Changes in oxygen saturation and transcutaneous carbon dioxide and oxygen levels in patients undergoing fibreoptic bronchoscopy

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Patients undergoing bronchoscopy are usually monitored with pulse oximetry to measure arterial oxygen saturation, but this can fail to detect hypoventilation, particularly if added inspired oxygen is used. Transcutaneous oxygen and carbon dioxide tensions can be measured, the later reflecting respiratory drive. We compared transcutaneous \( PO_2 \) and \( PCO_2 \) values with oxygen saturation in patients undergoing day-case bronchoscopy, to see if this information would further improve the safety of the bronchoscopic procedure.

Twenty-two consecutive patients undergoing routine fibreoptic bronchoscopy (15 male, mean age 62.3 years; range 45–82 years), were studied using pulse oximetry (OXimeter, Radiometer) and transcutaneous \( PCO_2/PO_2 \) monitoring (TCM3, TINA, Radiometer).

We documented a statistically significant increase in transcutaneous \( PCO_2 \) from mean (sd) stable baseline levels of 5.8 (0.3) kPa (range 4.2–7.9 kPa) to mean peak levels during bronchoscopy of 7.0 (1.0) kPa (range 5.0–8.7 kPa). The time to first adverse change in transcutaneous \( PCO_2 \) \((P=0.046)\) and \( PO_2 \) \((P=0.035)\) occurred more rapidly than reduction in oxygen saturation in 19 of the 22 cases; median times for change in \( PCO_2 \) of 67 s (range 10–1800 s), \( PO_2 \) of 120 s (range 26–559 s) and oxygen saturation of 174 s (range 43–1332 s), timed from administration of i.v. sedation prior to each bronchoscopy.

Transcutaneous \( PCO_2/PO_2 \) monitoring during fibreoptic bronchoscopy provided evidence of hypoventilation with significantly elevated levels of transcutaneous \( PCO_2 \). This method of monitoring provides an earlier indication of respiratory depression during fibreoptic bronchoscopy compared with pulse oximetry.

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Introduction

Fibreoptic bronchoscopy (FOB) is a common invasive pulmonary investigation providing direct visualization of the bronchial tree as far as the subsegmental bronchi, allowing diagnostic samples to be taken. FOB is a safe procedure with a complication rate of approximately 0.1%. Exclusion criteria include central cyanosis at rest, breathlessness, poorly controlled angina and recent myocardial infarction. Patients with a low FEV\(_1\) (<1 l) can be safely examined but sedation is usually withheld.

Introduction of the fibreoptic bronchoscope into the airways results in partial airway obstruction and reductions in oxygen saturation, measured by pulse oximetry, are recognized in patients undergoing endoscopic procedures using benzodiazepines and narcotics for sedation (1–3). Patients are usually monitored by clinical observation and with pulse oximetry (monitoring adequacy of arterial oxygenation) and supplemental oxygen is given via nasal cannulae. Failure of pulse oximetry to detect intra- and post-operative hypoventilation is recognized, especially with a high inspired oxygen concentration (4,5) and marked hypoxaemia is sometimes difficult to detect clinically (6). Elevated values of \( PaCO_2 \) are a better guide to type II respiratory failure secondary to atelectic hypoventilation, which is the mechanism induced by the procedure of FOB and/or the sedation.

Transcutaneous \( PCO_2/PO_2 \) monitoring was introduced in the early 1980s using the principle that if an area of skin is warmed to 44°C dissolved gases diffuse through the skin. This technique is now widely used by anaesthetists in the operating theatre and on Intensive Care and Special Care Baby Units in the assessment of gas tensions in high-risk situations (7,8).

A literature search using Medline (key words: carbon dioxide, transcutaneous and bronchoscopy) was unable to find any previous studies on the use of transcutaneous \( PCO_2/PO_2 \) monitoring during FOB. We wished to assess if transcutaneous \( PCO_2/PO_2 \) monitoring provided any additional information during routine FOB in adults, as compared with pulse oximetry.

Methods

Twenty-two consecutive patients undergoing routine FOB (15 male, mean age 62.3 years; range 45–82 years) were
entered into the study. The patients were examined clinically and underwent routine chest X-ray, spirometry, arterial blood gas analysis and electrocardiogram (ECG), before being considered as suitable for the procedure. Spirometry demonstrated mean values of FEV1 (SD) of 61.8 (20.1)% and FVC 74.1 (23.1)% (results expressed as percentages of predicted values) with FEV1/FVC of 66.3 (13.0)%.

Monitoring of the patients using finger clip pulse oximetry (OXImeter, Radiometer) and transcutaneous PCO2/PO2 electrodes (TCM3, TINA, Radiometer) was performed for 1 h prior to FOB for a total of 4 h.

Atropine (mean dose 0.59 mg; range 0.30-0.60 mg) and Papaveratum (Omnopon®; mean dose 9.43 mg; range 2.50-10.0 mg) were administered intramuscularly 30 min before the FOB with the doses modified on the basis of the clinical condition of the patient and results of spirometry. Patients took their maintenance treatment 4 h prior to the FOB, which in four patients included the use of inhaled bronchodilators. Patients were administered oxygen at a rate of 2 l/min via nasal cannulae and the procedure was initiated when a stable trace was obtained in the values of oxygen saturation and transcutaneous PCO2/PO2. Alterations in supplemental oxygen were made according to clinical need and to maintain the oxygen saturation above 90%. Intravenous diazepam suspension (Diazemuls; diazepam 10 mg/2 ml), administered through an indwelling i.v. catheter, was given at the beginning of the procedure to induce light sedation and throughout the bronchoscopy to keep the patient comfortable (mean total dose 7.60 mg; range 2.50-20.0 mg). Topical anaesthesia of the oropharynx and pharynx was obtained by spraying with xylocaine (2%).

The fibreoptic bronchoscope (Olympus BF20D) was passed orally under direct vision with the patient supine. Laryngeal and endobronchial anaesthesia was obtained by placing lignocaine 2% (mean dose 17.1 ml; range 6.0-26.0 ml) topically through the bronchoscope. Bronchoscopic washings were performed by passing 20 ml aliquots of normal saline through the bronchoscope with biopsies taken from lesions seen in the major airways. During the FOBs patients were also monitored continuously with ECG.

Prior to each investigation a new electrode membrane and electrolyte solution was applied and the transcutaneous PCO2/PO2 monitors were calibrated against a standard concentration compressed gas (consisting of 5.0% carbon dioxide, 20.9% oxygen, 74.1% nitrogen), via the TCC3 Calibration Unit provided by Radiometer, as outlined by the manufacturer. The transcutaneous electrodes were attached to the patient by an adhesive ring, over the pectoral region, with the temperature set at 44°C. The area was shaved (if necessary), minimally abraded and cleaned with alcohol. The 90% response times for the electrode were 20 s, with continuous sampling.

The pulse oximeter's response time was 2 s with weighted average sampling every 6 s. Both the oximeter and the transcutaneous monitor printed their information using compact digital recorders by Radiometer, which provided real time recordings. The results of transcutaneous PCO2/PO2 were ‘blinded’ from the physician performing the FOB.

Ethical approval was provided by the local ethics committee and all patients gave written informed consent prior to the study.

### Results

All subjects underwent uncomplicated FOB without any serious side-effects, with no patients requiring reversal of their sedation. The median duration of each FOB was 703 s (range 250-885 s) with 20 patients undergoing bronchial washings, 10 in combination with biopsy. All subjects received supplemental oxygen at rates varying between 2–10 l/min via nasal cannulae.

**Table 1. Stable pre-bronchoscopy transcutaneous and arterial blood gas tensions**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TcPCO2</td>
<td>8.6 (2.5) kPa</td>
</tr>
<tr>
<td>Arterial PO2</td>
<td>10.7 (3-5) kPa</td>
</tr>
<tr>
<td>TcPO2</td>
<td>5.8 (0.8) kPa</td>
</tr>
<tr>
<td>Arterial PCO2</td>
<td>5.3 (0.6) kPa</td>
</tr>
<tr>
<td>SpO2</td>
<td>93.6 (3.0) %</td>
</tr>
<tr>
<td>SaO2</td>
<td>94.2 (3.0) %</td>
</tr>
</tbody>
</table>

Values expressed as means with standard deviations in parenthesis.


PO2 values were ‘blinded’ from the physician performing the FOB.

The mean peak value of transcutaneous PO2 was 7.0 (1.0) kPa with a mean difference in this peak compared with resting values of 1.3 (0.7) kPa. This difference is statistically significant with a P-value of <0.001 (Student's paired t-test; Table 3). It took a median time of 372 s (range 67–5940 s), for the transcutaneous PO2 to reach its peak and a median time of 951 s (range 266–4440 s) to return to baseline levels. The varying flow rates of inspired oxygen, given in response to clinical need and/or oxygen...
Changes in oxygen and carbon dioxide in fibreoptic bronchoscopy

Table 2. Timing of events for the 22 patients

<table>
<thead>
<tr>
<th>Event</th>
<th>Median time (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to first reduction, 2% from baseline, in $\text{SpO}_2$</td>
<td>174 (43-1332) s</td>
</tr>
<tr>
<td>Time to first reduction, 0.4 kPa from baseline, in $\text{TcPO}_2$</td>
<td>120 (26-560) s*</td>
</tr>
<tr>
<td>Time to first increase, 0.4 kPa from baseline, in $\text{TcPCO}_2$</td>
<td>67 (10-1800) s*</td>
</tr>
<tr>
<td>Time to maximum increase in $\text{TcPCO}_2$</td>
<td>372 (67-5940) s</td>
</tr>
<tr>
<td>Time for $\text{TcPCO}_2$ return to baseline</td>
<td>951 (266-4440) s</td>
</tr>
<tr>
<td>Time to introduction of bronchoscope into the trachea</td>
<td>185 (85-400) s</td>
</tr>
<tr>
<td>Duration of bronchoscopy</td>
<td>703 (250-885) s</td>
</tr>
</tbody>
</table>

*Statistically significant when compared with the time to the first reduction in $\text{SpO}_2$, using the independent samples t-test.

Time zero is taken as the time of administration of i.v. diazepam suspension.

Discussion

We found a significant increase in transcutaneous $\text{PCO}_2$ in patients undergoing FOB. This increase in transcutaneous $\text{PCO}_2$ occurred against a varying concentration of inspired oxygen, administered in response to reduced oxygen saturation and the patient’s clinical need. The increase in transcutaneous $\text{PCO}_2$ implies alveolar hypoventilation induced by FOB with the combination of i.v. sedation, underlying lung disease and the insertion of the bronchoscope into the major airways.

We observed the time taken to the first reduction in transcutaneous $\text{PO}_2$ and the first increase in transcutaneous $\text{PCO}_2$ (a change of at least 0.3 kPa from a steady baseline with the administration of supplemental oxygen via nasal cannulae) was quicker than the first reduction in oxygen saturation (at least 2% from baseline), in 19 of the 22 cases. This can be partly explained by extrapolation from the oxygen dissociation curve. In patients receiving supplemental oxygen via nasal cannulae an inspired oxygen concentration of up to 68% can be generated if the patient is hypoventilating (9) which will increase the $\text{PaO}_2$ to greater than 13.3 kPa. With further hypoventilation the reduction in $\text{PaO}_2$ will need to be large to reduce the $\text{SpO}_2$ because of the sigmoid shape of the oxygen dissociation curve. The more rapid reduction in transcutaneous $\text{PO}_2$ than $\text{SpO}_2$ is made more interesting in view of the fact that the response times for the TCM3 transcutaneous $\text{PCO}_2/\text{PO}_2$ monitor are slower than the corresponding times for the OXIMeter.

When comparing the changes in oxygen saturation and transcutaneous $\text{PCO}_2/\text{PO}_2$ with the time taken to introduce the bronchoscope into the trachea and the duration of the bronchoscopy, it can be seen that many of these changes either precede or follow the bronchoscopic procedure. Changes preceding the presence of the bronchoscope in the major airways can be explained by the effects of sedation in patients with underlying airways disease. The changes in oxygen saturation and transcutaneous $\text{PCO}_2/\text{PO}_2$ following the procedure is due to the sedation having a half-life greater than the duration of the bronchoscopy and the cessation of the sympathetic drive caused by the presence of the bronchoscope within the airways.

Fibreoptic bronchoscopy (FOB) is a safe, established technique but patients undergoing FOB are at risk of type II respiratory failure. Sedation can cause hypoventilation during endoscopic procedures secondary to central respiratory depression (1,2) and this can be aggravated in FOB by the introduction of the bronchoscope into the major airways, leading to a reduction in the cross-sectional area with an increase in resistance to airflow. This is in contrast to patients undergoing upper gastrointestinal endoscopy, in whom the presence of the endoscope does not appear to contribute to the airway obstruction or endoscope-induced CO$_2$ retention (10). Patients investigated for suspected lung cancer often have smoking-related obstructive airways disease which further increases the risk of procedural complications, but careful assessment can exclude patients at high risk.

Patients undergoing FOB are usually monitored with pulse oximetry. Maranetzra (3) documented a reduction in oxygen saturation of 1–25% (mean 5.6%) in 97 out of 100
patients undergoing FOB. Despite this, recent papers (4,5) have questioned the reliability of pulse oximetry as a monitoring technique in the presence of high inspired oxygen concentrations. Oximetry measures the oxygenation of blood and will detect hypoxaemia, it can not detect hypercapnia and therefore the adequacy of ventilation. Alveolar hypoventilation, produced by sedation during FOB, leads to a rise in alveolar and arterial carbon dioxide tension and a reduction in alveolar oxygen tension with resultant hypoxaemia. When breathing room air the oxygen saturation will fall rapidly, giving a good indication of ventilatory status, but when the patient is receiving supplemental oxygen the alveolar PCO₂ will have to rise further to cause hypoxaemia which will result in significant desaturation.

In our study we used transcutaneous PCO₂/PO₂ values to monitor respiratory status in patients undergoing FOB. Transcutaneous gas electrodes work on the principle that elevation of skin temperature (usually to 44°C) makes the skin permeable to gas diffusion by dissolving the lipid structure of dead, keratinized cells. This heating also increases cutaneous blood flow, arterializes the capillary blood and results in a shift in the oxygen dissociation curve to the right which amounts to a c. 6% increase in PO₂ per °C. This increase is offset by diffusion of oxygen out of the capillary loops and uptake of oxygen by metabolically active cells. In the measurement of transcutaneous PCO₂ the increase in temperature increases the PCO₂ in blood by c. 4.8% per °C and CO₂ is also produced by living cells. These factors result in transcutaneous levels of PCO₂ being greater and PO₂ being lower than the corresponding arterial levels. These principles are based upon haemodynamically stable subjects with adequate skin perfusion.

The TCM3 transcutaneous PCO₂/PO₂ electrode combines a Clark-type O₂ electrode and a Severinghaus-type CO₂ electrode. The O₂ electrode produces a current directly proportional to the O₂ concentration against the outer membrane. The CO₂ electrode measures a voltage generated by hydrogen ions produced after the CO₂ diffuses across the membrane to produce hydrogen and bicarbonate ions. Prior to use the electrodes must be calibrated against a gas of fixed concentration (one point calibration using 5% CO₂/95% O₂ or two point calibration 10% CO₂/90% N₂).

Transcutaneous levels of PO₂ and PCO₂ are valid in their own right; they do not correspond directly to the arterial values but can reflect change in arterial gas tensions. There are no normal ranges for transcutaneous measurements and our calculations and conclusions are based on the observed trends in transcutaneous PCO₂/PO₂.

The advantages of transcutaneous monitoring are the direct measurement of transcutaneous PCO₂ and PO₂ (important with high inspired oxygen concentrations) and a rapid response time. The disadvantages are that the procedure is time-consuming, requiring calibration, a 'warm-up' period and electrode repositioning at 4 h due to drift from the calibrated baseline. As with oximetry, transcutaneous monitoring requires good skin perfusion and it is not known if the potential thermal damage and oedema caused by the electrode affects sensitivity and response time. Data calibrating transcutaneous values against arterial blood samples are based on studies with neonates and it is not known how transcutaneous monitoring compares in adults, disease states, anxiety and in response to medication.

This study highlights the limitations of pulse oximetry in detecting respiratory failure in patients undergoing fibreoptic bronchoscopy who are receiving supplemental oxygen. This problem can be overcome by using a transcutaneous PCO₂/PO₂ monitor to assess the patient's respiratory status during the FOB. The clinical relevance of transcutaneous PCO₂/PO₂ monitoring during FOB over clinical observation and pulse oximetry is uncertain as in this study no further adverse events were prevented by this monitoring, but no patients in this study were severely compromised or required reversal of their sedation. This study has not changed our clinical practice in that we continue to use pulse oximetry to monitor patients during bronchoscopy, rather than the more complex transcutaneous PCO₂ analysis, but we are aware of the limitations of oximetry and place greater emphasis on clinical evidence of hyperventilation.

References


