

## Research Paper

# End-tidal Carbon Dioxide Measurements in Unintentional Non-fire-related Carbon Monoxide Poisoning



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## ABSTRACT

**Background:** Poisoning with carbon monoxide occurs occasionally worldwide, and the gold diagnostic standard is to measure carboxyhemoglobin level in the blood. This study investigated the correlation between carboxyhemoglobin and the end-tidal carbon dioxide levels in 50 patients with carbon monoxide poisoning.

**Methods:** We recruited 50 volunteer patients who had been admitted to the Emergency Services of Istanbul Medipol University Hospital between January 2017 and January 2018. They had been diagnosed with carbon monoxide poisoning unrelated to fire accidents. The arterial and venous blood gases, and other blood and clinical parameters were also measured. The patients' end-tidal carbon dioxide levels were measured from the nose and mouth air, using a Capnostream 20p bedside monitor. Pearson's correlation analyses were performed and the results were compared with the end-tidal carbon dioxide, carboxyhemoglobin and oxygen saturation in the arterial and venous blood samples.

**Results:** The Mean±SD age was 33.98±10.89 years. The Mean±SD arterial and venous carboxyhemoglobin values were 18.05±7.10 and 12.11±9.67, respectively. There were no statistically significant differences between the oxygen saturation, and the arterial and venous blood levels of carboxyhemoglobin (P=0.870, P=0.950), respectively. Also, no statistically significant correlations were found between the end-tidal carbon dioxide, and the arterial and venous carboxyhemoglobin levels (P=0.529, P=0.601), respectively.

**Conclusion:** The results from the blood analyses demonstrated that there was no statistically significant difference between the end-tidal carbon dioxide and the carboxyhemoglobin levels in these patients who had been earlier diagnosed with carbon monoxide poisoning, unrelated to fire accidents.

**Keywords:** Carbon dioxide, Carboxyhemoglobin, Carbon monoxide poisoning, End-tidal CO<sub>2</sub>

## Introduction

Carbon monoxide (CO) is an odorless, tasteless, colorless, and non-irritating gas formed by the combustion of hy-

drocarbons. It binds hemoglobin with stronger affinity than oxygen, where carboxyhemoglobin (COHb) is formed and interferes with oxygen transport and utilization in the body [1]. Carbon monoxide spreads rapidly through the pulmonary capillary membranes and

binds the iron moiety of heme (and other porphyrins) about 240 times stronger than the affinity of oxygen [2]. The half-life of CO is about 250 to 320 minutes in room air, approximately 90 minutes when inhaled with high flow oxygen via non-rebreathing face mask, and around 30 minutes with 100 percent hyperbaric oxygen (HBO) treatment. The diagnosis of CO poisoning is based on a history consistent with a high COHb level and physical examinations [1]. A noninvasive pulse CO and oxygen meter to perform spectrophotometric measurements of COHb are in the process of being developed. However, preliminary observational studies question the accuracy of this method [3].

The term capnography refers to the non-invasive measurement of the partial pressure of CO<sub>2</sub> in the inhalation air over time. The end-tidal CO<sub>2</sub> (EtCO<sub>2</sub>) level, which is the maximum fraction of CO<sub>2</sub> at end expiration, measures the physiological state of patients continuously, non-invasively and indirectly. Capnography may be used to confirm the status of a number of clinical tests, e.g. endotracheal tube location in intubated patients, and to continually monitor tube placement during transport. It is also used to check the effectiveness of resuscitation during cardiac arrest and is a prognostic indicator of “return of spontaneous circulation (ROSC)” during chest compressions. Further, capnography is used for assessing the prognosis of trauma and adequacy of ventilation [4-9]. In addition, in patients with non-intubated spontaneous breathing, capnography is utilized for the quick evaluation of critical diseases, determination of response to treatment in acute respiratory distress syndrome, ventilation adequacy in unconscious or procedural sedoanalgesia patients, and to obtain a prognostic basis in patients in septic shock [10-13].

**Aim of the study:** The aim of this study was to investigate the relationship between the arterial and venous COHb levels versus the EtCO<sub>2</sub> levels in patients with unintentional non-fire-related poisoning with CO. To the best of our knowledge, such an investigation has not been conducted previously in patients with respiratory conditions.

## Materials and Methods

This prospective observational study was conducted between January 2017 and January 2018 at the Emergency Department of the University Hospital, Faculty of Medicine, [Istanbul Medipol University](#), Istanbul, Turkey. This department serves an average of 97,000 patients per year. The study protocol was approved by the Ethics Committee, Faculty of Medicine, [Istanbul Medipol Uni-](#)

[versity](#), as a clinical trial (Session No: 2017/02, Decision No: 01). This study was subsequently conducted over a 12-month period (January 2017 and 2018).

**Selection of subjects:** Patients who were admitted to the emergency department on suspicion of CO poisoning and having a blood COHb level of  $\geq 10\%$ , and aged above 18 years were included in this study. Patients with fire-associated smoke poisoning were excluded, because of the suspicion that they could have been exposed to toxic gases other than CO, e.g. cyanide. In addition, patients were also excluded from the study if they were involved in any of the following: suicidal CO intoxication, drug abuse, metabolic acidosis (uremia, diabetic ketoacidosis, lactic acidosis, renal tubular acidosis, sepsis etc.), respiratory diseases (chronic obstructive pulmonary disease, asthma, pneumonia, pulmonary thromboembolism etc.), multiple traumas or being under 18 years of age.

**Study protocol:** The Etiological, demographic and clinical characteristics of patients were recorded in standardized forms. The patients' blood samples were collected to check the arterial and venous blood gases, hemogram, and basic biochemical and cardiac parameters if they were suspected of CO poisoning. Blood samples collection was carried out in the emergency room within 20 minutes of admission. A 12-lead electrocardiography (ECG) was performed for each of these patients. End-tidal CO<sub>2</sub> (EtCO<sub>2</sub>) levels were recorded for the patients with spontaneous respiration, using a Capnostream 20p bedside monitor. This device had a dual-purpose nasal cannula connection that supplied oxygen to patients and collected EtCO<sub>2</sub> samples from the mouth and nose air. This device had been programmed to deliver oxygen without affecting the CO<sub>2</sub> measurements. Thus, patients were given 100% oxygen at 10 L/min and the EtCO<sub>2</sub> levels and the waveforms were recorded simultaneously.

Patients suspected of CO poisoning received 10L/min normobaric oxygen (NBO) treatment independently of SpO<sub>2</sub>, and without waiting to check the COHb levels. The CO poisoning was diagnosed based on a clinical triad: a) symptoms consistent with CO poisoning; b) history of recent CO exposure; and c) elevated COHb levels [14]. The NBO treatment was continued until the patients were discharged with the oxygen supply of 10 L/min for 25 minutes and a pause for 5 minutes. The COHb, SpO<sub>2</sub> and EtCO<sub>2</sub> levels, ECG and other cardiac markers were tested repeatedly at 4-hour intervals. Patients with a COHb level less than 5% and with improved symptoms were discharged from the emergency department. The levels of EtCO<sub>2</sub>, arterial and venous COHb, and SpO<sub>2</sub> were also compared with those obtained at admission.

**Statistical analyses:** Data were analyzed, using SPSS software, version 21.0 (IBM Corporation; Armonk, NY, USA). The data were also analyzed by Kolmogorov-Smirnov normality test to check the distribution of continuous variables. The data were expressed as means±standard deviations, nonparametric or normal distribution as median (minimum-maximum) and the qualitative data were expressed as percentages. The data reflecting the normal distributions were checked with Pearson's correlation analysis. Also, the data that did not meet the normal distribution, were tested by Spearman's correlation analysis. The statistical significance level was set at  $P<0.05$ .

## Results

Fifty patients presenting to the university hospital were included in this study, the majority of whom were women (72%) with the remainder being men (28%). During the study, the total number of referrals to the emergency department were 97,468 individuals. Among them, CO poisoning represented 0.05% of all referrals to that emergency department. The patients mean age was  $33.98\pm 10.89$  years old, and 60% of them were brought to the hospital by ambulance. The patients' admission process to the hospital took between 10 to 710 minutes, with the mean duration for the admission process being 130

**Table 1.** Demographic and clinical findings of the patients (n=50)

Demographic & Clinical Feature	Values	Mean±SD
Age (y)	-	33.98±10.89
Gender	Female	-
	Male	-
Systolic BP (mmHg)	-	124.44±17.57
Diastolic BP (mmHg)	-	70.56±9.82
Heart rate (HR)	-	91.22±14.94
Respiratory rate (RR)	-	20.40±3.42
Oxygen saturation	-	97.39±2.18
Admission type (%)	By ambulance	60%
	Outpatient	40%
Admission location (%)	City center	86%
	Village	14%
CO Exposure (hr)	-	3.75±2.83

**Table 2.** Patients' symptoms (n=50)

Symptom	No. (%)
Headache	92
Dizziness	78
Nausea	70
Vomiting	14
Chest Pain	6
Shortness of breath	18
Syncope	18

**Table 3.** Laboratory findings in patients (n=50)

Laboratory Finding	Mean±SD
Arterial COHb (%)	18.05±7.10
Venous COHb (%)	12.11±9.67
EtCO <sub>2</sub> (mmHg)	35.69±4.31

COHb: Carboxyhemoglobin; EtCO<sub>2</sub>: End-tidal CO<sub>2</sub>; SpO<sub>2</sub>: Oxygen saturation; SD: Standard deviation

minutes. The duration of exposure to CO gas was calculated at 3.75±2.83 hours. The demographic and clinical characteristics of the patients are summarized in Tables 1 and 2. The patients' mean arterial and venous COHb values were 18.05±7.10 and 12.11±9.67, respectively. Table 3 summarizes the data representing other laboratory tests.

Based on Pearson's correlation analysis, the CO gas exposure time and EtCO<sub>2</sub> values were positively correlated (P=0.326, r=0.142). In addition, while the duration of exposure to CO gas, and the arterial and venous COHb values showed a positive correlation (P=0.471; r=0.104; & P=0.986, r=0.006, respectively), no correlation was found with SpO<sub>2</sub> (P=0.552, r=0.086). There was no statistically significant difference between the SpO<sub>2</sub> and arterial and venous COHb values (P=0.870 & P=0.950, respectively). Based on the Pearson's correlation analysis, there was no correlation between the EtCO<sub>2</sub> level and the arterial and venous COHb values (P=0.529, r=-0.091 & P=0.601, r=-0.076, respectively). In addition, the EtCO<sub>2</sub> level was found to be positively correlated with SpO<sub>2</sub> (P=0.508, r=0.096) (Table 4).

## Discussion

Due to the socioeconomic status and climatic characteristics of many countries, CO poisoning occurs at varying frequencies throughout the world. In Turkey during 2010, the rate of CO poisoning was 14 in hundred thousand in-

dividuals (0.014%), while the death rate was five in ten million people (0.38%) [15]. The CO poisoning rate in the U.S. has been estimated at 50,000 per year (0.016%) [1]. Recent studies indicate that the deaths associated with CO poisoning have declined to approximately 1,300 in 2014 from about 2,700 in the mid-2000s [1]. Based on the national data in the United States, the total mortality rate due to CO poisoning is 1-3% [2]. Between 2001-2003, the rate of CO poisoning was 0.23% among all poisoning cases in South Korea, with the mortality rate being 1.85% [16].

Similar to other studies, the frequency of CO poisoning in the current study was 0.051%; however, there were no deaths. This was mainly due to the fact that the geographic region is windy, thus the people are aware of CO poisoning originating from the household stoves. Therefore, they go to the hospitals as soon as they notice the early signs of CO poisoning. In addition, on days when there is a high risk of stove poisoning from wind-borne stoves in our region, the local government warns the public about precautions against stove poisoning. There is a high probability of death due to stove poisoning in the elderly who live alone. However, individuals in advanced age group do not usually live alone in Turkey. The fact that the patients in our study came from a younger age group and the absence of comorbidities could be the factors that contributed to no deaths in our study.

**Table 4.** Correlation of end-tidal CO<sub>2</sub> with other continuous variables

EtCO <sub>2</sub>	r	P
pHa	-0.405	0.004
pCO <sub>2</sub>	0.493	<0.001
Lactate	-0.526	<0.001
Arterial COHb	-0.091	0.529
Venous COHb	-0.076	0.601
SpO <sub>2</sub>	0.096	0.508

EtCO<sub>2</sub>: End-tidal CO<sub>2</sub>; pHa: Arterial pH; COHb: Carboxyhemoglobin; SpO<sub>2</sub>: Oxygen saturation; P: Level of statistical significance; r: Correlation coefficient

The gold standard in the diagnosis of CO poisoning is detecting a high level of COHb in the blood after exposure [14]. In a previous study on CO poisoning, the COHb concentrations measured by CO-oximetry were strongly correlated with the CO<sub>2</sub> concentration [17]. However, there was no significant relationship between standard pulse oximetry and COHb values in patients in advanced stages of CO poisoning. They also showed hemodynamic impairment, in whom no significant correlation was found between the COHb values as measured by oximetry and the patients' severe clinical condition [17, 18]. In one of the earlier studies [17], no statistically significant correlation was found between the exhaled CO value and the patients' clinical severity. In another study, it was emphasized that since pulse oximetry did not show arterial oxygenation, relying overly on it may cause delay in detecting clinically significant hypoxemia [19]. Arterial carbon dioxide pressure, an indicator of ventilation, cannot be detected by pulse oximetry. Therefore, if hypercapnia or hypoventilation is suspected, arterial blood gas should be measured immediately. Alternatively, the patients' ventilation should be evaluated by EtCO<sub>2</sub> monitoring [20].

In a former study [21], it was reported that COHb level did not correlate with the SpO<sub>2</sub> values. In CO poisoning cases, high SpO<sub>2</sub> values obtained from pulse oximeters may lead to false confidence and mask the life-threatening arterial oxygen desaturation due to high COHb levels. This can cause mortality and morbidity in the victims. Our study results were consistent with those reported in the literature earlier [21]. In this context, we did not find a significant correlation between SpO<sub>2</sub> value and both the arterial and venous COHb levels. Further, there was no significant relationship between the EtCO<sub>2</sub> and SpO<sub>2</sub> values. The main reasons for this may be the fact that our patients were in a younger age group and there was no severe CO poisoning to cause mortality. Furthermore, the lack of hospitalization in our case series may be due to the fact that not all patients received hyperbaric oxygen therapy since their CO poisoning was fairly mild.

Nowadays, capnography is part of the routine care of all patients receiving general anesthesia in Turkey and elsewhere. It is also a necessary method of monitoring metabolic and respiratory conditions in pre-hospital and emergency departments. Many studies have emphasized that EtCO<sub>2</sub> values are promising in predicting severe clinical and respiratory conditions. In the literature, the initial finding of return of spontaneous circulation (ROSC) during cardiopulmonary resuscitation (CPR) has been emphasized as a sudden increase in EtCO<sub>2</sub>

pressure [5]. In previous studies, it has been reported that capnography can be used as the gold standard in the emergency department to confirm the placement of the endotracheal tube [4, 6].

In patients who underwent procedural sedoanalgesia in the pediatric emergency department, there was a statistically significant increase in EtCO<sub>2</sub> levels at 5, 10 and 15 minutes compared to the baseline, although there was no decrease in the SpO<sub>2</sub> level. During the sedoanalgesia procedure, it was emphasized that the routinely used EtCO<sub>2</sub> level could signify respiratory safety in terms of the development of respiratory depression [12]. Another study has reported that EtCO<sub>2</sub> measurement could accurately predict arterial pCO<sub>2</sub> in patients admitted to the emergency room with complaints of dyspnea [10]. It has been suggested in the literature that EtCO<sub>2</sub> levels are significantly different between cardiac and obstructive causes of respiratory distress [9].

In a study conducted in emergency rooms by Kheng, et al. [11], they investigated the relationship between the EtCO<sub>2</sub> levels and hypotensive patients' mortality, either in the hospital or 30-day post-discharge. These authors found that in the patients with decreased EtCO<sub>2</sub> the level was significantly lower than in those who survived. Thus, the non-invasive monitoring of EtCO<sub>2</sub> levels in emergency department patients who were in shock was found to be a critical monitoring method [11]. Upon our literature review, it was revealed that there was an inverse relationship between mortality and EtCO<sub>2</sub> values in patients with trauma. Also, studies have shown a correlation between low EtCO<sub>2</sub> levels and the severity of the patients' injuries [7, 8].

In the current study, we did not find a significant correlation between EtCO<sub>2</sub> and COHb levels from the arterial or venous blood. We believe that the main reason is that there was no mortality in our patient group because there was no serious CO poisoning. Further studies are warranted to confirm this hypothesis.

## Conclusions

To the best of our knowledge, the association between end-tidal carbon dioxide level and COHb in unintentional, non-fire-related CO poisonings have not been studied before. This study found no statistically significant difference between the COHb and EtCO<sub>2</sub> levels in patients suffering from CO poisoning not associated with fire. We believe that this was a pioneer step for future studies to be conducted in larger sample sizes of patients with moderate to severe CO poisoning.

**Limitations of the study:** The main limitation of our study was that it conducted over a 1-year period at a single center. Also, the fact that we had limited number of patients due to exclusions, e.g. suicidal cases, was another limitation. Lastly, the lack of patients with moderate to severe CO poisoning and without mortality may be considered as another limitation of this study.

**Recommendations for future studies:** This research was a single-center study, consisting of patients with mild CO poisoning. Our data can potentially guide other multicenter studies on patients with moderate to severe CO poisoning.

## Ethical Considerations

### Compliance with ethical guidelines

This study was approved by the Ethics Committee for Clinical Trials from both the Department of Emergency Medicine, Faculty of Medicine, [Istanbul Medipol University](#), and Department of Cardiology, Faculty of Medicine, [Kahramanmaraş Sutcu Imam University](#), Kahramanmaraş, at Istanbul and Kahramanmaraş, Turkey, respectively (Joint session #: 2017/02, Decision No: 01).

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### Authors' contributions

Developed the research concept: Fatih N. Yaman and Hakan Hakkoymaz; Developed the theory and performed the computations: Ozlem Guler and Murat Kerkutluoglu; Verified the analytical methods: Murat Kerkutluoglu; Discussed the results, and contributed to the writing of the final draft of the manuscript: All authors.

### Conflict of interest

The authors declare no conflict of interest with any internal or external entities in conducting this research.

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