

# The use of capnometry to predict arterial partial pressure of CO<sub>2</sub> in non-intubated breathless patients in the emergency department

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Received: 16 July 2009 / Accepted: 26 August 2010 / Published online: 9 December 2010  
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## Abstract

**Background** Capnometry measures carbon dioxide in expired air and provides the clinician with a noninvasive measure of the systemic metabolism, circulation and ventilation. This study was carried out on patients with acute breathlessness to define the utility and role of capnometry in the emergency department.

**Aim** The objectives of the study were:

1. To determine the correlation between end tidal CO<sub>2</sub> and PaCO<sub>2</sub> in non-intubated acutely breathless patients.
2. To determine factors that influence the end tidal carbon dioxide (ETCO<sub>2</sub>).
3. To determine the correlation between ETCO<sub>2</sub> with PaCO<sub>2</sub> in patients presenting with pulmonary disorders.

**Methods** One hundred fifty acutely breathless patients arriving at the emergency department and fulfilling the inclusion and exclusion criteria were chosen during a 6-month study period. The patients gave written or verbal consent, and were triaged and treated according to their presenting complaints. Demographic data were collected, and the ETCO<sub>2</sub> data were recorded. Arterial

blood gas was taken in all patients. The data were compiled and analyzed using various descriptive studies from the Statistics Program for Social Studies (SPSS) version 12. Correlation between ETCO<sub>2</sub> and PaCO<sub>2</sub> was analyzed using the Pearson correlation coefficient. Other variables also were analyzed to determine the correlation using simple linear regression. The agreement and difference between ETCO<sub>2</sub> and PaCO<sub>2</sub> were analyzed using paired sample t-tests.

**Results** There is a strong correlation between ETCO<sub>2</sub> and PaCO<sub>2</sub> using the Pearson correlation coefficient: 0.716 and p value of 0.00 (p<0.05). However, the paired t-test showed a mean difference between the two parameters of 4.303 with a p value<0.05 (95% CI 2.818, 5.878). There was also a good correlation between ETCO<sub>2</sub> and acidosis state with a Pearson correlation coefficient of 0.374 and p value 0.02 (p<0.05). A strong correlation was also observed between ETCO<sub>2</sub> and a hypocapnic state, with a Pearson correlation coefficient of 0.738 (p<0.05). Weak correlation was observed between alkalosis and ETCO<sub>2</sub>, with a Pearson correlation coefficient of 0.171 (p<0.05). A strong negative correlation was present between ETCO<sub>2</sub> and hypercapnic patients presenting with pulmonary disorders, with a Pearson correlation coefficient of -0.738 (p<0.05) and of -0.336 (p<0.05), respectively.

**Conclusion** This study shows that ETCO<sub>2</sub> can be used to predict the PaCO<sub>2</sub> level when the difference between the PaCO<sub>2</sub> and ETCO<sub>2</sub> is between 2 to 6 mmHg, especially in cases of pure acidosis and hypocapnia. Using ETCO<sub>2</sub> to predict PaCO<sub>2</sub> should be done with caution, especially in cases that involve pulmonary disorders and acid-base imbalance.

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**Keywords** Capnometry · End tidal · Dyspnea · Blood gas

## Introduction

Acute breathlessness has been recognized as an important medical problem, and attempts have been made to improve the management of such conditions [1, 2]. It is a complex sensation that involves both objective and subjective elements. Unlike other noxious sensations, dyspnea does not have a defined neural pathway, and the perceived difficulty probably arises from the interaction of several pathophysiological mechanisms [3]. It occurs in healthy individuals as well, e.g., during intense emotional states and heavy labor or exercise. As a symptom, it can be hard to treat and often causes poor quality of life and severe disability [4, 5].

Therefore, whenever a patient presents with dyspnea, it is important to perform a thorough examination, including objective as well as subjective measures. Approximately two thirds of patients presenting to the ED with dyspnea have either cardiac or pulmonary disorders [6, 7]. Assessment of cardiopulmonary status is a must in every patient presenting with dyspnea. Physicians can usually distinguish these on the basis of patient history, physical examination and occasionally with the help of ancillary tests because dyspnea may result from many disorders. Monitoring and evaluation of dyspneic patients in the emergency department (ED) require devices that can provide information regarding the respiratory, cardiovascular and also metabolic status of the patient [8–10]. One important parameter that is commonly associated with dyspnea is an arterial partial pressure of carbon dioxide ( $P_a\text{CO}_2$ ). Carbon dioxide ( $\text{CO}_2$ ) is produced during cellular metabolism. It is first transported by the venous system to the right side of the heart and is then pumped to the lung by the right ventricle, reflecting cardiac output and blood flow to the pulmonary system [11]. Although an arterial blood gas (ABG) examination provides accurate information, an arterial blood analyzer is not available in every ED, and blood is usually sent to the laboratory. This whole procedure is time consuming and in some cases requires multiple arterial blood punctures to get one ABG sample. Furthermore, the ABG procedure is very painful and invasive for the patients.

In recent years, capnometry has emerged as a useful way to measure carbon dioxide tension in intubated patients [12]. Capnometry is a non-invasive tool that can measure end tidal  $\text{CO}_2$  ( $\text{ETCO}_2$ ). Capnometry has been used to monitor the adequacy of alveolar ventilation during anesthesia, to wean patients from mechanical ventilation, and as an indirect measure of cardiac output and likelihood of resuscitation during cardiac arrest [13–15]. However, the utility and accuracy of portable capnometers in non-intubated patients have not been fully examined [16]. Capnometry provides clinicians with a noninvasive

measure of several dynamic systems in the body, including the systemic metabolism; circulatory system, particularly the cardiac output and blood flow to the lung; and the ventilatory system [17]. Changes in the  $\text{CO}_2$  level in the expired air reflect changes in one or more of these systems. For example, if there are no changes in cardiac output or minute ventilations, hypermetabolic states such as sepsis or malignant hyperthermia will show increases in  $\text{CO}_2$  levels [18].

This original research was undertaken to determine if the capnometer has the potential to be used as a method of predicting arterial carbon dioxide in acutely breathless patients in the ED. We hope that it can be used as a non-invasive, rapid assessment tool that will greatly facilitate the evaluation and reassessment of dyspneic patients in the ED. The Ethics and Research Committee of the University Sains Malaysia approved this research in 2006.

## Objectives

The objectives of the study were:

1. To determine the correlation between end tidal  $\text{CO}_2$  and  $\text{PaCO}_2$  in non-intubated acutely breathless patients.
2. To determine factors that influence the end tidal carbon dioxide ( $\text{ETCO}_2$ ).
3. To determine the correlation between  $\text{ETCO}_2$  and  $\text{PaCO}_2$  in patients presenting with pulmonary disorders.

## Methodology

We conducted a cross-sectional study on acutely breathless patients who presented to the emergency department over a 6-month period from 1 October 2005 to 30 April 2006. The study was conducted at the Emergency Department of the Hospital University Sains Malaysia, a regional tertiary referral center with an attendance rate exceeding 55,000 patients per year in the emergency department. The target population was patients presenting to the emergency department with acute dyspnea of various etiologies. Various demographic and clinical data were recorded. A purposive sampling method was used for selection of the study group. Written or verbal informed consent was obtained from each subject before enrollment into the study. All patients received standard emergency treatment according to their presenting complaint and clinical findings.

The  $\text{ETCO}_2$  in the exhaled air was measured using microstream Oridion Smart CapnoLine<sup>®</sup>  $\text{CO}_2$  sampling, which was connected to a monitoring system to display an

**Table 1** Characteristics of the study population

Parameter	Results
Patients (n)	150
Male (%)	66.7
Female (%)	33.3
Age (years)	54.2 (95% CI, 43.6–59.8)
SBP (mmHg)	138.4 (95% CI, 135.5–141.5)
DBP (mmHg)	79.2 (95% CI, 75.7–84.8)
Heart rate (beat/min)	94.6 (95% CI, 91.4–98.5)
Respiratory rate (per min)	24.2 (95% CI, 19.3–26.7)
Temperature (C)	37.3 (95% CI 36.5–38.7)

accurate waveform and numerical value of ETCO<sub>2</sub>. The Oridion Smart Capnoline® uses a special canulla that is capable of measuring exhaled CO<sub>2</sub> from both the nose and mouth [19]. The oral position of the canulla was adjusted to place the ports in the midline at the mid-position of the open mouth. Each patient was monitored by one investigator until a consistent 3-min ETCO<sub>2</sub> reading was obtained using a capnometer built into a Datascope® cardiac monitor (manufactured in the USA). Measurement of ETCO<sub>2</sub> was taken every 1 min for three readings. Within 3 min of sampling time, the patient had an arterial blood gas sampling. This was analyzed immediately with the Radiometer® ABL 700 series available in the ED, which was calibrated daily by same technologist working in the department.

A data collection sheet was used to record the patient's age, sex, race, arterial blood gas result, ETCO<sub>2</sub>, all the vital signs, diagnosis and treatment. Patients were grouped as having primary acidosis, alkalosis or being normal using an acid-base normogram, or as hypocapnic if the PaCO<sub>2</sub> was less than 40 mmHg and hypercapnic if the PaCO<sub>2</sub> was more than 40. Patients were also grouped according to pulmonary and non-pulmonary pathology. Correlation between PaCO<sub>2</sub> and ETCO<sub>2</sub> was analyzed using a bivariate Pearson correlation coefficient. The difference in PaCO<sub>2</sub> and ETCO<sub>2</sub> between the groups was analyzed by independent t-test.

**Table 2** Investigations carried out on patients

Investigations	Number of patients	Percentage (%)
Arterial blood gas (ABG)	150	100
Electrocardiogram(ECG)	148	98.7
Blood tests	135	90
Chest radiograph(CXR)	146	97
Abdominal ultrasound	18	12
Echocardiography(ECHO)	58	39
Abdominal radiograph(AXR)	13	8.7
Urine analysis(UFEME)	34	23

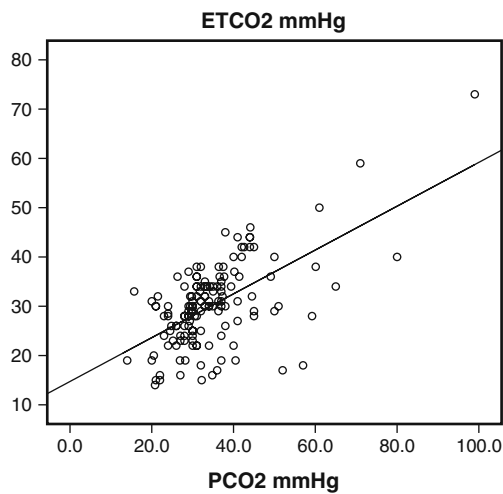
## Results

In this study, 165 acutely breathless patients were seen in three triage areas in the ED, namely red, yellow and green. The red zone signifies critical cases, yellow signifies semi-critical and green non-critical. Fifteen patients were excluded because of incomplete data collection. Data from 150 patients were submitted for analysis. The patients' demography and initial vital signs are shown as in Table 1.

In this study group, 99 patients (60%) had one or more existing pre-morbid conditions. In our sample study with pre-morbid conditions, 30% were patients with heart disease, 28% had hypertension, 23% diabetes and 18% bronchial asthma. The distribution of all investigations carried out on the patients is shown in Table 2. Arterial blood gas sampling was carried out in all of the patients. ABG was done on all patients, and of these, 96 (64%) had normal acid-base status, 26 (17.3%) had acidosis and 28 (18.7%) alkalosis. The most common site for ABG was the radial artery (127; 84.7%).

In our study, the highest number of breathless patients had acute coronary syndrome (n=38; 25.3%). Other causes included acute heart failure (20 cases; 13.3%), pneumonia (21; 14%), bronchial asthma (19; 12.7%) and stroke (16; 10.7%). Sepsis was identified in 9 patients (6%), chest trauma in 6 (4%) and diabetic ketoacidosis in 4 (2.7%). Other breathless patients in this study had epilepsy, poisoning and hematological disorders (7 cases; 4.7%). After treatment in the ED, 9 (6%) patients were discharged home, and 141 (94%) had to be admitted. The mean duration of stay was 2.95 h (95% CI; 1.87–3.25 h).

A correlation between ETCO<sub>2</sub> and PaCO<sub>2</sub> was tested using a bivariate Pearson correlation. Results showed a strong linear correlation between ETCO<sub>2</sub> and PaCO<sub>2</sub> with a Pearson correlation coefficient of 0.716 and two-tailed P-value of 0.00. Figure 1 shows a positive correlation between the ETCO<sub>2</sub> and PaCO<sub>2</sub> of non-intubated acute dyspneic patients. However, the paired t-test measuring mean differences between ETCO<sub>2</sub> and PaCO<sub>2</sub> among all patients showed a mean difference of 4.303 (p<0.05) with a



**Fig. 1** Linear correlation curve of  $\text{ETCO}_2$  and  $\text{PCO}_2$  of the whole group

95% CI 2.818, 5.789. The outcome of correlation analysis between the  $\text{ETCO}_2$  and  $\text{P}_a\text{CO}_2$  for various other conditions is as shown in Table 3. Positive correlation was only seen for hypocapnic and non-pulmonary causes of breathlessness. Other conditions, such as pulmonary disorders and hypercapnic state, showed negative correlation.

## Discussion

Patients in respiratory distress presenting to the ED require close assessment of their oxygenation, ventilation and acid-base balance. In this study, capnometry, a non-invasive tool that can measure  $\text{ETCO}_2$ , was used in acutely breathless patients to monitor and estimate arterial  $\text{P}_a\text{CO}_2$ , another important index for evaluating ventilation. The majority of acutely breathless patients were elderly: 60–69 years old (44%). This trend is due to the accumulation of chronic pathologies as patients age. Consequently, multiple pathologies resulting in multiple

symptoms are common phenomena in the elderly. A number of chronic conditions often occur simultaneously at different levels of severity. Thus, an accumulation of long-term non-fatal diseases is common in the elderly, many of which are degenerative in nature. In addition, antibiotics and new medical technologies provide the means to prevent premature death caused by diseases in the middle-aged and young old. This has resulted in the survival of much larger populations of patients, even at older ages [20]. In this study, 60% of our breathless patients had at least one of the premorbid conditions as stated above. The other 34% of our sample population did not have any premorbid conditions.

Studies have proven that premorbid conditions have some influence on the outcome of arterial  $\text{PCO}_2$ , especially in patients with impaired gas exchange or pulmonary disorders. For example, patients with chronic obstructive airway disease (COAD) will show a decrease in  $\text{ETCO}_2$  and an increase in the  $\text{P}_a\text{CO}_2/\text{ETCO}_2$  gradient because of physiological dead space [21]. In this study, similarity was found in certain premorbid conditions such as asthma and COAD where there was a weak correlation between  $\text{P}_a\text{CO}_2$  and  $\text{ETCO}_2$ , with a Pearson correlation coefficient of 0.152 and p value of 0.03 ( $p < 0.05$ ). This is in accordance with the findings in a study done by Corbo et al. showing that there was a positive correlation between  $\text{ETCO}_2$  and  $\text{PCO}_2$  in adult asthmatic patients. The mean difference between the  $\text{ETCO}_2$  and  $\text{PCO}_2$  level was 1 mmHg (95% confidence interval -0.1 to 2.0 mmHg) [22]. In a state of primary acidosis, many metabolic derangements occur. Acidemia has numerous negative physiologic consequences that impair the function of many different organs and body systems. In a study done by Barton et al., there was a strong correlation between an acidotic state and  $\text{ETCO}_2$  with regression analysis of  $r^2 = 0.889$  and a p value of 0.005 ( $p < 0.05$ ). Our study also showed similar findings with a good correlation with a Pearson correlation coefficient of 0.374 and p value of 0.02 [23].

**Table 3** The correlation between  $\text{ETCO}_2$  and  $\text{P}_a\text{CO}_2$  in various conditions

	N	$\text{PCO}_2$	$\text{ETCO}_2$	Pearson's correlation	P	r <sup>2</sup>
All	150	34.3(95% CI; 32.5, 37.9)	29.9(95% CI; 26.7, 33.5)	0.716	0.000	0.512
Acidotic	25	40.6(95% CI; 37.5, 45.3)	29.5(95% CI; 25.6, 32.4)	0.374	0.02	0.613
Alkalosis	28	30.2(95% CI; 28.9, 33.6)	27.9(95% CI; 24.8, 29.4)	0.171	0.037	0.029
Hypocapnia	118	29.9(95% CI; 26.4, 31.7)	28.1(95% CI; 25.8, 33.7)	0.738	0.000	0.544
Hypercapnia	32	50.3(95% CI; 48.1, 53.2)	36.8(95% CI; 34.5, 38.7)	-0.738	0.000	0.544
Pulmonary	43	40.3(95% CI 36.5, 43.2)	31.0(95% CI; 28.6, 33.4)	-0.336	0.000	0.113
Non-pulmonary	107	31.8(95% CI 27.4, 34.2)	29.5(95% CI; 26.7, 32.6)	0.336	0.000	0.113
Temperature (febrile)	29	32.6(95% CI; 30.1, 35.7)	30.4(95% CI; 27.2, 34.5)	0.074	0.370	0.005



There was also good correlation between  $\text{ETCO}_2$  and hypocapnia with a Pearson correlation coefficient of 0.738 ( $p < 0.05$ ). Fischer et al. found that the capnometer may not be able to detect changes in end tidal carbon dioxide at higher respiratory rates because low alveolar carbon dioxide in these patients was associated with an increase in airway tone, which is supported by an in vitro animal study that suggested that low levels of  $\text{P}_a\text{CO}_2$  cause bronchoconstriction [24, 25]. We also found that there was a negative correlation between  $\text{ETCO}_2$  and  $\text{PCO}_2$  with a Pearson correlation of 0.738 and  $p$  value of 0 ( $p < 0.05$ ). Normally hypercapnia is associated with hypoventilation. Surprisingly, previous study showed that there is strong correlation between hypercapnia and  $\text{ETCO}_2$ . This could be due to the presence of some abnormality of the ventilation perfusion ratio because of the primary pulmonary pathology resulting in hypercapnia. This subsequently led to a widened arterial to alveolar difference for  $\text{CO}_2$ . This has been confirmed by Fletcher, who showed a proportional increase in the end tidal carbon dioxide to  $\text{PaCO}_2$  difference as dead space was increased [26].

We also looked into the relationship between  $\text{ETCO}_2$  and  $\text{P}_a\text{CO}_2$  recorded in patients who have primary pulmonary disorders as well as without pulmonary disorders. Normally patients with pulmonary disorders will have some abnormalities in the ventilation perfusion ratio because of an impaired gas exchange mechanism [27]. We found that there was no correlation between  $\text{ETCO}_2$  and  $\text{PCO}_2$  in patients with pulmonary disorders, with a  $p$  value of 0.00. There was also a weak correlation between  $\text{ETCO}_2$  and  $\text{PCO}_2$  in patients without primary pulmonary disorders.

There were a few limitations of the study:

1. Categorizing patients into pulmonary and non-pulmonary disorders or other disorders is difficult. Most of the patients in this study group had a wide variety of underlying medical problems, and some of them had mixed disorders, making generalization difficult. We were unsure how significantly these different underlying pathologies would affect the  $\text{ETCO}_2$  reading in the study group.
2. This was a single center study, which might not represent the whole population.
3. There was no normogram of healthy people's  $\text{ETCO}_2$ s as a comparison or reference for the study group.

In the future we will continue similar studies with larger sample recruitment, and an attempt will be made to distinctly compare different lung pathologies or other medical conditions, even though this is very difficult to achieve. Further studies should be performed to determine a normogram of healthy people's  $\text{ETCO}_2$ s and to examine the influence of different parameters on the curve of the whole group and normal population specifically.

## Conclusion

Capnometry can be a potential form of non-invasive cardiopulmonary monitoring in non-intubated acutely breathless patients. However, the use of  $\text{ETCO}_2$  to predict  $\text{PaCO}_2$  should be done with caution, especially in cases that involve pulmonary disorders and acid-base imbalance.

**Conflicts of interest** None.

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