RESEARCH

Correlation Between $ScVO_2$, PCO_2 Gap (Artery-Vein PCO_2 Gradient) and Respiratory Quotient with Cardiac Output in Septic Shock Patients

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ABSTRACT

Background: Septic shock is a life-threatening condition characterized by cardiovascular dysfunction, which can cause multi-organ failure and high mortality rates. Central venous oxygen saturation ($ScVO_2$), PCO_2 Gap, and respiratory quotient are potential markers of tissue perfusion and oxygenation, while cardiac output is an important indicator of cardiovascular function. These findings may help guide clinical management and prognosis assessment in patients with septic shock.

Objective: To determine the correlation between $ScVO_2$, PCO_2 Gap (arterial-venous PCO_2 gradient), and respiratory quotient on cardiac output in septic shock patients.

Methods: This observational study was conducted on 30 septic shock patients treated in the intensive care unit (ICU). Blood samples were taken via central venous catheter (CVC) and arterial line to check central venous saturation ($ScVO_2$), PCO_2 Gap, and calculate the respiratory quotient. Then cardiac output is measured using echohemodynamics.

Results: Based on analysis using the Spearman Rho test, the results show that the correlation between $ScVO_2$ and cardiac output has a correlation value of -0.367 ($p = 0.046$). The correlation between PCO_2 Gap and cardiac output has a correlation value of -0.520 ($p=0.003$). The correlation between respiratory quotient and cardiac output in this study had a correlation value of -0.833 ($p < 0.001$).

Conclusion: There is a correlation between $ScVO_2$, PCO_2 Gap (arterial-venous PCO_2 gradient), and respiratory quotient on cardiac output in septic shock patients.

Keywords: cardiac output; PCO_2 Gap; respiratory quotient; $ScVO_2$; septic shock

INTRODUCTION

Sepsis is a systemic response to an infection. Sepsis is a syndrome that occurs due to impaired circulation and perfusion. Sepsis management is based on three components, namely infection control, hemodynamic stabilization, and modulation of the sepsis response.[1] Hemodynamic optimization remains the cornerstone of resuscitation in sepsis management. Delay or inadequate management causes a person to fall into septic shock.[2] According to the Surviving Sepsis Campaign (SSC) 2021, septic shock is a part of sepsis where severe circulatory, cellular, and metabolic abnormalities substantially increase mortality and is operationally defined as persistent hypotension requiring vasopressor therapy to maintain mean arterial pressure or average arterial blood pressure above 65 mmHg and an increase in lactate levels> 2 mmol/L despite adequate fluid resuscitation.[3,4]

Sepsis occurs in about 2% of all hospitalizations and 6-30% of all patients treated in the intensive care unit (ICU). Sepsis and septic shock are major causes of morbidity and mortality in the ICU (21% and 28%, respectively).[5] An estimated 48.9 million cases of sepsis and 11.0 million sepsis-related deaths occurred in 2017.[6] The global burden of sepsis is greater than previously estimated and requires urgent attention. Therefore, early recognition and timely treatment are critical to the outcome of sepsis and septic shock.[7] The hemodynamic support algorithm for patients with septic shock focuses on the following initial responses: central venous pressure 8-12 mmHg, urine output above 0.5 ml/kgBW, and central venous oxygen saturation ($ScVO_2$) 70% or more.[8]

During infection, the interaction between microbes and the immune system results in an inflammatory cascade involving the production of cytokines and other mediators, which in turn trigger a systemic response. The resulting effects include vasodilation, increased vascular permeability, myocardial depression, and disruption of the coagulation cascade, resulting in a global imbalance in systemic oxygen supply and demand and multiple organ dysfunction.[9]

Cardiovascular dysfunction in sepsis is associated with a significantly higher risk of mortality of 70-90% compared with patients with sepsis without cardiovascular dysfunction.[10] The manifestations of cardiovascular dysfunction in sepsis are complex and differ between hyperdynamic and hypodynamic sepsis. During hyperdynamic septic shock, decreased peripheral resistance and stimulation of the adrenergic system cause tachycardia, increased cardiac output, and increased surface temperature due to decreased afterload. Hypodynamic sepsis causes decreased diastolic filling pressure, bradycardia, decreased cardiac output, and ischemic organ damage.[11] Therefore, in an effort to reduce mortality in sepsis patients, identification of cardiovascular dysfunction is important. Decreased cardiac output can be an indicator of the hypodynamic phase of sepsis before the patient falls into septic shock, so early identification of decreased cardiac output can be part of early goal-directed therapy (EGDT) in sepsis management. Unfortunately, echocardiography is not available in all hospitals, especially in ICU, so other markers are needed to identify decreased cardiac output in sepsis patients.

Central venous blood saturation ($ScVO_2$) is one of the parameters that determines the global adequacy of oxygen transport and oxygen requirements. In septic shock patients, $ScVO_2$ is often found to decrease due to global organ perfusion disorders due to decreased cardiac output caused by shock.[12]

A decreased $ScVO_2$ generally indicates that oxygen delivery is inadequate relative to tissue oxygen demand, which may occur due to reduced cardiac output, hypovolemia, anemia, or hypoxemia. In such conditions, tissues compensate by increasing oxygen extraction, resulting in lower venous oxygen saturation, a situation commonly seen during the early phase of septic shock when hypoperfusion is prominent. However, patients with septic shock may also exhibit normal or elevated $ScVO_2$ values, which do not necessarily reflect adequate tissue perfusion. Sepsis-related microcirculatory dysfunction, including endothelial injury, capillary obstruction, and heterogeneous blood flow distribution, can impair oxygen diffusion to tissues despite adequate systemic oxygen delivery. Additionally, inflammatory responses in sepsis may cause mitochondrial dysfunction or cytopathic hypoxia, where cells are unable to utilize oxygen effectively, leading to reduced oxygen extraction and higher venous oxygen saturation. The hyperdynamic circulatory state often observed in septic shock, characterized by increased cardiac output and reduced systemic vascular resistance, may further limit oxygen extraction by shortening capillary transit time. Therefore, both low and high $ScVO_2$ values in septic shock must be interpreted cautiously, as each may indicate impaired tissue oxygenation depending on the underlying pathophysiological mechanisms.[31,32]

PCO_2 Gap is the difference in partial pressure of CO2 between the veins and arteries. PCO_2 Gap can reflect Venous Return from the Capillary bed and the adequacy of microcirculation. Under normal conditions (normal cardiac output and capillary bed) all CO_2 production is cleared quickly and the PCO_2 Gap is minimal. PCO_2 Gap > 6 mmHg is a cut-off value that describes insufficient cardiac output, in this case the choice of therapy is to increase cardiac output with the aim of normalizing the PCO_2 Gap.[13]

In septic shock, persistent elevation of the PCO_2 Gap may indicate ongoing tissue hypoperfusion, even when systemic hemodynamic parameters appear acceptable. Carbon dioxide (CO_2) is highly soluble and diffuses more readily than oxygen; therefore, impaired microcirculatory blood flow leads to CO_2 accumulation in the venous compartment. As a result, the PCO_2 Gap may serve as a sensitive marker of inadequate tissue perfusion. Persistent increase in PCO_2 shows a correlation with poor prognosis.[13,33]

According to the Fick Principle, cardiac output can use oxygen as part of the calculation. Total oxygen use (VO_2) is equal to cardiac output and the difference in arterial-venous oxygen content. This principle can be extended to measure the uptake or release of any substance, for example CO_2, and can be used for the body as a whole or for certain tissues or organs.[14] Until now, there has been no research that assesses the correlation between $ScVO_2$, PCO_2 Gap and respiratory quotient to cardiac output in patients with septic shock. This study was conducted to determine the correlation between $ScVO_2$, PCO_2 Gap and respiratory quotient to cardiac output in patients with septic shock.

METHOD

The design of this study will be cross-sectional with a numerical correlative analytical observational method. Primary data were collected from laboratory tests and echocardiography. This research method is used to determine the correlation between $ScVO_2$, PCO_2 Gap and respiratory quotient with cardiac output in patients with septic shock. The sample of this study was patients who had been diagnosed with septic shock with a sequential organ failure assessment (SOFA) score >= 2 at Dr. Kariadi Hospital.

Inclusion criteria patients who have been diagnosed with septic shock who are treated at Dr. Kariadi Hospital and confirmed with a SOFA score >= 2. Exclusion criteria: (1) patients aged <18 years during the study period, (2) patients have a history of heart disease before or during the study, (3) patients who have been declared brain dead 4. Patients who refuse to have a subclavian/jugular central venous catheter (CVC) installed.

Sampling was conducted at Dr. Kariadi Hospital during the study period. Patients diagnosed with septic shock were then selected as research subjects who were selected by consecutive sampling who met the inclusion and exclusion criteria (n = 30).

Data collection on research participants who had met the inclusion and exclusion criteria. Subclavian/jugular CVC installation, arterial line installation, blood sampling (for routine blood tests), arterial and venous blood gas analysis (BGA) and echo hemodynamic examination were performed to measure stroke volume (SV) and cardiac output. Non-invasive ultrasound was carried out alternately with 2 operators to reduce bias. After the results of routine blood tests and BGA came out, PCO_2 Gap and respiratory quotient were calculated.

The data that has been collected will be processed using statistical software (IBM® SPSS® version 27) to determine the correlation between $ScVO_2$, PCO_2 Gap and respiratory quotient with CO. After the data is obtained, a normality test and a variance test will be carried out on the data using the pearson correlation analysis method (if the distribution is normal) and Spearman (if the distribution is not normal). A p-value < 0.05 was considered statistically significant. Data analysis was performed using the unpaired numerical correlative analytical method.

RESULTS

The data used in this study are primary data, namely patients treated at Dr. Kariadi Hospital and diagnosed with septic shock with a sofa score ≥ 2 and patients using vasopressors to maintain mean arterial pressure (MAP) ≥ 65. The subjects in this study were 30 patients with 14 male patients and 16 female patients. The data taken included body weight, height, body surface area, systolic blood pressure, diastolic blood pressure, hemoglobin, SaO_2, $ScVO_2$, PaO_2, $PaCO_2$, $PvCO_2$, PCO_2 Gap, respiratory quotient and cardiac output.

Based on Table 1, mong the 30 patients enrolled in the study, there were 14 male patients or 46.7% and 16 female patients or 53.3% with an average age of 48.37 years with the youngest age being 21 years and the oldest age being 68 years. Patients had an average body weight of 62.97 kg. MAP in this research sample had an average of 66.01 mmHg with the lowest MAP being 47 mmHg and the highest being 95.33 mmHg. The average

hemoglobin in this study was 10.25. The average SaO_2, $ScVO_2$, PaO_2, PvO_2, $PaCO_2$, $PvCO_2$, PCO_2 Gap, respiratory quotient, cardiac output and cardiac index in these patients were 96.22%; 84.28%; 143.75 mmHg; 33.16 mmHg; 37.01 mmHg;40.29 mmHg; 3.27 mmHg; 0.958 ; 4.448 L/min,2.714.

In this study, data on body weight, BSA, MAP, systolic, diastolic, hemoglobin, PaO2, PvCO2, cardiac output, and cardiac Index were normally distributed. While data on age, gender, height, SaO2, ScVO2, PaCO2, PCO2 Gap, and respiratory quotientwere not normally distributed. Inhomogeneous data results may be caused by other confounding factors that may influence the results of this study.

Based on the analysis with the Spearman Rho test (Table 2), the results show that the correlation between $ScVO_2$ and cardiac output has a correlation value of 0.376 with a p value of 0.041, this means that there is a sufficient and significant correlation with the opposite direction of the correlation where the greater the $ScVO_2$, the smaller the cardiac output will be and vice versa.

For the correlation between PCO_2 Gap and cardiac output, there is a correlation value of -0.627 with a p value <0.001. This means that there is a strong and significant correlation with the opposite direction of the correlation, where the greater the PCO_2 Gap, the smaller the cardiac output will be and vice versa (Figure 2).

The correlation between respiratory quotient and cardiac output in this study has a correlation value of -0.831 with a p value <0.001 (Table 2), this means that there is a strong and significant correlation between respiratory quotient and cardiac output with the opposite direction of the correlation where the greater the respiratory quotient, the smaller the cardiac output and vice versa. So it can be concluded that there is a correlation between $ScVO_2$, PCO_2 Gap and respiratory quotient to cardiac output in septic shock patients (Figure 3).

Table 1. Characteristics of research samples

Variable	N (%)	Mean ± SD	Median (Min s/d Max)	p€
Age (years)		48.37 ±16.79	56.5 (21 s/d 68)	< 0.001
Gender				
Male Female	14 (46.7%) 16 (53.3%)			
Weight (kg)		62.97 ±13.48	65 (35 s/d 85)	0.197*
Heights (cm)		159.3 ±8.163	160 (135 s/d 170)	0.009
BSA		1.661 ±0.205	1.70 (1.22 s/d 2.0)	0.312*
MAP		66.01 ± 9.23	67.16 (47 s/d 95.33)	0.088*
Systole		88.23 ± 9.56	90.5 (68 s/d 110)	0.070*
Diastole		54.9 ± 10.04	54.5 (36 s/d 88)	0.062*
Hemoglobin		10.25 ± 1.53	10.1 (6.3 s/d 13.4)	0.666*
SaO_2		96.22 ± 8.52	98.5 (58.1 s/d 99.7)	<0.001
$ScVO_2$		84.28 ± 12.15	87 (42.1 s/d 97.2)	<0.001
PaO_2		143.75 ± 58.5	142.75 (28.7 s/d 313.1)	0.481*
$PaCO_2$		37.01 ± 14.24	35.25 (16.4 s/d 92.5)	<0.001
$PvCO_2$		40.29 ± 12.07	37.5 (19.1 s/d 68.6)	0.276*
PvO2		33.16 ± 10.74	35 (7.0 s/d 50.0)	0.297*
PCO_2Gap		3.273 ± 9.82	4.35 (-23.9 s/d 22.9)	0.001
Respiratory quotient		0.9584 ± 8.28	1,61 (-20.28 s/d 25.05)	0.001
Cardiac output		4.448 ± 1.21	4.4 (2.1 s/d 7.2)	0.908*
Cardiac index		2.714 ± 0.76	2.82 (1.21 s/d 4.53)	0.487*

*Significant (p >0,005); €Saphiro-Wilk normality test

Table 2. Spearman Test $ScVO_2$, PCO_2 Gap, respiratory quotienton cardiac output

Variabel	r	p
ScVO2	-0.376	0.041
PCO_2 Gap	-0.627	<.001
Respiratory quotient	-0.831	<0.001

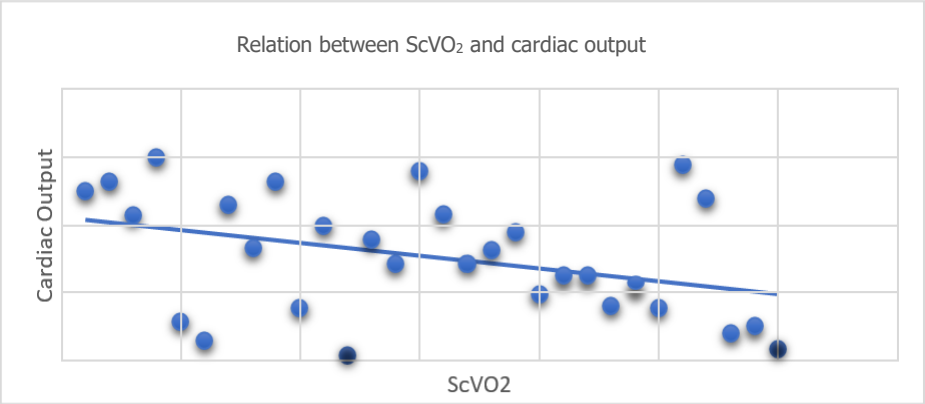


Figure 1. Relation between $ScVO_2$ and cardiac output

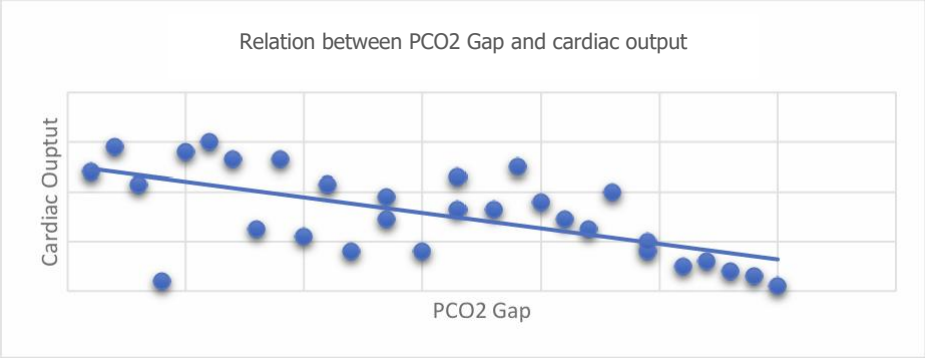


Figure 2. Relation between PCO_2 Gap and cardiac output

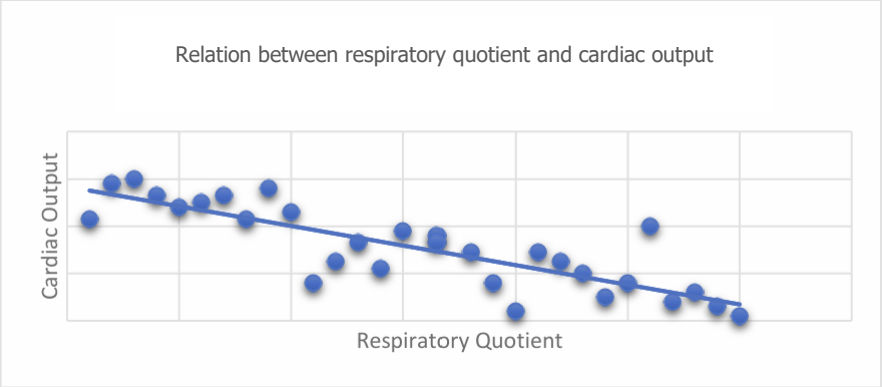


Figure 3. Relation between respiratory quotient and cardiac output

DISCUSSION

In this study, all data was taken at 1 time, from January until December 2024. Based on the data above from a total of 30 research samples, there were 14 male patients or 46.7% and 16 female patients. Evidence on the relationship between gender and arterial PCO_2 Gap remains limited, necessitating further investigation. However, several studies have identified that physiological factors that affect PCO_2 Gap can vary by gender.

In a study conducted by Zied et al. stated that variability in hemodynamic and metabolic parameters such as cardiac output and oxygen consumption can affect PCO_2 Gap. These factors can differ between men and women due to differences in physiology and response to certain clinical conditions such as sepsis or septic shock. Differences between men and women include body composition, blood volume, and cardiovascular function that can affect cardiac output and oxygen consumption. For example, women tend to have lower cardiac output than men. This can be caused by differences in muscle mass, hormones, and heart structure between men and women. Additionally, women also have a smaller blood volume, which can impact the ability to maintain cardiac output during clinical conditions such as sepsis or septic shock.[15]

Previous studies suggest that men and women may respond differently to sepsis or septic shock. Women tend to have a stronger inflammatory response, which can affect hemodynamic and metabolic parameters. This may be related to hormonal differences, especially estrogen, which plays an important role in modulating the immune response. These differences in response can affect the hemodynamic profile, oxygen consumption, and CO_2 production in male and female patients with sepsis. Hormonal differences between men and women, such as estrogen and testosterone, can affect metabolism and the body's response to sepsis. This can affect parameters such as oxygen consumption and CO_2 production. Estrogen, for example, is known to have a protective effect on cardiovascular function, while testosterone can affect energy metabolism. These differences in hormonal profiles can contribute to variability in hemodynamic and metabolic parameters between men and women.[15]

In this study, the results were 53.3% with an average age of 48.37 years with the youngest age being 21 years and the oldest age being 68 years. Age conditions greatly affect the determination of the PCO_2 Gap which can affect the study. In a cross-sectional study by Hari et al. it was stated that age is an important factor affecting the difference between arterial and venous PCO_2. Specifically, the correlation between arterial and venous PCO_2 was more consistent in younger patients compared to older patients. the correlation between arterial and venous PCO_2 was more consistent in younger patients compared to older patients. This may be due to age-related physiological changes, including impaired organ function, more comorbidities, and a reduced capacity to handle stress. In younger patients, the arterial-venous PCO_2 regulatory mechanism tends to be more stable, so the correlation between the two is also stronger. Conversely, in older patients, various age-related changes can cause greater variability in the arterial-venous PCO_2 gradient.[16]

In this study, patient respondents had an average body weight of 62.97 kg. Body weight can affect the PCO_2 Gap gradient. From research conducted by Alexander et al. stated that people with obesity tend to experience hypoventilation, which is breathing that is too slow or shallow to

effectively remove CO_2. This condition is known as obesity hypoventilation syndrome (OHS). This hypoventilation causes a buildup of CO_2 in the blood, which increases PCO_2 levels. In addition, in people with overweight conditions with excess weight including decreased lung function and related medical conditions such as sleep apnea and chronic obstructive pulmonary disease (COPD). These conditions can create an imbalance between ventilation and perfusion, which further increases CO_2 levels in the blood.[17]

MAP in this study sample had an average of 66.01 mmHg with the lowest MAP of 47 mmHg and the highest MAP of 95.33 mmHg. Determination of characteristics will correlate with increased mortality and multiorgan dysfunction in sepsis patients. Decreased MAP often causes inadequate tissue perfusion, which in turn increases the Pv-aCO_2 gradient due to accumulation of CO_2 in the tissue due to poor perfusion. Therefore, monitoring MAP and PCO_2 Gap can provide important information about tissue perfusion conditions in patients with sepsis. The correlation between MAP and PCO_2 Gap can be used as an indicator to assess the adequacy of perfusion and direct appropriate management in the management of patients with sepsis.[18]

The average hemoglobin in this study was 10.25. Hemoglobin plays an important role in CO_2 transport and buffering in the blood. When hemoglobin levels are low, the blood's capacity to carry CO_2 decreases, potentially affecting the PCO_2 Gap. In conditions such as anemia, decreased hemoglobin can lead to a higher PCO_2 Gap due to reduced CO_2 transport capacity and altered blood flow dynamics. et al. have shown that increased hemoglobin levels can lead to a reduction in plasma space, which in turn reduces bicarbonate (HCO_3-) and CO_2 content at a given PCO_2 level, thereby shifting the CO_2 dissociation curve to the right. This means that higher hemoglobin concentrations can result in a lower PCO_2 for the same CO_2 content, thus affecting the veno-arterial PCO_2 Gap.[19]

The mean SaO_2, $ScVO_2$, PaO_2, PvO_2, $PaCO_2$, $PvCO_2$, PCO_2 Gap, respiratory quotient, cardiac output and cardiac index in these patients were 96.22%; 84.28%; 143.75 mmHg; 33.16 mmHg; 37.01 mmHg; 40.29 mmHg; 3.27 mmHg; 0.958; 4.448 L/min; 2.714, respectively. These parameters collectively provide a comprehensive picture of the patient's hemodynamic status and guide management and resuscitation strategies in septic shock. Continuous monitoring of $ScVO_2$ and mixed central venous oxygen saturation (SvO_2) can provide insight into tissue oxygenation and perfusion in septic shock. High venous-to-arterial carbon dioxide (Pv-aCO_2) gap during early resuscitation is associated with poor outcomes, indicating inadequate tissue perfusion and oxygenation.[20] Arterial partial pressures of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) are critical in assessing respiratory function. Studies have shown that a high Pv-aCO_2 gap indicates inadequate cardiac output and poor tissue perfusion, often observed in septic patients despite adequate PaO2 and $PaCO_2$ values. The difference between combined venous and arterial CO_2 (Pv-aCO_2 gap) is used as an indicator of cardiac output and tissue perfusion. A persistently high Pv-aCO_2 gap is associated with increased mortality and poorer outcomes in patients with septic shock, suggesting that it may be a valuable marker for resuscitation efficiency.[21] The respiratory quotient, which is the ratio of CO_2 production to oxygen consumption, helps assess metabolic status. In septic shock, changes in respiratory quotient may indicate a shift in metabolic pathways, often due to hypoperfusion and anaerobic metabolism.[22]

Recent studies have shown that there is a significant correlation between $ScVO_2$ and cardiac output in the context of critical patient management. $ScVO_2$ is often used as an indirect indicator of the balance between oxygen supply and demand in the body. Research shows that low $ScVO_2$ values can indicate inadequate cardiac output, where the heart is unable to pump enough blood to meet the metabolic needs of the tissue. Conversely, normal or high $ScVO_2$ values can indicate adequate cardiac output and sufficient oxygen supply. Clinical studies have also shown that $ScVO_2$ monitoring can be a useful tool for optimizing hemodynamic therapy in patients with conditions such as sepsis and heart failure, because changes in $ScVO_2$ often reflect changes in cardiac output faster than other parameters. Thus, $ScVO_2$ monitoring can assist in clinical decision making for more appropriate interventions and faster times in critical patient management.[23]

This study shows that there is an important correlation between PCO_2 Gap (the difference in partial pressure of CO_2 between the veins and arteries) and cardiac output, especially in the context of critically ill patients. PCO_2 Gap is used as an indicator of tissue perfusion, with increased values often indicating hypoperfusion and anaerobic metabolism. The study showed that a high PCO_2 Gap can indicate low cardiac output, because decreased tissue perfusion causes CO_2 accumulation in the tissues that cannot be efficiently transported to the lungs for elimination. The study also found that PCO_2 Gap monitoring can provide additional information about the patient's hemodynamic status in addition to other parameters such as blood pressure and $ScVO_2$. In clinical interventions, a decrease in PCO_2 Gap accompanied by an increase in cardiac output indicates improved tissue perfusion and metabolic efficiency. Therefore, monitoring PCO_2 Gap together with cardiac output can help in assessing circulatory conditions and in decision making for more effective therapy in critically ill patients such as septic shock.[24,25]

Recent studies have shown that there is a significant correlation between respiratory quotient and cardiac output in patients with septic shock. In septic shock, the body experiences metabolic dysfunction and poor tissue perfusion, which can affect the respiratory quotient value. Study showed that in patients with septic shock, high respiratory quotient (close to or more than 1) often indicates increased carbohydrate metabolism and a shift to anaerobic metabolism due to tissue hypoperfusion and oxygen insufficiency. This is associated with low cardiac output, where the heart is unable to pump enough blood to meet tissue oxygen needs.[26] Studies have also found that medical interventions aimed at increasing cardiac output, such as fluid administration and vasopressors, can reduce respiratory quotient by improving tissue perfusion and restoring aerobic metabolism. Therefore, monitoring respiratory quotient together with cardiac output can provide important information about the patient's hemodynamic and metabolic status, assisting in clinical decision-making for more effective and timely therapy in patients with septic shock.[27]

However, in the study, 4 patients with negative PCO_2 Gap were found. The underlying cause of the finding of negative PCO_2 Gap differences is very rare and only possible in some conditions is that there are large cyclic changes in alveolar CO_2 during one respiratory cycle. During breathing, CO_2 levels in the alveoli can change significantly, especially with high tidal volumes (the volume of air inhaled or exhaled with each breath) and low respiratory rates. High tidal volumes cause

more air to enter and exit the lungs with each breath, which means more CO_2 is transported. With low respiratory rates, the time for gas exchange is also longer, causing greater variations in alveolar CO_2 levels.[28] In addition, other influences such as acute hyperoxygenation, Acute hyperoxygenation, or the administration of very high amounts of oxygen in a short time, can increase the partial pressure of CO_2 (PCO_2) in the veins. This can occur without changes in hemodynamic status (changes in blood flow or blood pressure). As a result, there is an increase in the PCO_2 Gap, which is the difference between the CO_2 pressure in the arteries and veins. This condition can occur because hyperoxygenation reduces the respiratory stimulus, resulting in CO_2 retention. The combination of acute hyperventilation with respiratory alkalosis can cause a negative PCO_2 Gap. Acute hyperventilation causes an increase in the respiratory rate and volume, which reduces the blood CO_2 level ($PaCO_2$). A decrease in $PaCO_2$ from 44 to 34 mmHg, for example, can cause respiratory alkalosis, a condition in which the blood becomes more alkaline because excess CO_2 is exhaled. This decrease in $PaCO_2$ can also significantly increase the PCO_2 Gap, because the reduction in arterial CO_2 is greater than the reduction in venous CO_2.[29] The researcher's BIAS error can also affect the results, measurement errors, such as sample contamination with liquid or air bubbles, and inaccuracies in the gas analyzer. Errors in blood gas measurements can occur due to several factors, such as sample contamination with liquid or air bubbles, and inaccuracies in the gas analyzer. Contamination can cause inaccurate results, for example, the presence of liquid can reduce the concentration of the gas being measured, while air bubbles can add certain gases that are not from the blood sample itself. Inaccuracy of the analyzer can also give incorrect results, either too high or too low. Another factor that cannot be ignored is when the sampling time is not right, the blood gas sampling is not timely, such as taking samples before the end of expiration, can give inaccurate results. At the end of expiration, the CO_2 level in the alveoli is most stable and representative of the body's metabolic conditions. If the measurement is done before this point, the results may not reflect the actual CO_2 levels in the alveoli and blood, thus causing errors in the interpretation of the results.[30]

CONCLUSION

The conclusion of this study is that in patients with septic shock, there is a correlation between several variables with cardiac output. One of them is $ScVO_2$, where an increase in $ScVO_2$ tends to be followed by a decrease in cardiac output. In addition, PCO_2 Gap (the difference between arterial and venous PCO_2) is also related to cardiac output, where the greater the PCO_2 Gap, the smaller the cardiac output that occurs. Likewise with the respiratory quotient, which shows that the higher the respiratory quotient value, the lower the cardiac output in patients with septic shock.

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