The use of capnometry to predict arterial partial pressure of CO₂ in non-intubated breathless patients in the emergency department

Nik Hisamuddin Nik Ab Rahman · Amiruddin Fairuz Mamat

Received: 16 July 2009 / Accepted: 26 August 2010 / Published online: 9 December 2010 © The Author(s) 2010. This article is published with open access at Springerlink.com

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 CO₂ and PaCO₂ in non-intubated acutely breathless patients.
2. To determine factors that influence the end tidal carbon dioxide (ETCO₂).
3. To determine the correlation between ETCO₂ with PaCO₂ 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 ETCO₂ 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 ETCO₂ and PaCO₂ 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 ETCO₂ and PaCO₂ were analyzed using paired sample t-tests.

Results There is a strong correlation between ETCO₂ and PaCO₂ 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 ETCO₂ 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 ETCO₂ and a hypocapnic state, with a Pearson correlation coefficient of -0.738 (p<0.05) and of -0.336 (p<0.05), respectively. A strong negative correlation was present between ETCO₂ 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 ETCO₂ can be used to predict the PaCO₂ level when the difference between the PaCO₂ and ETCO₂ is between 2 to 6 mmHg, especially in cases of pure acidosis and hypocapnia. Using ETCO₂ to predict PaCO₂ should be done with caution, especially in cases that involve pulmonary disorders and acid-base imbalance.

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 dyspnic 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 (PaCO2). Carbon dioxide (CO2) 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 CO2 (ETCO2). 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 CO2 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 CO2 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 CO2 and PaCO2 in non-intubated acutely breathless patients.
2. To determine factors that influence the end tidal carbon dioxide (ETCO2).
3. To determine the correlation between ETCO2 and PaCO2 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 ETCO2 in the exhaled air was measured using microstream Oridion Smart CapnoLine® CO2 sampling, which was connected to a monitoring system to display an
accurate waveform and numerical value of ETCO₂. The Oridion Smart Capnoline® uses a special canulla that is capable of measuring exhaled CO₂ 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 ETCO₂ reading was obtained using a capnometer built into a Datascope® cardiac monitor (manufactured in the USA). Measurement of ETCO₂ 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 the same technologist working in the department.

A data collection sheet was used to record the patient’s age, sex, race, arterial blood gas result, ETCO₂, 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 PaCO₂ was less than 40 mmHg and hypercapnic if the PaCO₂ was more than 40. Patients were also grouped according to pulmonary and non-pulmonary pathology. Correlation between PₐCO₂ and ETCO₂ was analyzed using a bivariate Pearson correlation coefficient. The difference in PaCO₂ and ETCO₂ between the groups was analyzed by independent t-test.

### 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 ETCO₂ and PₐCO₂ was tested using a bivariate Pearson correlation. Results showed a strong linear correlation between ETCO₂ and PₐCO₂ with a Pearson correlation coefficient of 0.716 and two-tailed P-value of 0.00. Figure 1 shows a positive correlation between the ETCO₂ and PₐCO₂ of non-intubated acute dyspneic patients. However, the paired t-test measuring mean differences between ETCO₂ and PₐCO₂ among all patients showed a mean difference of 4.303 (p<0.05) with a

### 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) |

### 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             |
95% CI 2.818, 5.789. The outcome of correlation analysis between the ETCO\textsubscript{2} and P\textsubscript{a}CO\textsubscript{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.

**Table 3** The correlation between ETCO\textsubscript{2} and P\textsubscript{a}CO\textsubscript{2} in various conditions

|          | N   | P\textsubscript{CO\textsubscript{2}} | ETCO\textsubscript{2} | Pearson’s correlation | P   | r^2  |
|----------|-----|------------------------------------|-----------------------|-----------------------|-----|------|
| 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 |

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 ETCO\textsubscript{2}, was used in acutely breathless patients to monitor and estimate arterial P\textsubscript{a}CO\textsubscript{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 PCO\textsubscript{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 ETCO\textsubscript{2} and an increase in the P\textsubscript{a}CO\textsubscript{2}/ETCO\textsubscript{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 P\textsubscript{a}CO\textsubscript{2} and ETCO\textsubscript{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 ETCO\textsubscript{2} and PCO\textsubscript{2} in adult asthmatic patients. The mean difference between the ETCO\textsubscript{2} and PCO\textsubscript{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 ETCO\textsubscript{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].
There was also good correlation between ETCO₂ 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 PaCO₂ cause bronchoconstriction [24, 25]. We also found that there was a negative correlation between ETCO₂ and PCO₂ 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 ETCO₂. 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 CO₂. This has been confirmed by Fletcher, who showed a proportional increase in the end tidal carbon dioxide to PaCO₂ difference as dead space was increased [26].

We also looked into the relationship between ETCO₂ and PaCO₂ 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 ETCO₂ and PCO₂ in patients with pulmonary disorders, with a p value of 0.00. There was also a weak correlation between ETCO₂ and PCO₂ 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 ETCO₂ 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 ETCO₂ 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 ETCO₂ 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 ETCO₂ to predict PaCO₂ should be done with caution, especially in cases that involve pulmonary disorders and acid-base imbalance.

**Conflicts of interest** None.

**Open Access** This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited.

**References**

1. Trochtenberg DS, Belue R (2007) Descriptors and perception of dyspnea in African-American asthmatics. J Asthma 44(10):811–815
2. Waggner D, Stokes J, Romero F, Casale TB (2008) Dyspnea and obesity in African American women. Ann Allergy Asthma Immunol 101(6):644–645
3. Aliverti A et al (2002) Respiratory muscle dynamics and control during exercise with externally imposed expiratory flow limitation. J Appl Physiol 92(5):1953–1963
4. Mahler DA, Harver A, Lentine T, Scott JA, Beck K, Schwartzstein RM (1996) Descriptors of breathlessness in cardiorespiratory diseases. Am J Respir Crit Care Med 154(5):1357–1363
5. Skevington SM, Pilaar M, Routh D (1997) On the language of breathlessness. Psychol Health 12:677–679
6. Cuervo Pinna MA, Mota Vargas R, Redondo Moralo MJ, Sánchez Correas MA, Pera Blanco G (2009) Dyspnea—a bad prognosis symptom at the end of life. Am J Hosp Palliat Care 26(2):89–97
7. Huijnen B et al (2004) Dyspnea in elderly family practice patients. Occurrence, severity, quality of life and mortality over an 8-year period. Fam Pract 21(1):34–39
8. Ander DS, Aisiku IP, Ratcliff JJ, Todd KH, Gotsch K (2004) Measuring the dyspnea of decompensated heart failure with a visual analog scale: how much improvement is meaningful. Congest Heart Fail 10(4):188–191
9. Knudsen CW et al (2004) Diagnostic value of B-type natriuretic peptide and chest radiographic findings in patients with acute dyspnea. Am J Med 116(6):363–368
10. Malas O, Çağlayan B, Fidan A, Ocal Z, Ozdoğan S, Torun E (2003) Cardiac or pulmonary dyspnea in patients admitted to the emergency department. Respir Med 97(12):1277–1281
11. Weil MH, Sun S (2001) Tissue capnometry. Crit Care Med 29:460
12. Niehoff J et al (1988) Efficacy of pulse oximetry and capnometry in postoperative ventilatory weaning. Crit Care Med 16(7):701–705
13. Turner KE, Sandler A, Vosu H (1989) End tidal CO₂ monitoring in spontaneously breathing adults. Can J Anaesth 36:248–249
14. Raemer D, Francis D, Philip J (1983) Variation in PCO₂ between arterial blood and peak expired gas during anesthesia. Anaesth Analg 62:1065–1069
15. Callahan M, Barton C (1990) Prediction outcome of cardiopulmonary resuscitation from end tidal carbon dioxide concentration. Crit Care 18:358–362
16. Liu S, Lee T, Bongard F (1992) Accuracy of capnography in non intubated surgical patients. Chest 102:1512–1515
17. Wiklund L, Sorderberg S, Hennerberg S (1986) Kinetics of carbon dioxide during cardiopulmonary resuscitation. Crit Care Med 14 (12):1015–1022
18. Smallhout B (1983) A quick guide to capnography and its use in differential diagnosis. Acta Anaesthesiol Scand 27:199-202
19. Casati A, Gallioli G (2000) Accuracy of end tidal carbon dioxide monitoring using the NBP-75 microstream capnometers. A study in intubated ventilated and spontaneously breathing non intubated patients. Eur J Anaesthesiol 17(10):622–666
20. WHO Study on global ageing and adult health (SAGE): http://www.who.int/healthinfo/systems/sage/en/index.html
21. Wabha WM (1983) Influence of aging on lung function–clinical significance of changes from age 20. Anaesth Analg 62:764–776
22. Corbo J, Bijur P, Lahn M, Gallagher EJ (2005) Concordance between capnography and arterial blood gas measurements of carbon dioxide in acute asthma. Ann Emerg Med 46(4):323–327
23. Barton CW, Wang ESJ (1994) Correlation of end tidal CO2 measurements to arterial PaCO2 in non intubated patients. Ann Emerg Med 23:145
24. Fischer AR, Rosenberg MA, Roth M, Loper M, Jungerwirth S, Israel E (1997) Effect of a novel 5-lipoxygenase activating protein inhibitor, BAYx 1005, on asthma induced by cold dry air. Thorax 52(12):1074–1077
25. Reynolds AM, McEvoy E (1989) Tachykinins mediated hypocapnia induced bronchoconstriction in guinea pig. J Appl Physiol 67(6):2453–2467
26. Fletcher R (1989) Relationship between alveolar deadspace and arterial oxygenation in children with congenital cardiac disease. Br J Anaesth 62:168–176
27. Liu Z, Vargas F, Stansbury D, Sasse SA, Light RW (1996) Comparison of the end-tidal arterial PCO2 gradient during exercise in normal subjects and in patients with severe COPD. Chest 110(3):865–866

Dr. Nik Hisamuddin Nik Ab. Rahman is a consultant and Associate Professor of Emergency Medicine in the School of Medical Sciences, University Sains Malaysia. He obtained a MBChB from the University of Glasgow and pursued postgraduate training in the field of Emergency Medicine in Malaysia. In 2002, he was one of the first Emergency Medicine Specialists to become certified in a fully accredited local residency training program. He also completed clinical fellow training at the Royal Infirmary of Edinburgh, Scotland in 2006. He has special interests in the field of trauma and injury prevention, resuscitation, pre-hospital care and hyperbaric medicine. He is also the Chairman of the Malaysian Conjoint Board of Emergency Medicine. His hobbies include traveling, golfing and gardening.