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 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$_2$)).

In the ETCO$_2$ capnography the gas to be analysed reaches the sample chamber in one of two ways: 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 P$_{CO2}$, especially in haemodynamically stable patients. ETCO$_2$ monitoring is in daily use in general anaesthesia in adults, in anaesthetised and non-intubated children, and in non-intubated adult patients. Sidestream ETCO$_2$ (SSETCO$_2$) 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 P$_{CO2}$, which falls only later. ETCO$_2$ has also been used as a measure of pulmonary circulation, especially in circulatory shock, where it may have a prognostic value, and in massive pulmonary embolism. During endotracheal intubation, low values of ETCO$_2$ can indicate false location of the tube.

ETCO$_2$ 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. The numerical value of the SSETCO$_2$ can be of great importance for the immediate evaluation of patients in severe respiratory distress, who are potentially CO$_2$ retainers.

To verify whether SSETCO$_2$ can accurately predict the arterial P$_{CO2}$ 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, SSETCO$_2$ was measured by a sidestream capnometer (OHMEDA Model 4700 Oxycap monitor, a division of the BOC Group, Colorado, USA) using its standard nasal cannula.
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 SSETCO$_2$ results. The study was approved by the local hospital ethics committee and all the patients signed a form of informed consent.

**Statistical analysis**

SSETCO$_2$/PCO$_2$ 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. SSETCO$_2$ 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 SSETCO$_2$ and PCO$_2$ 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 SSETCO$_2$/CO$_2$ 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 ETCO$_2$ and the PCO$_2$ with high respiratory rates (above 30/min).

**DISCUSSION**

Our results clearly show a good correlation between the arterial PCO$_2$ and the SSETCO$_2$. The mismatch between ETCO$_2$ and arterial PCO$_2$ 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 ETCO$_2$ and increases the PCO$_2$/ETCO$_2$ gradient.$^{34,51}$ A gradient of 5–6 is considered normal in haemodynamically stable patients.$^{3,4,10}$ Pulmonary embolism and circulatory shock decrease the ETCO$_2$ level and increase the PCO$_2$/ETCO$_2$ gradient.$^{3,15,16}$ Aging has been shown to increase the PCO$_2$/ETCO$_2$ gradient, probably by increasing the anatomical dead space.$^{25}$ In another study of 314 patients, ETCO$_2$ decreased with age only in men.$^{26}$ In our younger group of patients (under 50 years), we recorded lower values of PCO$_2$, and thus lower values of ETCO$_2$, and the PCO$_2$/ETCO$_2$ correlation curve for patients under age 50 compared with the whole group.

**Table 1** Characteristics of study population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Result</th>
</tr>
</thead>
<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 PaCO2/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 PaCO2/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/PaCO2 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 respiratory rate, From and Scamman state that the capnometer could not accurately predict changes in the ETCO2 with respiratory rate, 4 thus resulting in a low correlation coefficient. In our study we also had the impression that the correlation between the SSETCO2 and the PaCO2 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 PaCO2 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 PaCO2 in the ED setting. Young age may increase the arterial PaCO2/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.

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REFERENCES
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