End tidal carbon dioxide as a predictor of the arterial P\textsubscript{CO}_2 in the emergency department setting

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

**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 SSETCO\textsubscript{2} 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 SSETCO\textsubscript{2} and arterial P\textsubscript{CO}_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 SSETCO\textsubscript{2}/P\textsubscript{CO}_2 correlation curve.

**Conclusions:** There is a good correlation between SSETCO\textsubscript{2} and arterial P\textsubscript{CO}_2 in the ED setting. Young age may increase the arterial P\textsubscript{CO}_2/SSETCO\textsubscript{2} gradient while raised temperature may decrease this gradient. Further studies are needed to confirm these findings in the normal healthy population.
for ETCO₂ 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 multivariant 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. A gradient of 5–6 is considered normal in haemodynamically stable patients. Pulmonary embolism and circulatory shock decrease the ETCO₂ level and increase the PCO₂/ETCO₂ gradient. Aging has been shown to increase the PCO₂/ETCO₂ gradient, probably by increasing the anatomical dead space. In another study of 314 patients, ETCO₂ decreased with age only in men. In our younger group of patients (under 50 years), we recorded lower values of PCO₂, and thus lower values of ETCO₂,

### Table 1 Characteristics of study population

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

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

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REFERENCES