

Can End-tidal Carbon Dioxide Levels Be Used for Determining Tissue Oxygen Saturation in Smokers and Nonsmokers?

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ABSTRACT

Objectives: End-tidal carbon dioxide (EtCO₂) monitoring has been used in a range areas, such as invasive sedation procedures, during cardiopulmonary resuscitation, ventilation monitoring of patients with altered mental status, trauma and respiratory system diseases in emergency departments. Near-infrared spectroscopy (NIRS) allows continuous, noninvasive measurement of tissue hemoglobin oxygen saturation (StO₂) in muscle and has been studied for a wide range of conditions.

In this study we aim to determine the levels of EtCO₂ and StO₂ in smokers and nonsmokers and the relationship between these two parameters.

Methods: We examined 201 healthy volunteers including 156 smokers and 45 nonsmokers. We measured thenar muscle StO₂ via NIRS device. The EtCO₂ was determined by a capnograph. Baseline measurements were obtained from all participants after not-smoking for two h. Second measurements were taken 5 min after smoking was finished.

Results: The mean baseline EtCO₂ of the smoker group was 42.61±4.94 (min:31, max:54) mmHg and mean baseline StO₂ for this group was 77.81±4.71% (min:69, max:86). In smokers, mean baseline EtCO₂ (p:0.00), baseline StO₂ (p:0.001), fifth min EtCO₂ (p:0.00), fifth min StO₂ (p:0.02) levels, and pack*year (p:0.00) were significantly different between males and females. In nonsmokers baseline EtCO₂ and baseline StO₂ levels were correlated (p:0.035,r:0.315). But we couldn't find significance between females and males for mean baseline EtCO₂ (p:0,246) and baseline StO₂ (p:0.264) levels in this group.

Conclusion: Smoking affects tissue oxygenation. In nonsmokers baseline EtCO₂ and baseline StO₂ levels were correlated, but there was no correlation for these two parameters in smokers. Clinicians may use EtCO₂ levels for predicting the tissue oxygenation in nonsmoker patients.

Keywords: end-tidal carbon dioxide, near-infrared spectroscopy, tissue hemoglobin oxygen saturation, smoker, nonsmoker

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INTRODUCTION

Capnograph was originally used for validation of endotracheal tube placement during cardiopulmonary resuscitation (CPR) studies in emergency practice [1,2]. But during recent years, end-tidal carbon dioxide (EtCO₂) monitoring via capnograph has been used in a range of areas such as invasive sedation procedures, cardiopulmonary resuscitation effectivity, ventilation monitoring of patients with altered mental status, trauma and respiratory system diseases [3-7].

Near-infrared spectroscopy (NIRS) allows continuous, noninvasive measurement of tissue hemoglobin oxygen saturation (StO₂) in muscle and has been studied in a wide range of conditions [8]. NIRS has been used in critically ill patients especially in intensive care units (ICUs) and also researches have still been studying the efficiency and areas of use of this device [9]. It has also been used to classify the severity of hemorrhagic shock, as a guide for fluid resuscitation, and during elective surgery septic shock, multiple - organ dysfunction

EtCO₂ and StO₂ in Smokers

Table 1. Baseline StO₂ and baseline EtCO₂ levels of smokers and nonsmokers according to gender

	Smoker Group		Nonsmoker group		p
	F	M	F	M	
Age	30.94±8.0 (min:18,max:52)	34.06±10.58 (min:18,max:67)	36.12±10.9 (min:23,max:49)	37.61±16.74 (min:24,max:71)	0.55
	Total: 33.02±9.88 (min:18,max:67)		Total: 37.04±17.72 (min:23,max:71)		
Baseline EtCO ₂ (mmHg)	39.79±3.53 (min:31,max:45)	44.02±4.94 (min:35,max:54)	44.06±3.24 (min:38,max:50)	43.11±4.71 (min:36,max:54)	0.195
	Total: 42.61±4.94 (min:31,max:54)		Total: 43.47±4.21 (min:36,max:54)		
Baseline StO ₂ (%)	75.96±5.23 (min:69,max:86)	78.73±4.16 (min:70,max:86)	75.71±5.58 (min:65,max:81)	75.32±2.85 (min:70,max:80)	0.004
	Total: 77.81±4.71 (min:69,max:86)		Total: 75.47±4.05 (min:65,max:81)		

syndrome and as a vital sign prehospitally [10-16].

In this study we aim to determine the levels of EtCO₂ and StO₂ in smokers and nonsmokers and the relationship between these two parameters and besides whether EtCO₂ levels may help clinicians to predict the StO₂ levels in smoker and nonsmoker patients.

METHOD

Subjects

We examined 201 healthy volunteers: 156 smokers and 45 nonsmokers. Participants with chronic or acute diseases, history of drug usage, pregnant women and volunteers under 18 years of age were excluded from the study.

StO₂

In our study, we measured thenar muscle StO₂ via wide-gap second-derivative NIRS (InSpectra; Hutchinson Technology). This device serves to predict the hemoglobin StO₂ in the microvasculature of muscle tissue, including the arteriolar, capillary and venular compartments [17].

EtCO₂

EtCO₂ was determined by the EMMA capnograph (Masimo Sweden AB), which measures end-tidal pressure by means of an infrared-absorbing sensor. We also used an EMMA airway adaptor and 7.0 endotracheal tube to evaluate the expiratory breathing.

Experimental Protocol

Our study was approved by the Human Study Committee of our medical faculty (10.04.2014/43). The volunteers were informed and gave their informed consent. All measurements took place in a temperature-controlled environment. The study group was comfortably seated and breathing normally before, during and after the measurement. Smokers had been instructed to refrain from smoking for at least 2 h prior to the measurements. Baseline measurements were obtained from all participants. Smokers were then asked to smoke a cigarette according to their

habitual brand, maintaining the usual aspiration pattern. Second measurements were taken 5 min after smoking was finished. Each participant held the endotracheal tube placed to the airway adapter of the capnograph in his or her mouth and expired for 5 seconds. The last values on the screen were recorded before and after smoking. Similarly, for the StO₂ levels, an InSpectra device was placed to the right thenar muscle for 10 s and mean of the first, fifth and 10th second values were noted.

Statistical Analysis

The normal distribution and homogeneity of each parameter were tested using the Shapiro–Wilk test and the Kolmogorov–Smirnov test. Age, gender, baseline EtCO₂, baseline StO₂, fifth min EtCO₂ and fifth min StO₂ values did not suit the normal distribution. A Mann–Whitney–U test was used for differences between and among two groups. A Spearman correlation test was used for correlation analysis. In all tests the significance level was p<0.05. SPSS (Statistical Package for the Social Sciences) software 18.0 was used for all analysis.

RESULTS

Our study included 156 smokers (77.6%) and 45 nonsmokers (22.4%), a total of 201 healthy volunteers. Mean age of the smoker group was 33.02±9.88 (min:18, max:67). The mean baseline EtCO₂ of the smoker group was 42.61±4.94 (min:31, max:54) mmHg, and the mean baseline StO₂ for this group was 77.81±4.71% (min:69, max:86). Age, baseline EtCO₂, baseline StO₂, and cigarette pack*year distributions of the groups according to gender are detailed in **Table 1**. The mean fifth min EtCO₂ of the smoker group was 47.11±5.33 (min:33, max:57) mmHg and the mean fifth min StO₂ of the smoker group was 81.71±4.73% (min:73, max:91). The mean cigarette pack*year was 14.24±14.88 (min:1, max:80) in the smoker group. In the smoker group, mean baseline EtCO₂ (p:0.00), baseline StO₂ (p:0.001), fifth min EtCO₂ (p:0.00), fifth min StO₂ (p:0.02) levels, and

pack*year (p:0.00) were significantly different between males and females (**Table 1**).

In the smoker group the mean second ET_{CO}₂ level was 43.77±4.373 (min:33, max:50) mmHg, mean second StO₂ level was 80.29±5.17% (min:73, max:88) and mean pack*year was 8.71±7.59 (min:1, max:30) in females and; these values for males were 48.78±4.99 mmHg (min:36, max:57) mmHg, 82.42±4.35% (min:74, max:91), and 17.01±16.77 (min:1, max:80) respectively.

In nonsmokers baseline EtCO₂ and baseline StO₂ levels were correlated (p:0.035, r:0.315). But we couldn't find significance between females and males for mean baseline EtCO₂ (p:0.246) and baseline StO₂ (p:0.264) levels in this group.

Age was correlated with baseline StO₂ (p:0.00, r:0.29) but there was no correlation with baseline EtCO₂ (p:0.216, r:-0.08). In the smokers group, baseline EtCO₂ was correlated with fifth min EtCO₂ (p:0.00, r:0.75) and pack*year (p:0.003, r:0.23) but we couldn't determine a correlation with baseline StO₂ (p:0.880, r:-0.012) and fifth min StO₂ (p:0.47, r:0.57). Similarly baseline StO₂ was correlated with fifth min StO₂ (p:0.00, r:0.84) and pack*year (p:0.00, r:0.30). Pack*year was also positively correlated with fifth min EtCO₂ (p:0.003, r:0.23) and fifth min StO₂ (p:0.001, r:0.26).

DISCUSSION

In our study, baseline StO₂ levels of smokers were higher than those of nonsmokers, but ET_{CO}₂ values didn't differ. In nonsmokers baseline EtCO₂ and baseline StO₂ levels were correlated. But there was no correlation for these two parameters in smokers.

In the literature, there have been several studies on the use of emergency EtCO₂ monitoring. Wahlen et al's study investigated EtCO₂ values in spontaneously breathing patients. They searched for the influence of EtCO₂ monitoring on therapeutic decisions by emergency physicians. Their study included 299 patients were included and reported pathological EtCO₂ values in 19 patients (6.3%). They suggested that EtCO₂ monitoring may be a useful additional variable in spontaneously breathing patients [17].

Another study examined the relationship among EtCO₂ and other oxygenation markers such as arterial oxygen pressure (PaO₂), carbon dioxide pressure (PaCO₂), and pulse oximetry (SpO₂). Giner performed this study in 57 active smokers. In this study, the mean SpO₂ of the participants was 95%, and oxygen saturation measured directly in arterial blood samples was 95.1%. The mean EtCO₂ was 37.9 mm Hg and PaCO₂ by arterial blood gas analysis was 40.6 mm Hg. They found a correlation between EtCO₂-SpO₂ and EtCO₂-PaCO₂. Although this suggested that both oxyhemoglobin saturation assessments and both CO₂ pressure determinations were closely related, it cannot be inferred that the 2 techniques in each case are completely

congruent. Pulse oximetry and capnography are both useful for monitoring oxyhemoglobin saturation or ventilation [18].

In a recent study, StO₂ monitoring for acute effects of smoking has been reported by Sifaka et al. Similarly to our study, they used NIRS to compare the microcirculatory function of smokers with that of nonsmokers. They studied 25 smokers and 40 nonsmokers, but their study protocol was different from ours except for its measurement times and measurement area (thenar region). They asked the participants not to smoke for 2h before the baseline measurement and took the last measurement after 5 min too. But they preferred a continuous StO₂ monitoring. They recorded StO₂ baseline values for 5 min. Subsequently, the brachial artery occlusion technique was applied to evaluate microcirculatory function before, during, and after smoking a cigarette. In that study, StO₂ before smoking was 85, which did not differ significantly between genders and StO₂ did not change significantly during smoking. O₂ consumption rate was significantly greater in women at baseline and throughout the smoking session. O₂ consumption rate was reduced during smoking and at 5 min after the smoking session. Smoking had a significant effect on vascular reactivity, with no significant differences between genders. Five minutes after smoking, vascular reactivity had returned to approximately normal levels [19].

In another study, Terborg et al searched for cerebral blood flow and oxygenation in 24 healthy volunteers via transcranial Doppler sonography and near-infrared spectroscopy before, during, and after smoking a cigarette. They simultaneously recorded cerebral blood flow velocity of both middle cerebral arteries, mean arterial blood pressure, skin blood flow, EtCO₂, changes in concentration of cerebral oxyhemoglobin, deoxyhemoglobin, and total hemoglobin, and a cerebral tissue oxygenation index. Smoking increased cerebral blood flow velocity, oxyhemoglobin, and total hemoglobin. After smoking, the increase in cerebral blood flow velocity and total hemoglobin persisted, while oxyhemoglobin returned to baseline. Deoxyhemoglobin and cerebral tissue oxygenation index did not change during the whole procedure. During smoking, but not after, cerebral blood flow velocity increase was correlated to ipsilateral changes in oxyhemoglobin and total hemoglobin. In this study, tissue oxygenation index was 64.6% before smoking, 65.5% during smoking, and 64.6% after smoking. Mean EtCO₂ was 41.4 mmHg and 40.2 mmHg after smoking [20].

Akca et al examined brain tissue oxygenation and EtCO₂ monitoring in 20 surgical patients to determine the postoperative infection risk. In this study, the patients underwent intraoperative EtCO₂, tissue oxygen partial pressure, transcutaneous oxygen tension and cerebral oxygen saturation monitoring via cerebral oximeter. The authors reported that mild intraoperative hypercapnia increased subcutaneous and cerebral oxygenation [21].

CONCLUSION

In our study, baseline StO₂ levels of smokers were higher than those of nonsmokers, but EtCO₂ values did not differ. As a result of this, we can say that smoking affects tissue oxygenation. In nonsmokers, baseline EtCO₂ and baseline StO₂ levels were correlated, but there was no correlation for these two parameters in smokers. EtCO₂ measurement devices are much more easy to reach and cheaper when compared with near infrared spectroscopy both in emergency departments and intensive care units. These results indicate that EtCO₂ levels of nonsmoker patients may help clinicians to predict the StO₂ levels.

Because of lack of literature, further comprehensive studies are needed to explain the reasons for these changes in acute and long term smoking the use of these parameters especially the StO₂ in the ED.

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