure

Statistical Analysis

In our study, the data were evaluated in the IBM SPSS Statistics 22.0 statistical package program. As descriptive statistics. the number of units (n), percent (%), mean±standard deviation ($\bar{x}\pm ss$), and median values are

given. The normal distribution of the data of numerical variables was evaluated with Shapiro Wilk. normality test. and Q-Q charts. Mann-Whitney U and Kruskal Wallis analyses were used to compare the groups. Spearman correlation analysis was used to determine the relationship between two numerical values. A $p < 0.05$ value will be considered significant in statistical analysis.

RESULTS

The patients included in the study, 63 (59.4%) were male and 43 (40.6%) were female. The mean age of the patients was 70.36 ± 10.5 (minimum 49- maximum 90) years. The mean age of women was 73.60 ± 9.94 years, and the mean age of men was 68.14 ± 10.37 years; the mean age of women was statistically significant and higher than the mean age of men (Table 1).

Table 1. Age distribution by gender

	Mean±Standard Deviation	Median	z	p
Female	73.60±9.94	74.00	-2.476	0.013
Male	68.14±10.37	67.00		
Age (total)	70.36±10.5	69.50		

The mean values of arterial blood gases (pH, pCO₂, HCO₃, BE, pO₂, Lactate), BUN, creatinine, glucose, albumin, sodium, potassium, magnesium, chlorine of the patients is given in Table 2.

Table 2. Mean blood gas and blood biochemistry values of the patients

	Mean±Standard Deviation	Median	Min.-Max.
pH	7.40±0.61	7.40	(7.29-7.57)
pCO ₂	57.2±14.62	56.10	s(29.4-94)
HCO ₃	33.95±7.59	33.65	(15.9-60.7)
BE	7.78±6.23	7.20	(-5.4-30.7)
pO ₂	61.99±24.72	57.15	(25.7-192.8)
Lactate	1.2±0.66	1.01	(0.02-3.56)
BUN	24.68±15.49	20.50	(8-86)
Creatinin	0.9±0.44	.80	(0.43-3.78)
Glucose	132.76±67.08	113.00	(54-446)
Albumin	29.86±4.68	29.60	(18.3-40.8)
Sodium	139.91±4.36	141.00	(123-152)
Potassium	4.23±0.67	4.26	(2.7-6.19)
Magnesium	1.92±0.31	1.90	(1.2-2.6)
Chlorine	96.03±5.73	97.00	(80-111)

Min: minimum. Max: maximum BUN: Blood Urea Nitrogen

It was determined that 74 (69.8%) of the patients were discharged and 32 (30.2%) died. A statistically significant difference was found between BUN values according to the clinical outcome (death-discharge). The BUN value of the group that died was found to be higher than the group that survived (Table 3).

Table 3. Mean blood gas values according to the clinical outcome of the patients

	Discharged		Death		P
	Mean±std deviation	Median	Mean±std deviation	Median	
pH	7.4±0.1	7.4	250.9±1.333.8	7.4	0.647
pCO ₂	57.2±15.8	55.1	57.1±11.7	57.1	0.915
HCO ₃	34±8.1	33.7	33.9±6.4	33.9	0.736
BE	7.6±6.6	7.2	8.3±5.4	8.0	0.384
pO ₂	62.1±25.1	57.4	61.7±24.2	53.5	0.836
Lactate	1.2±0.7	1.0	1.1±0.5	1.1	0.818
BUN	22.9±14.1	19.0	28.8±17.8	24.5	0.035
Creatinine	0.9±0.5	0.8	0.9±0.4	0.9	0.318
Glucose	127.3±50.2	114.5	145.4±95.2	105.5	0.853
Albumin	30.2±4.5	30.1	29±5.1	29.1	0.224
Sodium	139.7±4.3	141.0	140.4±4.6	141.0	0.174
Potassium	4.2±0.6	4.2	4.3±0.8	4.3	0.445
Magnesium	1.9±0.3	1.9	1.9±0.3	1.9	0.718
Chlorine	95.9±6	97.0	96.3±5.1	97.0	0.548

The comorbidities of the patients were examined. it was determined that 96 (89.7%) of them were COPD, 48 (44.8%) were hypertension, 28 (26.1%) were heart failure. Other additional diseases are given in Table 4.

Table 4. Additional Diseases of the Patients

	n(%)
Chronic obstructive pulmonary disease	96 (89.7)
Hypertension	48 (44.8)
Heart failure	28 (26.1)
Diabetes mellitus	27 (25.2)
Coronary artery disease	16 (14.9)
Pulmonary thromboembolism	9 (8.4)
Pneumonia	9 (8.4)
Chronic kidney disease	5(4.6)

A moderate and negative correlation was found between pH value and pCO₂ and potassium ($p = -0.557$ $p = -0.409$, respectively). A weak and negative correlation was found between pCO₂ and lactate, creatinine and chlorine ($p = -0.391$ $p = -0.201$ $p = -0.328$, respectively). There was a weak positive correlation between pCO₂ and sodium ($p = 0.336$). and a good and positive correlation between pCO₂ and bicarbonate ($p = 0.773$). A weak and negative correlation was found between pO₂, albumin, and potassium ($p = 0.277$. $p = 0.279$. respectively). There was a weak and negative correlation between HCO₃, lactate, and creatinine ($p = -0.394$. $p = -0.270$, respectively), and a moderate negative correlation between HCO₃ and chlorine ($p = -0.565$). There was a weak and negative correlation between albumin and BUN ($p = -0.232$), and a weak and positive correlation between albumin and potassium ($p = 0.293$) (Table 5).

Table 5. Correlations of pH, pCO₂, pO₂, HCO₃, Lactate, Albumin values

	Ph	pCO ₂	pO ₂	HCO ₃	Lactate	Albumin
pH	-					
pCO ₂	-0.557**	-				
pO ₂	-0.081	-0.031	-			
HCO ₃	-0.173	0.773**	-0.150	-		
Lactate	0.209*	-0.391**	0.057	-0.394**	-	
Albumin	-0.110	-0.184	0.277**	-0.212*	0.187	-
BUN	-0.102	-0.005	-0.068	-0.116	0.054	-0.232*
Creatinine	0.013	-0.201*	-0.052	-0.270**	-0.016	-0.014
Na	-0.090	0.336**	0.037	0.342**	-0.179	-0.123
K	-0.409**	0.095	0.279**	-0.184	0.139	0.293**
Mg	-0.012	0.020	0.165	-0.041	0.031	-0.005
Cl	-0.083	-0.328**	0.020	-0.565**	0.089	-0.022

*p<0.005, **p<0.01

A statistically significant difference was found between the development of acidosis in the patients and the treatment costs (p=0.038). The cost of treatment of patients who did not develop acidosis was found to be statistically significant and lower than patients who developed metabolic and respiratory acidosis. A statistically significant correlation was found between hypoalbuminemia in patients and treatment costs (p=0.005). The patients with hypoalbuminemia were evaluated as mild-moderate-severe, a statistically significant relationship was found between the severity of hypoalbuminemia and the cost of treatment, In the post-hoc test performed to find the group that made the significant difference, the patient group with severe hypoalbuminemia (serum albumin value < 15 g/L) was found to have higher treatment costs than the other groups (p=0.005). Treatment costs increase significantly as albumin levels decrease in patients with hypoalbuminemia (Table 6).

The mean length of stay of the patients in the intensive care unit was determined as 17.48±10.58 (minimum 6-maximum 68) days.

Table 6. Comparison of metabolic status and treatment costs

	Mean	Median	p*	p**
Acidosis	None	10119.08±8.587.21	5.428.00	0.691 0.038
	Metabolic	15089.47±9.515.71	12.760.00	
	Respiratory	15840.21±9.146.63	13.196.00	
Alkalosis	None	13874.2±15.015.89	8.216.00	0.443 0.459
	Metabolic	15050.12±9.198.01	12.641.50	
	Respiratory	15548.4±2.469.89	15.668.00	
Albumin	None	18.985.00±11.646.83	15.346.00	0.005 <0.001
	mild hypoalbuminemia	13.034.43±8.571.62	10.424.00	
	moderate hypoalbuminemia	16.530.50±8.003.92	15.120.00	
	severe hypoalbuminemia	37.912.00±2.081.72	37.912.00	

*Mann Whitney U. **Kruskall Wallis

A statistically significant difference was found between the development of acidosis and the length of stay in the intensive care unit. A statistically significant difference was found between the development of hypoalbuminemia and the length of stay in the intensive care unit (p=0.009). The patients who developed hypoalbuminemia were evaluated within themselves, a statistically significant relationship was found between the severity of hypoalbuminemia and the length of stay in the intensive care unit. In the post-hoc test performed to find the group that made the significant difference, it was determined that the patient group with severe hypoalbuminemia (serum albumin value < 15 g/L) had longer intensive care unit stays than the other groups (p=0.009) (Table 7).

Table 7. Comparison of metabolic status and length of stay in intensive care unit

	Mean	Median	p*	p**
Acidosis	None	12.08±11841	10.00	0.357 0.040
	Metabolic	16.59±15980	14.00	
	Respiratory	18.61±36434	15.00	
Alkalosis	None	16.6±12359	15.00	0.962 0.919
	Metabolic	17.65±30956	15.00	
	Respiratory	15.2±30376	16.00	
Albumin	None	17.43±9.96	16.00	0.009 <0.001
	mild hypoalbuminemia	15.44±8.9	12.00	
	moderate hypoalbuminemia	19.21±9.47	19.50	
	severe hypoalbuminemia	52.5±21.92	52.50	

*Mann Whitney U. **Kruskall Wallis

No statistically significant correlation was found between the metabolic status of the patients (acidosis, alkalosis, hypoalbuminemia) and their clinical outcomes (Table 8).

Table 8. Comparison of metabolic status and clinical outcome

	Discharged n(%)	Deathn(%)	p	
Acidosis	None	9(69.23)	4(30.77)	0.843*
	Metabolic	13(76.47)	4(23.53)	
	Respiratory	52(68.42)	24(31.58)	
Alkalosis	None	4(80)	1(20)	0.237**
	Metabolic	65(67.71)	31(32.29)	
	Respiratory	5(100)	0(0)	
Albumin	None	5(71.43)	2(28.57)	0.458**
	mild hypoalbuminemia	47(74.6)	16(25.4)	
	moderate hypoalbuminemia	21(61.76)	13(38.24)	
	severe hypoalbuminemia	1(50)	1(50)	

*Chi-square test. **Fisher's Exact Test

DISCUSSION

A change in patients' pH indicates that there must be a change in one of the independent variables (strong anion and cation) and cannot simply be explained by the influx of hydrogen ions or bicarbonate into body fluids (10). An

analysis of the complex acid/base disturbances commonly seen in critically ill patients can be performed using the approach that includes weak acids (10). The moderate-negative correlation between pH value and pCO₂ and potassium in our study supports this approach.

In a study conducted in respiratory intensive care patients at Trakya University, it was reported that 43% of the patients had COPD, 36% had severe pneumonia/sepsis + multi-organ failure, 7% had a massive pulmonary embolism, and 5% had acute respiratory distress syndrome. In this study, mortality rates were reported as 32% (11). In Çanakkale in 2014, 19% of the primary care intensive care patients had COPD, 8% had diabetes mellitus, 23% had hypertension, 9% had coronary artery disease, 14% had congestive heart failure, and 9% had chronic kidney disease co-diagnoses (12). Between 2014 and 2016, the diagnosis of hypertension (61%), coronary artery disease (27%), diabetes mellitus (27%), Alzheimer's 25%, 23% congestive heart failure, 20% previous CVA in the intensive care unit patients of sepsis patients in Ankara was available (13). In our study, 89% of the patients had COPD, 45% had hypertension, 26% had heart failure, 25% had diabetes mellitus, 15% had coronary artery disease, and 8% had pulmonary thromboembolism. Although the general rates vary, comorbid conditions are similar in the admission of patients to the intensive care unit or among the patients admitted to the intensive care unit.

In the study of Uçgun et al. (14), it was emphasized that there was no significant difference between the mean pH values of patients who survived in the intensive care unit (pH=7.2±0.6) and the pH values of patients who lost their lives. In another study, it was reported that there was no statistically significant relationship between pH values of patients who died and survived (11). In our study, the mean pH value was found to be 7.4±0.1 in patients who survived and died. The difference between the pH values found in the study of Uçgun et al. and our study may be due to the effect of differences in patient profiles on metabolic conditions (such as cancer cases). The fact that the pH values of the deceased and surviving patients were the same suggested that metabolic disorders may not be the main cause. even if metabolic processes were effective in the death of the patients.

In a study conducted in intensive care patients in China (n: 1003), it was emphasized that the albumin value was statistically higher in surviving intensive care patients (15). In a study examining the relationship between albumin values and mortality in intensive care trauma patients (n:200), a significant relationship was found between albumin and mortality. It was reported that the albumin values of the patients who died were found to be lower (16). In a study conducted in the respiratory intensive care unit of Trakya University, it was reported

that albumin values were found to be lower in deceased patients than in surviving patients (11). In our study, the mean albumin value of the surviving patients was 30.2±4.5, while the mean albumin value of the deceased patients was 29±5.1. No statistically significant difference was found between the two groups. While albumin values were associated with mortality in trauma patients, it was thought not to be associated with mortality in respiratory intensive care patients. In our study, no relationship was found between albumin value and clinical outcome. In our study, a statistically significant relationship was found between albumin and the length of stay in the intensive care unit. Patients with severe hypoalbuminemia were found to have longer intensive care unit stays. Due to albumin being a negative acute phase reactant, as the amount of inflammation in the patient increases, the albumin value may decrease and the length of stay in the intensive care unit may be prolonged. Or, depending on the severity of the metabolic events in the patient, the albumin value may have decreased and these metabolic events may have prolonged the intensive care unit stay of the patient.

In a study examining the relationship between lactate levels and mortality in 200 trauma patients in the intensive care unit, a significant relationship was found between lactate and mortality. It was reported that the lactate values of patients who died were found to be higher (14). In our study, no significant difference was found between the lactate values of the patients who survived and those who died. The reason why lactate values, which were not included in the Hasselbach calculation method and were included in the SID calculation, were not found to be statistically significant in our study may be due to the fact that patients with metabolic problems. such as kidney failure and severe traumas, were not included in our study. Conditions and diseases in which lactate is elevated are well known. In the foreground, sepsis is one of the most important causes of lactic acidosis. Apart from this, inotropic infusion, metformin toxication, diseases causing peripheral circulatory disorders, etc. conditions such as can increase lactate. Since all of these conditions require 3rd level intensive care treatment, it may be natural that lactate does not make a significant difference in terms of mortality in the 2nd level respiratory intensive care unit where the study was conducted.

In a study by Uçgun et al. (14) it was stated that there was no significant difference between the mean PO₂ and PCO₂ values of surviving patients when compared to the values of deceased patients. In a study conducted in the respiratory intensive care unit, it was reported that no significant correlation was found between PaO₂ and PaCO₂ values in patients who died and survived (11). Similar to the literature, in our study, no significant

difference was found between the mean PaCO₂ and PaO₂ values of the discharged and deceased patients.

In a study by Uçgun et al. (14), it was stated that there was no significant difference between the mean leukocyte, hematocrit, thrombocyte, glucose, Na, K, albumin values of the patients who survived in the intensive care unit and the laboratory values of the patients who died. In the same study, it was emphasized that the mean pH and HCO₃ values of surviving patients were statistically lower than those of deceased patients. It was emphasized that the mean BUN and LDH values of the surviving patients were statistically lower than those of the deceased patients. In our study, no difference was found between discharged and deceased patients in the HCO₃, BE, lactate, creatinine, glucose, albumin, sodium, potassium, magnesium, chlorine values of the patients. BUN values of the patients who died were found to be significantly higher than those of the patients who survived. BUN values were thought to be more successful than other laboratory values in predicting the probability of mortality in patients.

In our study, the average length of stay in the intensive care unit was found to be 17 days.

In the study of Bıyıklı et al. (13), the 30-day mortality rate of intensive care patients was reported as 45%. In this study, it was reported that age, lactate, and SpO₂ did not affect 30-day mortality.

Mortality rates were found to be 40% in male patients and 45% in females in patients with acute respiratory failure followed in the intensive care unit (n:508) in Berlin (17). In our study, mortality rates in patients with respiratory failure followed in the intensive care unit were found to be 35% in male patients and 25% in female patients. These data show that the mortality rate is close to each other in both genders.

In a study conducted in Shanghai, the 30-day mortality of intensive care patients diagnosed with sepsis-associated pneumonia was reported as 33%(18). The rate of patients followed up with pneumonia in our study was 9% of the patients included in the study, and all of the patients were discharged, no mortality was detected. Due to the insufficient number of patients with pneumonia, it may be misleading to state the mortality rate in patients with pneumonia.

It was emphasized that the mortality rate of patients with lung injury was found to be 36% in patients with acute respiratory failure in Berlin (17). All patients participating in our study had acute or chronic lung disease and the mortality rate was found to be 30%.

CONCLUSION

While a significant relationship was found between albumin levels, which has an important place in the Stewart method, and the length of stay in the intensive care unit, no relationship was found between albumin or lactate levels and mortality. However, increased BUN values were associated with mortality. As the albumin value decreases in patients with hypoalbuminemia, treatment costs increase. There is a need for larger multicenter studies with a larger sample group that will evaluate metabolic status with the Stewart method and investigate its relationship with mortality.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was initiated with the approval of the Atatürk Sanatoryum Training and Research Hospital Ethics Committee (Date:13.09.2019, Decision No: 1953)

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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REFERENCES

1. Yarkin T. Solunum Yetmezliği: Fizyopatoloji ve Klinik Yaklaşım. *Toraks Derg* 2000; 2: 76-84.
2. Frutos-Vivar F, Nin N, Esteban A. Epidemiology of acute lung injury and acute respiratory distress syndrome. *Current Opinion in Critical Care* 2004; 10: 1-6.
3. Esteban A, Anzueto A, Frutos F, et al. Mechanical Ventilation International Study Group: Characteristics and outcomes in adult patients receiving mechanical ventilation: a 28-day international study. *JAMA* 2002; 287: 345-55.
4. Donlan M, Fontela PS, Puligandla PS. Use of continuous positive airway pressure (CPAP) in acute viral bronchiolitis: a systematic review. *Pediatr Pulmonol* 2011; 46: 736-46.
5. Lewandowski K. Contributions to the epidemiology of acute respiratory failure. *Critical Care* 2003; 7: 1-4.
6. Behrendt CE. Acute respiratory failure in the United States: incidence and 31-day survival. *Chest*. 2000; 118: 1100-5.
7. Aygencel G. Arter kan gazlarının yorumlanması. *Türk Kardiyol Dern Arş* 2014; 42: 195.
8. Szrama J, Smuszkiewicz P. An acid-base disorders analysis with the use of the Stewart approach in patients with sepsis treated in an intensive care unit. *Anaesthesiology Intensive Therapy* 2016; 48: 180-4.

9. Fencel V, Jabor A, Kazda A, Figge J. Diagnosis of metabolic acid-base disturbances in critically ill patients. *Am J Respir Crit Care Med* 2000; 162: 2246-51.
10. Nemec M. Interpretation of arterial blood gas analysis. *Praxis* 2019; 108: 269-77.
11. Altıay G, Tabakoğlu E, Özdemir L, et al. Mortality rates and related factors in respiratory intensive care unit patients. *Türk Thorax J* 2007; 8: 79-84.
12. Doğu T, Karakuzu Z, Katı ŞD, et al. Birinci basamak yoğun bakım ünitesi hastalarının prognozu. *Uluslararası Klinik Araştırmalar Derg* 2014; 2: 143-8.
13. Bıyıklı E. 65 yaş üstü sepsis ve septik şok hastalarında acil serviste ilk bakılan platelet lenfosit oranı ve laktat düzeyinin mortalite üzerine etkisi. Ankara. 2017.
14. Uçgun İ, Metintaş M, Moral M, Alataş F, Bektaş Y, Yıldırım H. To identify mortality rate and high risk patients in non-malignant respiratory intensive care unit patients. [Article in Turkish]. *Toraks Derg* 2003; 4: 151-60.
15. Yap FHY, Joynt GM, Buckley TA, Wong ELY. Association of serum albumin concentration and mortality risk in critically ill patients. *Anaesth Intens Care* 2002; 30: 202-7.
16. Yılmaz E, Bor C, Uyar M, Demirağ K, Çankayalı İ. Travma hastalarının yoğun bakıma kabulündeki laktat, albumin, C-reaktif protein, PaO₂/FiO₂ ve glukoz düzeylerinin mortaliteye etkisi. *J Turk Soc Intens Care/Türk Yoğun Bak Derg* 2014; 12: 3.
17. Lewandowski K, Metz J, Deutschmann C, et al. Incidence, severity, and mortality of acute respiratory failure in Berlin, Germany. *Am J Respir Crit Care Med* 1995; 151: 1121-5.
18. Chen YX, Wang JY, Guo SB. Use of CRB-65 and quick sepsis-related organ failure assessment to predict site of care and mortality in pneumonia patients in the emergency department: a retrospective study. *Crit Care* 2016; 20: 1-10.