

ORIGINAL ARTICLE

End tidal carbon dioxide as a predictor of the arterial PCO_2 in the emergency department setting

C Yosefy, E Hay, Y Nasri, E Magen, L Reisin

Emerg Med J 2004;21:557–559. doi: 10.1136/emj.2003.005819

See end of article for authors' affiliations

Correspondence to: Dr C Yosefy, Department of Cardiology, Barzilai Medical Center, Ashkelon, 78306, Israel; yosefy@barzi.health.gov.il

Objectives: Patients arriving in the emergency department (ED) need rapid and reliable evaluation of their respiratory status. Mainstream end tidal carbon dioxide (ETCO_2) is one of the methods used for this purpose during general anaesthesia of intubated patients in the operating theatre. Sidestream ETCO_2 (SSETCO₂) might be a non-invasive, rapid, and reliable predictor of arterial PCO_2 in non-intubated patients in respiratory distress. The aim of this study was to verify whether SSETCO₂ can accurately predict the arterial PCO_2 and to detect variables that may affect this correlation.

Methods: A prospective semi-blind study. The participants were 73 patients (47 men, 26 women) referred to the ED for respiratory distress. Arterial blood gas pressures and SSETCO₂ measurements were performed and recorded for all patients. Other parameters recorded were: age; body temperature; respiratory rate; blood pressure; pulse rate; and medical diagnosis.

Results: A significant correlation was found between SSETCO₂ and arterial PCO_2 ($r=0.792$). Compared with the correlation curve of the whole group, age under 50 years deflected the correlation curve to the left, while temperature above 37.6°C deflected it to the right. The rest of the parameters had no clear influence on the SSETCO₂/ PCO_2 correlation curve.

Conclusions: There is a good correlation between SSETCO₂ and arterial PCO_2 in the ED setting. Young age may increase the arterial PCO_2 /SSETCO₂ gradient while raised temperature may decrease this gradient. Further studies are needed to confirm these findings in the normal healthy population.

Patients in respiratory distress presenting in the emergency department (ED) require close assessment of their oxygenation, ventilation, and acid base balance. Arterial blood gases (ABG) examination provides accurate information, but an arterial blood analyser 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. Alternative methods for immediate and continuous non-invasive monitoring include pulse oximetry and capnography (end tidal CO_2 (ETCO_2)).¹

In the ETCO_2 capnography the gas to be analysed reaches the sample chamber in one of two ways²: a mainstream analyser that resides within the breathing circuit, usually between the end of the endotracheal tube and the Y connection. This method is usually used for infrared spectrometers. Because the sample chamber is part of the breathing circuit, the delay time is minimised and there are no problems with increased work of breathing or clogging by pulmonary secretions. However, the analyser is heavy, and may cause kinking or dislodgement of the endotracheal tube. The second method is using a sidestream analyser, in which the gas sample is aspirated from the breathing circuit through a small bore tube to a remote analyser. This method is mainly used for mass spectrometry and some infrared analysers. It adds little weight to the breathing circuit but the narrow lumen of the sampling tube is more likely to be clogged by pulmonary secretions and the delay time is much longer than the mainstream method. In addition, there may be some loss of the expired tidal volume caused by continuous sampling.

Both mainstream and sidestream ETCO_2 have been found to be closely correlated to arterial PCO_2 , especially in haemodynamically stable patients.^{3–4} ETCO_2 monitoring is in daily use in general anaesthesia in adults,^{5–6} in anaesthetised and non-intubated children,^{7–9} and in non-intubated

adult patients.^{10–11} Sidestream ETCO_2 (SSETCO₂) was found to be quite a sensitive mean for identification of CO_2 pulmonary depression in children under conscious sedation, undergoing painful procedures, rather than the PO_2 , which falls only later.⁹ ETCO_2 has also been used as a measure of pulmonary circulation, especially in circulatory shock, where it may have a prognostic value,^{12–15} and in massive pulmonary embolism.¹⁶ During endotracheal intubation, low values of ETCO_2 can indicate false location of the tube.^{17–18}

SSETCO₂ is very suitable in the ED setting for patients in respiratory distress. It is immediate, non-invasive, and does not require cooperation of the patient. Sidestream capnography provides numerical values of the ETCO_2 , ETCO_2 graph, and ETCO_2 trend. The last provides important information on circulatory status and ventilation.^{15–16–19–20} The numerical value of the SSETCO₂ can be of great importance for the immediate evaluation of patients in severe respiratory distress, who are potentially CO_2 retainers.²¹

To verify whether SSETCO₂ can accurately predict the arterial PCO_2 and to detect variables that can affect this correlation, we conducted the following prospective semi-blind study in our ED.

METHODS

Patients who were referred to our ED because of respiratory distress and needed ABG were included in the study. The study was conducted during the morning and evening shifts, between January and June 2000. ABG was performed before administration of supplemental oxygen. Simultaneously, SSETCO₂ was measured by a sidestream capnometer (OHMEDA Model 4700 Oxycap monitor, a division of the BOC Group, Colorado, USA) using its standard nasal cannula

Abbreviations: ED, emergency department; ETCO_2 , end tidal carbon dioxide; SSETCO₂, sidestream end tidal carbon dioxide; ABG, arterial blood gases

for ETCO_2 measurement. The highest reading was recorded because it best represents a full tidal volume. Immediately afterwards, oxygen was delivered according to the patient's state. The following initial parameters were recorded: age; body temperature; respiratory rate; blood pressure; pulse rate; and medical diagnosis. Exclusion criteria were: age under 18 years; any aetiology of shock and need for immediate resuscitation or intubation. The investigator measuring the ABG was totally blinded to the SSETCO₂ results. The study was approved by the local hospital ethics committee and all the patients signed a form of informed consent.

Statistical analysis

SSETCO₂/PCO₂ correlation curve was plotted and tested using the single variable analysis method. The influence of the other variables was tested using the multivariate linear regression analysis method.

RESULTS

Seventy three patients were enrolled in the study, 47 men and 26 women. The mean (SD) age was 64.95 (18.97) years, range 18–95. SSETCO₂ ranged from 12–74 mm Hg. The medical diagnosis included 55 patients with pulmonary oedema (75.3%), 14 patients with exacerbation of chronic obstructive lung disease (19.2%), and four patients with exacerbation of bronchial asthma (5.5%). Table 1 lists the patients' characteristics.

The correlation between SSETCO₂ and PCO₂ was found to be linear (fig 1), with a correlation coefficient of 0.792. Age under 50 years caused the curve to shift to the left compared with the linear correlation curve of the whole group, and temperature above 37.6°C resulted in a shift to the right (figs 2 and 3). We did not find an influence of blood pressure, respiratory rate, or blood pH on the SSETCO₂/CO₂ curve, however, we did note a weak trend of a shift to the right with pH below 7.35 and diastolic blood pressure above 90 mm Hg. These trends were not statistically significant. We had the impression of a weak correlation (0.620) between the ETCO₂ and the PCO₂ with high respiratory rates (above 30/min).

DISCUSSION

Our results clearly show a good correlation between the arterial PCO₂ and the SSETCO₂. The mismatch between ETCO₂ and arterial PCO₂ reflects the discrepancy between the perfused and the ventilated alveoli. An increase in anatomical and physiological dead space and disturbances in pulmonary circulation, decreases the ETCO₂ and increases the PCO₂/ETCO₂ gradient.^{3–4, 10} A gradient of 5–6 is considered normal in haemodynamically stable patients.^{3, 4, 24} Pulmonary embolism and circulatory shock decrease the ETCO₂ level and increase the PCO₂/ETCO₂ gradient.^{3, 15, 16} Aging has been shown to increase the PCO₂/ETCO₂ gradient, probably by

Table 1 Characteristics of study population

Parameter	Result
Patients (n)	73
Male, number (%)	47 (64.4)
Female, number (%)	26 (35.6)
Age (y)	64.95 (18.97)
SBP (mm Hg)	126 (4.2)
DBP (mm Hg)	78 (4.6)
Heart rate (beat/min)	76 (3.6)
Respiratory rate (per minute)	14 (2.1)
Temperature (°C)	37.2 (1.3)

Data in parentheses are SD unless stated otherwise. SBP, systolic blood pressure; DBP, diastolic blood pressure.

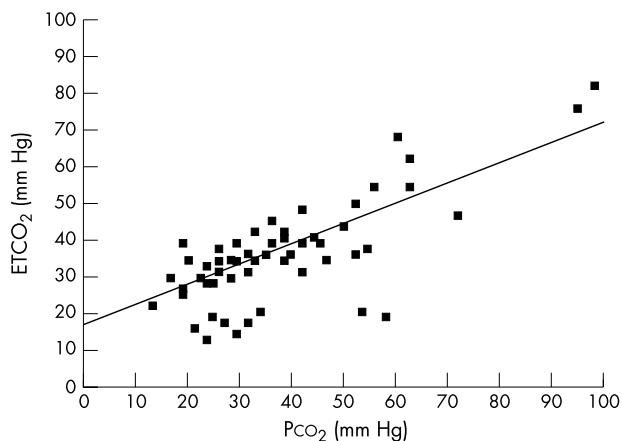


Figure 1 Linear correlation curve of ETCO₂ and PCO₂ of the whole group.

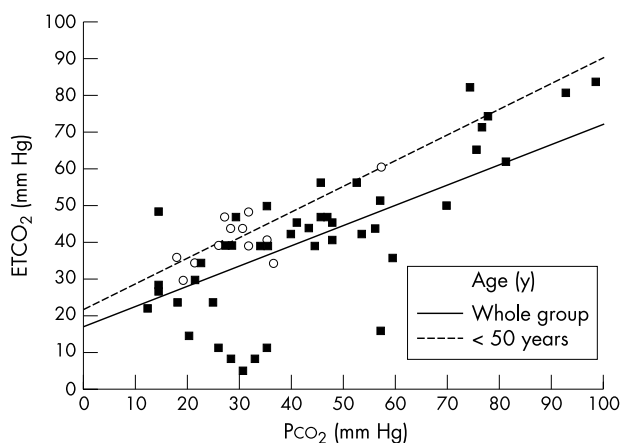


Figure 2 ETCO₂/PCO₂ correlation curve for patients under age 50 compared with the whole group.

increasing the anatomical dead space.²⁵ In another study of 314 patients, ETCO₂ decreased with age only in men.²⁶ In our younger group of patients (under 50 years), we recorded lower values of PCO₂, and thus lower values of ETCO₂,

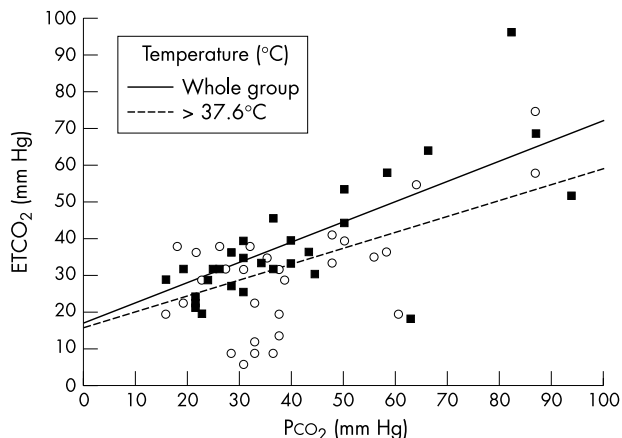


Figure 3 ETCO₂/PCO₂ correlation curve for patients with temperature above 37.6°C compared with the whole group.

resulting in an increase in the Pco₂/ETCO₂ gradient and a shift to the left of the curve. This may be explained by the higher frequency of asthma in the younger group than in the older one.

Increasing the temperature tends to decrease the Pco₂/ETCO₂ gradient. This can be explained by the fact that the blood analyser is set to 37°C and the solubility of CO₂ is increased with cooling.⁴ This causes the curve to shift to the right, exactly as we have found in our study. The probable effect of increased diastolic blood pressure on the ETCO₂/Pco₂ correlation curve in our study may be explained by the fact that most of the patients had pulmonary oedema, implying an increase in the dead space. In relation to the respiratory rate, From and Scamman state that the capnometer could not accurately predict changes in the ETCO₂ with high respiratory rates,²⁷ thus resulting in a low correlation coefficient. In our study we also had the impression that the correlation between the SSETCO₂ and the Pco₂ was weak with respiratory rate over 30/minute, but it did not reach statistical significance, probably because of the small number of patients.

We did not divide the patients into subgroups according to the level of arterial Pco₂ and the different pathogenesis. The subgroups would be too small to be compared and analysed statistically.

Our study has limitations. Firstly, the small number of patients. Secondly, the comparison of the influence of the different parameters on the curve of the whole group instead of a comparison with a nomogram in healthy controls. Therefore, further studies should be performed to determine a nomogram of healthy people and to examine the influence of the various parameters on this nomogram. Finally, the positioning of the sampling tube (mouth breathing) might have had some effect on the ETCO₂ estimate.

In summary, we found a good correlation between SSETCO₂ and arterial Pco₂ in the ED setting. Young age may increase the arterial Pco₂/SSETCO₂ gradient while raised temperature may decrease this gradient. Further studies are needed to confirm these findings in the normal healthy population. We recommend the use of this non-invasive method in other EDs.

CONTRIBUTORS

Chaim Yosefy: principal investigator and writer. Emile Hay: investigator and co-writer. Yusuf Nasri: investigator and data collector. Eliyahu Magen: investigator and data collector. Leonardo Reisin: investigator, co-writer, and statistical analyser.

Authors' affiliations

C Yosefy, L Reisin, Department of Cardiology, Barzilai Medical Center, Ashkelon, Israel
E Hay, Department of Emergency Medicine, Barzilai Medical Center
Y Nasri, Department of Primary Medicine, Ben-Gurion University of the Negev, Israel
E Magen, WHO Collaborative Center for Prevention of Cardiovascular Diseases, Barzilai Medical Center

Funding: this study did not receive any funding from sources other than the Barzilai Medical Center.

Conflicts of interest: none declared.

REFERENCES

- Soubany AO**. Noninvasive monitoring of oxygen and carbon dioxide. *Am J Emerg Med* 2001;**19**:141–6.
- Block FE Jr, McDonald JS**. Sidestream versus mainstream carbon dioxide analyzers. *J Clin Monit* 1992;**8**:139–41.
- Whitesell R, Asiddao C, Gollman D, et al**. Relationship between arterial and peak expired carbon dioxide pressure during anesthesia and factors influencing the difference. *Anesth Analg* 1981;**60**:508–12.
- Wahba RW, Tessler MJ**. Misleading end-tidal CO₂ tensions. *Can J Anaesth* 1996;**43**:862–6.
- Takki S, Aromaa U, Kausta A**. The validity and usefulness of end-tidal pCO₂ during anaesthesia. *Ann Clin Res* 1972;**4**:278–84.
- Bhuvani-Shankar K, Moseley H, Kumar AY, et al**. Capnometry and anaesthesia. *Can J Anaesth* 1992;**39**:617–32.
- Tabias JD, Flanagan JF, Wheeler TJ, et al**. Noninvasive monitoring of end-tidal CO₂ via nasal cannulas in spontaneously breathing children during the perioperative period. *Crit Care Med* 1994;**22**:1805–8.
- Campbell FA, McLeod ME, Bissonnette B, et al**. End-tidal carbon dioxide measurement in infants and children during and after general anaesthesia. *Can J Anaesth* 1994;**41**:107–10.
- Hart LS, Berns SD, Houck CS, et al**. The value of end-tidal CO₂ monitoring when comparing three methods of conscious sedation for children undergoing painful procedures in the emergency department. *Pediatr Emerg Care* 1997;**13**:189–93.
- Stock MC**. Capnography for adults. *Crit Care Clin* 1995;**11**:219–32.
- Bowe EA, Boysen PG, Broome JA, et al**. Accurate determination of end-tidal carbon dioxide during administration of oxygen by nasal cannulae. *J Clin Monit* 1989;**5**:105–10.
- Gasman JD, Fishman RS, Raffin TA**. Monitoring cardiopulmonary resuscitation: role of blood and end-tidal carbon dioxide tension. *Crit Care Med* 1995;**23**:799–800.
- Idris AH, O'Brien DJ, Melker RJ, et al**. End-tidal carbon dioxide during extremely low cardiac output. *Ann Emerg Med* 1994;**23**:568–72.
- Nakagawa Y, Weil MH, Tang W, et al**. Sublingual capnometry for diagnosis and quantitation of circulatory shock. *Am J Respir Crit Care Med* 1998;**157**:1838–43.
- Gudipati CV, Weil MH, Bisera J, et al**. Expired carbon dioxide: a noninvasive monitor of cardiopulmonary resuscitation. *Circulation* 1988;**77**:234–9.
- Hatle L, Rokseth R**. The arterial to end-expiratory carbon dioxide tension gradient in acute pulmonary embolism and other cardiopulmonary diseases. *Chest* 1974;**66**:352–7.
- Linko K, Paloheimo M, Tammisto T**. Capnography for detection of accidental oesophageal intubation. *Acta Anaesthesiol Scand* 1983;**27**:199–202.
- Linko K, Paloheimo M**. Capnography facilitates blind nasotracheal intubation. *Acta Anaesthesiol Belg* 1983;**34**:117–22.
- Yaron M, Padyk P, Hutsiniller M, et al**. Utility of the expiratory capnogram in the assessment of bronchospasm. *Ann Emerg Med* 1996;**28**:403–7.
- Bergman NA**. Temperature coefficients for PCO₂ and PO₂ in bloods with varying acid-base status. *J Appl Physiol* 1968;**24**:225–8.
- Brown LH, Gough JE, Seim RH**. Can quantitative capnometry differentiate between cardiac and obstructive causes of respiratory distress? *Chest* 1998;**113**:323–6.
- Barton CW, Wang ES**. Correlation of end-tidal CO₂ measurement to arterial PaCO₂ in nonintubated patients. *Ann Emerg Med* 1994;**23**:560–3.
- Sanders AB**. Capnometry in emergency medicine. *Ann Emerg Med* 1989;**18**:1287–90.
- Russel GB, Graybeal JM, Strout JC**. Stability of arterial to end-tidal carbon dioxide gradients during postoperative cardiorespiratory support. *Can J Anaesth* 1990;**37**:560–6.
- Wahba WM**. Influence of aging on lung function—clinical significance of changes from age twenty. *Anesth Analg* 1983;**62**:764–76.
- Anderson DE, Parsons DJ, Scuteri A**. End tidal CO₂ is an independent determinant of systolic blood pressure in women. *J Hypertens* 1999;**17**:1073–80.
- From RP, Scamman FL**. Ventilatory frequency influences accuracy of end-tidal CO₂ measurements. Analysis of seven capnometers. *Anesth Analg* 1988;**67**:884–6.