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ORIGINAL ARTICLE

Role of central venous to arterial carbon dioxide tension difference in hemodynamic optimization and its correlation with cardiac index after major abdominal surgery: "A prospective observational study"

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ABSTRACT

Introduction: The use of central venous to arterial carbon dioxide tension difference (ΔPCO_2) as a marker of overall perfusion even when macro-circulatory indicators appear to be normal is gaining ongoing support. This study aimed to assess the utility of ΔPCO_2 in hemodynamic optimization in patients following major abdominal surgeries.

Patients and methods: For this prospective observational study, 50 consecutive patients admitted to the ICU following major abdominal surgeries, with $ScvO_2 \geq 70\%$ and fitting our inclusion criteria were included once admitted (T_0). Patients were separated into low ΔPCO_2 group (n=27) and high ΔPCO_2 group (n=23) according to a threshold of 6 mmHg at T_0. Demographics, SAPS II and SOFA scores were recorded at T_0, while hemodynamic and biological parameters including CI, ΔPCO_2, $ScvO_2\%$ and lactate level were recorded at T_0, T_6, T_{12} and T_{24}.

Results: At T_0, there was a significant difference between the high and the low ΔPCO_2 patients for ΔPCO_2 (8.7±2.7vs4.1±1.5mmHg, P<0.001), serum lactate (2.5±1.1vs1.2±0.9mmol/L, P<0.05) and SOFA score (9(4-14)vs3(2-4), P<0.05) but not for $ScvO_2\%$. CI was significantly higher in the low ΔPCO_2 group (4.4±1.2vs2.4±0.9 $L/min/m^2$ P<0.001). From T_0 to T_{24}, CI at T_6 was still significantly higher in the low ΔPCO_2 group (4.3±1.0vs2.2±0.8$L/min/m^2$, P<0.001). At T_0 and T_6, ΔPCO_2 and CI values were inversely correlated (T_0:P<0.001 and T_6:P<0.05).

Conclusion: ΔPCO_2 could be used to mirror inadequate tissue perfusion when $ScvO_2 \geq 70\%$ has already been recorded and hence provide a valuable addition to resuscitation targets in post surgical patients.

Keywords: ΔPCO_2, PCO_2 gap, cardiac index, major abdominal surgery, hemodynamic.

INTRODUCTION

Identifying defects in the supply of both blood and oxygen to the tissue is of a major significance in early resuscitation of patients with critical illness **[1, 2]**. Tissue hypoxia is one of the most recognized causes of postoperative organ dysfunction and mortality following major surgery **[3]**. The rise in tissue requirements for oxygen associated with surgical trauma if not swiftly managed by boosting oxygen delivery to the tissues, grave outcomes are often encountered **[2, 3]**.

In practice, goal directed individual based fluid replacement therapy during the perioperative period of high risk surgical procedures, is having an ongoing support. Desiring optimal outcome, several markers of impaired tissue oxygenation have been explored in an attempt to identify patients who are inadequately perfused and consequently are at increased risk of complications **[4, 5]**. Postoperative organ dysfunction has been associated with reduced central venous oxygen saturation ($ScvO_2\%$), a measure that reflects the balance between O_2 delivery (DO_2) and tissue oxygen consumption (VO_2) **[6]**. However, $ScvO_2\%$ may not reflect tissue hypoxia when VO_2 is

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impaired by mitochondrial dysfunction or cytopathic hypoxia, or when microcirculatory failure results in shunting of blood away from metabolically active yet hypoxic tissues **[7, 8]**.

Numerous studies have established the utility of serum lactate concentration as a marker of global tissue hypoxia in circulatory shock. Hyperlactatemia is a common finding in the postoperative settings and strategies developed in an attempt to reduce high serum lactate levels were often accredited for the reduction of the length of stay and mortality **[9, 10]**. However, hyperlactatemia after surgery may not be a reliable option for judging the adequacy of tissue oxygenation, as other mechanisms such as the stress response to surgery and the use of B-adrenergic agonists may account for the rise in blood lactate rather than anaerobic metabolism **[2, 11]**. Nevertheless, a diagnostic elevation in blood lactate level is rather delayed, when it is put up against other indicators of tissue oxygenation, making its sensitivity questionable. So other circulatory parameters are often needed for a more efficient assessment of the resuscitation efforts **[3]**.

The gap between central venous and arterial carbon dioxide tensions (ΔPCO_2), which is the outcome of subtracting carbon dioxide tension in a blood sample drawn via a central venous catheter and that in an arterial blood sample, has been suggested. ΔPCO_2 values of 2-5 mmHg are considered normal under optimal conditions and usually don't exceed 6 mmHg **[12]**. ΔPCO_2 is now regarded as a marker of the efficiency of venous blood flow (cardiac output) to remove total carbon dioxide produced by peripheral tissue and its use as a marker of overall perfusion even when macrocirculatory indicators appear to be normal is gaining ongoing support **[12, 13]**. Hence, the defective perfusion of tissues brought about by an inadequate circulatory flow is thought to be the major influencing factor in a raised ΔPCO_2 **[14]**.

Previous studies examined the association between high ΔPCO_2, at different measuring points, and post admission outcome. However, the study of ΔPCO_2 in the context of major surgery and the perioperative period is still limited **[4, 15]**.

For the current study we hypothesized that ΔPCO_2 is useful in identifying patients who are still inadequately resuscitated on admission to the ICU following major abdominal surgery despite having a ScvO2% value of ≥70%.

PATIENTS AND METHODS

This study was conducted over a period of 2 years (from July 2017 to July 2019). 50 patients, admitted to ICU following major abdominal surgeries and with a $ScVO_2\% > 70\%$, were included. The study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Sample size:

By comparing mean and standard deviation of ΔPCO_2 in a previous study **[4]** and finding it to be 8.7±2.8 and 5.1±2.6mmHg respectively, sample size was calculated to be 50 cases using Epi Info with an 80% power of test and a confidence interval of 95%.

Study design: A prospective observational study (cross section).

Out of 63 patients admitted to the ICU following abdominal surgeries, the 1st 50 patients, fitting the inclusion criteria, were studied immediately after their admission to the ICU while 13 patients were excluded for failing to fit the inclusion criteria. The current study included patients of both sexes, aged between 18 to 60 years, ASA class I-III, admitted to the ICU following a major abdominal surgery (lasting more than 2 hours and/or with intraoperative blood loss exceeding 1000cc) provided that their $ScvO_2\% > 70\%$ at admission. On the other hand, this study excluded those aged less than 18 and more than 60 years, pregnant females, those with sepsis and/or coagulopathy on admission and chronic obstructive pulmonary disease patients. Once admitted, a written informed consent was obtained from all participants or their first degree relatives. Patient's demographics (age, sex and body

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mass index (BMI)), type and duration of the surgical procedure were recorded. SAPS II (Simplified Acute Physiology Score II) and SOFA (Sequential Organ Failure Assessment) scores were done as a part of the initial assessment. An electrical cardiometry was applied to monitor cardiac output. In all patients blood samples were analyzed for routine laboratory investigations, blood gases, $ScvO_2$%, serum lactate and ΔPCO_2 according to which patients were allocated into one of two groups based on their initial ΔPCO_2 value on admission to the ICU [a high gap group ($\Delta PCO_2 \geq 6$ mmHg) and a low gap group ($\Delta PCO_2 < 6$ mmHg)]. The cut off value of 6 mmHg was based on previous studies **[12, 16]**.

Samples were obtained at T_0 (at admission to the ICU), T_6 (6 hours later), T_{12} (12 hours later) and T_{24} (24 hours postoperatively). In addition mean arterial pressure (MAP), heart rate (HR) and cardiac index (CI) were recorded at the same preset time points. Regarding the hemodynamic and metabolic measurements, MAP was continuously measured using an arterial catheter. Lactate blood concentration was assessed in arterial blood samples. Central venous oxygen saturation was obtained via blood sampling from a central venous line positioned so that its tip is in the superior vena cava (position verified by X-ray), Cardiac index (L/min/m^2) was assessed via Electrical cardiometry by ICON - CARDIOTRONIC, OSYPKA MEDICAL. For this purpose, four sensors were applied- first: approximately 5 cm above left base of the neck, second on the left base of neck, third on the lower left thorax at level of xiphoid and the fourth one on the lower left thorax approximately 5 cm below the 3rd electrode at the level of anterior axillary line.

ΔPCO_2 was calculated as the difference between carbon dioxide tensions in blood samples obtained from central venous and arterial catheters. Patients were managed according to our standard ICU management protocol in accordance with the international guidelines. Resuscitation was done while assessing fluid responsiveness following boluses of 250:500 cc of fluids. Fluid responders were those with a 15% or more increase in their stroke volume. Crystalloids were of choice and blood products with a ratio of 1:1:1 for those with a hemorrhagic deficit. For non fluid responders vasopressors were considered when MAP was ≤50 mmHg. Norepinephrine was our 1st choice.

At T_0, patients of both groups were compared for their demographics (age, gender and BMI), type and duration of surgery, SAPS II score, SOFA score, biological ($ScvO_2$%, ΔPCO_2 and serum lactate concentration) and hemodynamic parameters (MAP, HR and CI). Biological and hemodynamic parameters were also compared at T_6, T_{12} and T_{24}. Moreover, CI was correlated with ΔPCO_2, $ScvO_2$% and serum lactate at each of our preset time points.

Study outcome measures:

The primary outcome of the current study was to establish the utility of ΔPCO_2 value in hemodynamic optimization, while the secondary outcome was to assess if ΔPCO_2 values were correlated to CI values in those patients.

STATISTICAL ANALYSIS

Analysis of data was done using *Statistical Program for Social Science version 2.0 (SPSS Inc., Chicago, IL, USA).* Quantitative variables were described in the form of *mean and standard deviation*. Qualitative variables were described as *number and percent*. In order to compare parametric quantitative variables between two groups, *Student t test* was performed. Qualitative variables were compared using *Chi-square (X2) test or Fisher's exact test* when frequencies were below five. *Pearson's correlation coefficients* were used to assess the association between two normally distributed variables. When a variable was not normally distributed, *Mann Whitney test* was used to compare two non parametric variables. Probability value (P value) ≤0.05 was considered statistically significant.

RESULTS

Initial data:

On admission, the low ΔPCO_2 group had a significantly lower SOFA score when compared to the high ΔPCO_2 group [3(2-4) versus 9(4-

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14)] respectively ($P<0.05$) **(Table 1)**. The mean values of ΔPCO_2 were significantly higher in the high ΔPCO_2 group compared to the low ΔPCO_2 group [8.7±2.7 versus 4.1±1.5 mmHg] respectively ($P<0.001$) **(Fig 1)**. On the other hand, cardiac index was significantly higher in the low ΔPCO_2 group when compared to the high ΔPCO_2 group [4.4±1.2 versus 2.4±0.9 L/min/m^2**]** ($P<0.001$). In addition, serum lactate mean values were significantly higher in the high ΔPCO_2 group [2.5±1.1 versus 1.2±0.9 mmol/L] ($P<0.05$) **(Table 2)**. There was no significant difference between the two groups for all the other parameters measured on admission (T_0) **(Table 1, 2).**

Trend of data between T0 and T24:

Observing the trend of changes in ΔPCO_2 mean values between admission and T_{24} reveals a progressive decline in ΔPCO_2 between admission and towards T_{24} in both the high and the low ΔPCO_2 groups in response to ongoing medical management. The decline was more prominent in the high gap group, in particular between admission and T_6, however no significant difference between the mean values of ΔPCO_2 in the two groups other than that found at T_0. For patients in the low ΔPCO_2 group, the decline was less notable and the trend was rather stationary between T_6 and T_{24} **(Fig 1)**.

As for serum lactate, an initial increase in the mean values of serum lactate in both the high and the low ΔPCO_2 group was observed between admission and T_6. This increase can be attributed to the relative delay in the lactate elevation. A significant difference in serum lactate values in both groups was observed at T_{12}. From T_6 up to T_{24} the trend of mean values was towards a decline in serum lactate levels **(Fig 2)**.

Trends in mean cardiac index values between admission and T_{24} was also observed, apart from T_0 to T_6, no significant difference has been shown between the two groups however, cardiac index values showed a progressive increase in response to ongoing resuscitation with a coexisting decrease in ΔPCO_2 values in patients of the high ΔPCO_2 group **(Fig 3)**.

It should be noted that at all of our preset time point of the study, $ScvO_2$% values were more than 70% with no statistically significant difference between both groups **(Fig 4)**.

Correlation analysis:

At T_0 and T_6, a strong negative (inverse) correlation was found between ΔPCO_2 and CI values while at T_{12} and T_{24} the correlation was rather weak and insignificant. On the other hand there was no correlation between $ScvO_2$% and CI values at each of our preset time points. At T_0 and T_{12}, a weak positive correlation was established between ΔPCO_2 and serum lactate values, while no correlation was found at other time points **(Table 3).**

Table (1): Patient's characteristics on admission (T_0):

Characteristics	High ΔPCO2 (N=23)	Low ΔPCO2 (N=27)	P value
Age (years)	**42±16**	**36±15**	**0.52**
Gender (male)	**10(43%)**	**12(44%)**	**0.46**
BMI (Kg/m2)	**27±0.8**	**26±3**	**0.42**
Type of surgery			
Bowel resection	**10(43%)**	**9(33%)**	**0.53**
Hepatectomy	**1(4.3%)**	**0(0%)**	**0.53**
Splenectomy	**7(30%)**	**10(37%)**	**0.51**
Nephrectomy	**2(8.6%)**	**3(11%)**	**0.56**
Hystrectomy	**3(13%)**	**5(18.5%)**	**0.52**
Duration of surgery (min)	**140±32**	**155±26**	**0.43**
SAPS II score	**26(19-34)**	**17(12-28)**	**0.06**
SOFA score	**9(4-14)**	**3(2-4)**	**<0.05***

- Age, BMI and duration of surgery are expressed as mean ± standard deviation.-

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- SOFA: Sequential Organ Failure assessment score, expressed as median.
- SAPS II: Simplified Acute Physiology Score II, expressed as median.
- Gender described as male out of the total number and percentage of patients within each group.
- Type of surgery is expressed as a number and percentage out of total patients within each group.
- ΔPCO_2: difference between central venous to arterial carbon dioxide tensions.
- *P values less than 0.05 are considered significant.

Table (2): Hemodynamic and biological parameters of the studied groups on admission (T_0):

Variables	High Δ PCO2 (N=23)	Low Δ PCO2 (N=27)	P value
Hemodynamic parameters:			
Vasopressors	**8(34.7%)**	**6(22.2%)**	**0.23**
Mechanical ventilation	**9(39.1%)**	**7(25.9%)**	**0.36**
MAP (mmHg)	**64±13**	**68±10**	**0.33**
HR (bpm)	**93±9**	**89±9**	**0.63**
CI (L/min/m2	**2.4±0.9**	**4.4±1.2**	**<0.001****
Biological parameters:			
ΔPCO2 (mmHg)	**8.7±2.7**	**4.1±1.5**	**<0.001****
Lactate (mmol/L)	**2.5±1.1**	**1.2±0.9**	**<0.05***
ScvO2 (%)	**76±4**	**77±3**	**0.069**

- Data are expressed as mean ± standard deviation.
- Patients on vasopressor therapy and mechanical ventilation are expressed as actual number and percentage out of total patients within each group.
- MAP: mean arterial pressure.
- HR: heart rate.
- C.I.: cardiac index.
- ΔPCO_2: difference between central venous to arterial carbon dioxide tensions.
- $ScvO_2$%: central venous oxygen saturation.
- *P values less than 0.05 are considered significant.
- **P values less than 0.001 are considered highly significant.

Table (3): Correlation between cardiac index (CI) and ΔPCO_2, $ScvO_2$% and serum lactate at different time points:

Variable	*R	P value
ΔPCO2 at T_0	**0.61**	**<0.001**
ΔPCO2 at T_6	**0.59**	**<0.05**
ΔPCO2 at T_{12}	**0.24**	**0.08**
ΔPCO2 at T_{24}	**0.35**	**0.13**
ScvO2% at T_0	**0.19**	**0.12**
ScvO2% at T_6	**0.21**	**0.16**
ScvO2% at T_{12}	**0.18**	**0.09**
ScvO2% at T_{24}	**0.24**	**0.08**
Lactate at T_0	**0.36**	**<0.05**
Lactate at T_6	**0.39**	**<0.05**
Lactate at T_{12}	**0.12**	**0.16**
Lactate at T_{24}	**0.19**	**0.12**

- *R: Pearson correlation.
- CI: cardiac index (L/min/m^2)
- ΔPCO2: difference between central venous to arterial carbon dioxide tensions (mmHg).

- ScvO2%: central venous oxygen saturation (%).
- Lactate (mmol/L).
- T0: on admission, T6: 6 hours after admission, T12: 12 hours after admission and T24: 24 hours after admission.
- *P values less than 0.05 are considered significant.

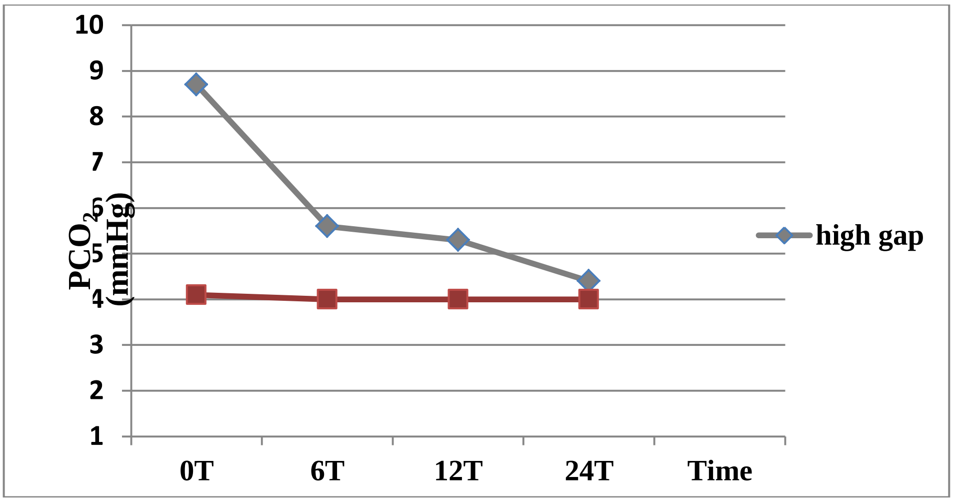

Figure (1): Changes in $\Delta PC0_2$ (mmHg) at different time intervals in the two studied groups.

- Data are expressed as mean values.
- ΔPCO_2: difference between central venous to arterial carbon dioxide tensions.
- Gap: difference between central venous to arterial carbon dioxide tensions.
- T0: on admission, T6: 6 hours after admission, T12: 12 hours after admission and T24: 24 hours after admission.

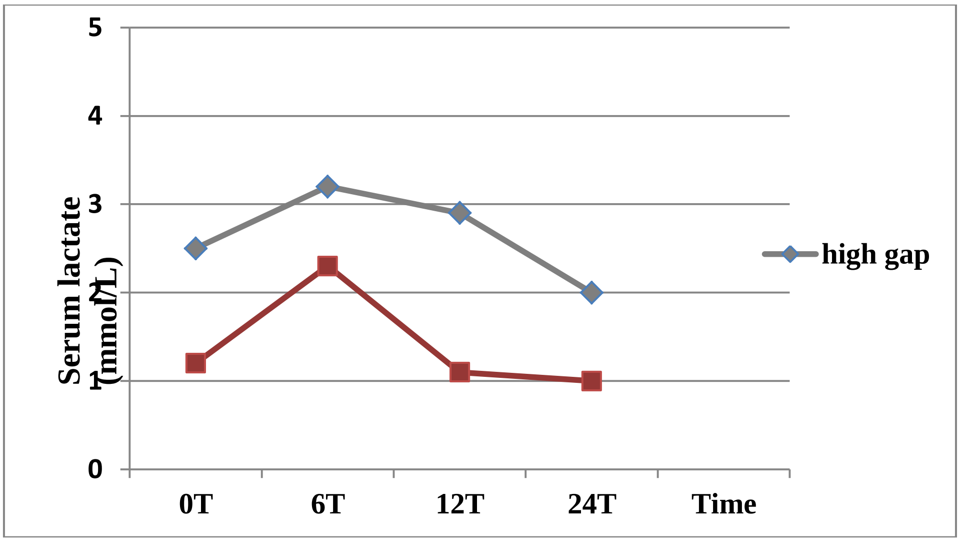

Figure (2): Changes in serum lactate values (mmol/L) at different time intervals in the two studied groups.

- Data are expressed as mean values.
- Serum lactate (mmol/L).
- Gap: difference between central venous to arterial carbon dioxide tensions.
- T0: on admission, T6: 6 hours after admission, T12: 12 hours after admission and T24: 24

hours after admission.

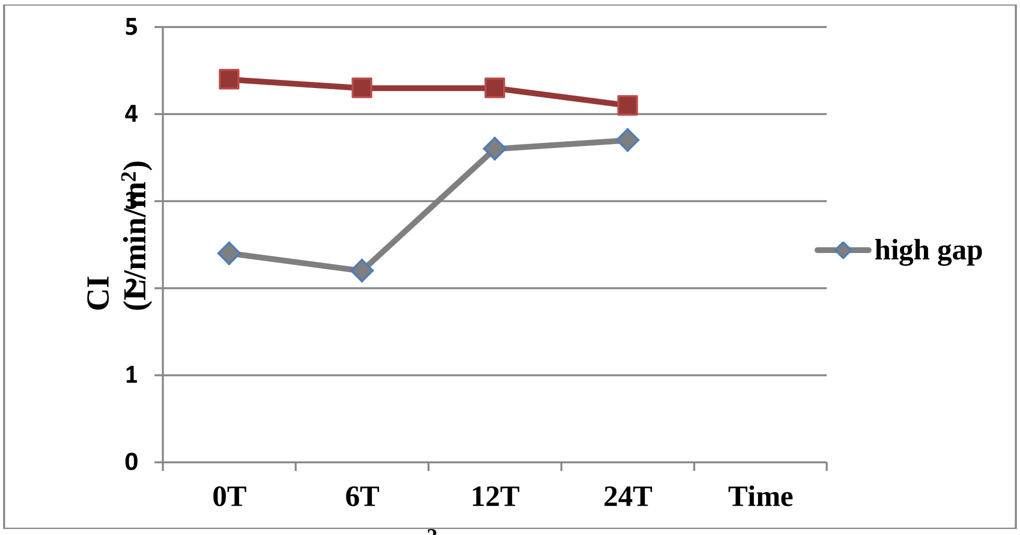

Figure (3): Changes in CI values (L/min/m^2) at different time intervals in the two studied groups.

- ➢ Data are expressed as mean values.
- ➢ C.I.: cardiac index (L/min/m^2).
- ➢ Gap: difference between central venous to arterial carbon dioxide tensions.
- ➢ T0: on admission, T6: 6 hours after admission, T12: 12 hours after admission and T24: 24 hours after admission.

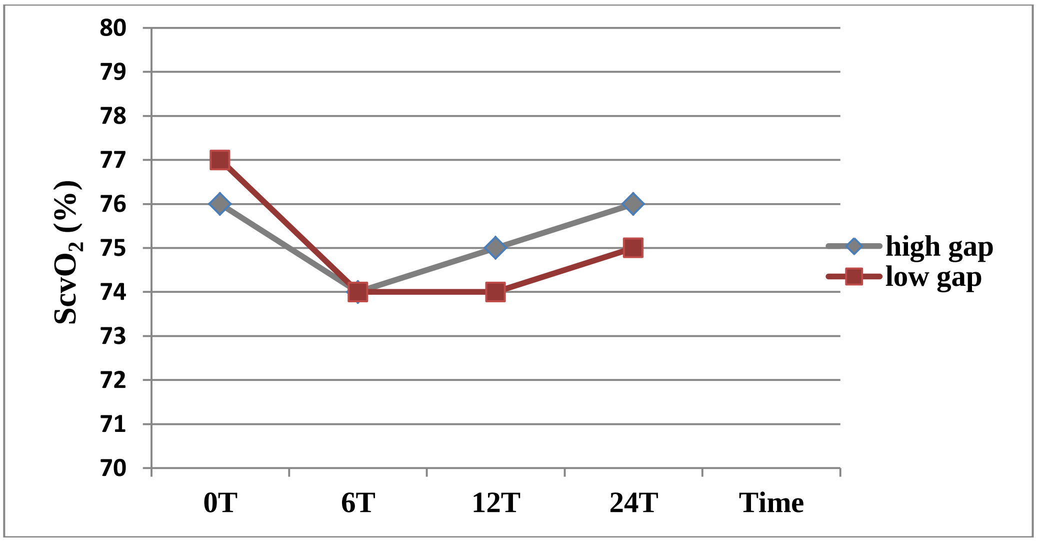

Figure (4): Changes in $ScvO_2$ (%) at different time intervals in the two studied groups.

- ➢ Data are expressed as mean values.
- ➢ $ScvO_2$%: Central venous oxygen saturation (%).
- ➢ Gap: difference between central venous to arterial carbon dioxide tensions.
- ➢ T0: on admission, T6: 6 hours after admission, T12: 12 hours after admission and T24: 24 hours after admission.

DISCUSSION

The main findings of our study were that, on admission to the ICU (T_0), ΔPCO_2 mean values were the highest compared those measured at other time points with a significant difference between the high and the low ΔPCO_2 groups (8.7±2.7 versus 4.1±1.5 mmHg) respectively (P<0.001).

$ScvO_2$% values were > 70% in both groups (76±4.0 and 77±3.0, P> 0.05). The high ΔPCO_2 group had a significantly lower cardiac index (2.4±0.9 versus 4.4±1.2 L/min/m^2, P<0.001) together with a significantly higher serum lactate (2.5±1.1 versus 1.2±0.9 mmol/L, P<0.05) which is consistent with a state of hypoperfusion. In general, the possible explanations for elevated ΔPCO_2 are **[12]**:

1- Low flow-induced CO_2 stagnation yielding a secondary build up in venous PCO_2 levels.

2- An increase in respiratory quotient attributed to the buffering induced increase in CO_2 by buffering excess H ions by bicarbonate with persistent additional CO_2 production (VCO_2), relative to the O_2 uptake.

3- A combined increase in CO_2 production and stagnation although $ScvO_2$ >70%**.**

For this last explanation, we can notice that at T_0 significantly higher ΔPCO_2 values were observed together with a lower CI in patients with high ΔPCO_2 which is consistent with a certain degree of hypoperfusion, while $ScvO_2$% values exceeded 70% in both groups. Because $ScvO_2$ is measured downstream from tissues, when a given tissue receives inadequate DO_2, the resulting low local oxygen venous saturations may be masked by admixture with highly saturated venous blood from tissues with better perfusion and DO_2, resulting overall in normal or even high $ScvO_2$% and so targeting $ScvO_2$ values ≥70% as a sole goal may not be sufficient to guide resuscitation efforts. In addition normal or high $ScvO_2$% values don't exclude microcirculatory failure **[3, 13]**. Reflecting on the previous results, ΔPCO_2 seems to be relevant in identifying patients who still need resuscitation efforts.

This is in accordance with the finding of **Robin et al. [4]**, who studied central venous-to-arterial carbon dioxide difference as a prognostic tool in high-risk surgical patients. Their study revealed a significant difference in the ΔPCO_2 and CI values between both groups at ICU admission following major abdominal and/or vascular surgeries and concluded that the increase in ΔPCO_2 was secondary to tissue hypoperfusion.

Another study is that preformed by **Vallee et al. [12],** who studied the gap as a possible additional target to goal directed therapy in septic patients. The study involved 50 patients showed that at T_0 low ΔPCO_2 group patients had a significantly lower ΔPCO_2, while patients with high ΔPCO_2 had significantly lower CI values. All this was associated with a ScvO2 value ≥70% and hence they concluded that targeting $ScvO_2$ % as a sole indicator was not sufficient to guide therapy and that the presence of a high ΔPCO_2 might be a useful tool to identify those who are not adequately resuscitated.

Mecher et al. [17], also observed that septic shock patients with ΔPCO_2 > 6 mmHg had a significantly lower mean cardiac output when compared to patients with ΔPCO2 ≤ 6 mmHg. Some studies suggested a relationship between mixed venous-to-arterial carbon dioxide difference [P (v-a) CO_2] and CI in circulatory failure and septic shock **[18, 19]**. **Cushieri and his colleagues [20]** showed that this correlation still existed when the venous-to arterial CO_2 tension difference was calculated with ΔPCO_2 measured from a central venous blood sample.

Furthermore, the current study demonstrated a strong negative correlation between ΔPCO_2 and CI on admission to the ICU (T_0) and 6 hours following admission (T_6) [T_0: r = 0.61, P<0.001 and T_6: r = 0.59, P<0.05].

These results are in accordance with those of **Robin et al. [4].** Their study was demonstrated a significant difference between the CI of both groups at ICU admission. **Van Beest and his colleagues [21]** preformed a post hoc analysis on 53 septic shock patients and found a negative correlation between ΔPCO_2 and CI values on admission to the ICU.

Vallee and colleagues [12] found a negative correlation between CI and ΔPCO_2 at 3 time points T_0, T_6 and T_{12}. The possible reason for the disagreement between our current results and that of **Vallee et al** at T_{12} is the difference in the type of patients. As our

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study involved postoperative patients while their study involved patients with sepsis and septic shock.

Similarly, several studies involving a more diverse group of patients with various types of shock come to results that are in accordance with the current study **[16, 17, 19, 23].**

On the other hand, there was no correlation between $ScvO_2$% and CI values [T_0: r = 0.19, P = 0.12; T_6: r = 0.21, P = 0.16; T_{12}: r = 0.18, P = 0.09 and T_{24}: r = 0.24, P = 0.08].

In previous studies, $ScvO_2$% also failed to correlate with CI or show any significant difference between the study groups at any time point **[12, 21]**. For **Vallee and colleagues [12]**, $ScvO_2$% values were above 70% in both groups throughout the study.

In our current study, patients with low ΔPCO_2 had lower lactate levels for the next 24 hours following admission compared to those of high ΔPCO_2 group. Moreover, at T_0 and T_{12}, a weak positive correlation was established between ΔPCO_2 and serum lactate values [T_0: r = 0.36, P <0.05 and T_{12}: r = 0.41, P <0.05] respectively.

The previous finding is consistent with the results obtained by **He et al. [3]**. Their study involved 84 septic patients and attempted to prove the association between the ΔPCO_2 and lactate clearance.

Another study in accordance with the current result is that of **Robin et al. [4]**, in which a median serum lactate level of 1.54 mmol/L in the group suffering post operative complications versus 1.06 mmol/L in the group with no complications. On the other hand, **Vallee and his colleagues [12]** failed to establish a correlation between ΔPCO_2 and serum lactate values. It should be noted that the absolute values of serum lactate in postoperative patients of the current study are less remarkable compared to septic patients of the previously mentioned studies. This difference is attributed to the fact that our surgical patients benefited from immediate hemodynamic support in the operating room and intensive care. Thus, lactate elevations may not be a reliable mean of judging the adequacy of tissue oxygenation as it may occur as a result of other mechanisms as previously mentioned **[3]**.

The results of our current study are rather suggestive of the upper edge of ΔPCO_2 over $ScvO_2$ and serum lactate values.

Limitations: This study had limitations including the relatively small sample size, its observational nature and hence we had no power over therapeutic interventions and also data were based on measurements at 4 time points and not continuous measurements which may not provide an ideal reflection of the course of variables alterations.

CONCLUSION

Reflecting on the previous findings, ΔPCO_2 could be used to mirror inadequate tissue perfusion when $ScvO_2$ > 70% has already been recorded and hence provide a valuable addition to resuscitation end points in post surgical patients. Further research is recommended to assess the optimal use of this measure as a resuscitation endpoint.

Conflict of interest: None

Financial disclosure: Nothing to declare

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