Continuous measurements of oxygen saturation during haemodialysis

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Abstract. A new technique for recording and analysing continuous measurements of oxygen saturation (SpO₂) by pulse oximeter during haemodialysis was used to compare changes in SpO₂ in eight patients during two 4 h periods of dialysis using a cuprophane membrane, once using an acetate dialysate, and once using bicarbonate. The computer-derived patterns of SpO₂ show whether hypoxaemia was caused mainly by extrapulmonary abnormalities (ventilatory control) or intrapulmonary abnormalities (V/Q distribution). The patterns of oxygen saturation were analysed for (i) stability, (ii) the lower median 20th centile of SpO₂, and (iii) time below a SpO₂ of 90%. Not all patients had reduced oxygenation during acetate dialysis. Three of eight patients had a stable pattern with acetate dialysis and six of eight were stable with bicarbonate. Five of eight patients had a lower SpO₂ with acetate but one patient had a lower SpO₂ with bicarbonate. Four patients had prolonged, clinically significant periods of oxygen desaturation with SpO₂ < 90%; two of these had particularly prolonged periods during acetate (62 min and 12 min), but one patient showed a longer period during bicarbonate than acetate dialysis (7 min). In two patients the SpO₂ declined to less than 84%. The patterns of SpO₂ suggested that the decrease in oxygen saturation was due more to extrapulmonary abnormalities causing an instability in ventilatory control rather than to venous admixture. It is recommended that pulse oximetry is used to identify patients at risk of hypoxaemia, to monitor these patients during haemodialysis, and to administer oxygen to those whose SpO₂ falls below 90%, particularly if they have anaemia or cardiovascular disease.

Key words: haemodialysis; oxygen saturation; oximeter

Introduction

Hypoxaemia during haemodialysis was first described in 1972 [1] and these observations have been supported by subsequent studies [2–9]. The cause of these changes is not known with certainty and has been attributed to two different mechanisms. Firstly, intrapulmonary leukostasis [2,3] may cause increased lung permeability, alveolar oedema and an enhanced ventilation-perfusion (V/Q) inequality. Haemodialysis using bioincompatible membranes such as cuprophane rather than polyacrylonitril (PAN) is claimed to cause a greater increase in V/Q inequality manifest as a rise in the alveolar to arterial oxygen tension gradient [4,5]. A decrease in cardiac output will worsen the effect of V/Q inequality on oxygen saturation. Secondly, impaired ventilatory control due to loss of carbon dioxide in the dialysate [6–8]. This may be reduced if carbon dioxide loss in the dialysate is prevented by using bicarbonate dialysate, and it has been shown that there is a greater fall in arterial oxygen tension (PaO₂) during acetate dialysis where there is a large loss of carbon dioxide compared to bicarbonate dialysate [9]. Impaired ventilatory control may also be associated with a decrease in alveolar...
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Oxygen saturation was measured on the same sample using a co-oximeter (Radiometer OSM 2 hemoximeter) and the result compared to the PaO₂ to determine the degree of shift of the oxygen dissociation curve. Students t test was used to measure the difference between the values of PaO₂, PaCO₂, pH, and base excess.

Results

Oxygen saturation

Distribution of SpO₂ in each 5 min interval was displayed as a spectrum of SpO₂ values against time for each patient during acetate and bicarbonate dialysis. The first six curves in each assay represent the predialysis period and provide information about baseline oxygen saturation over a 30 min period.

Five of the eight patients (nos 1–5) showed a highly stable pattern of SpO₂ during bicarbonate dialysis. The peaks in each time interval were tall and narrow, the peak in each interval almost superimposing the peaks in subsequent intervals. This is indicative of normal ventilatory control (Figure 1).

During acetate dialysis three patients (nos 1–3) showed little difference in the pattern of SpO₂ compared to bicarbonate dialysis (Figure 1) (Table 1). The readings were stable, with little spread of results around the mean values and no periods of saturation below 90%. In all three patients the SpO₂ was significantly reduced during acetate rather than bicarbonate dialysis.

Two patients (nos 4, 5) with a highly stable pattern of SpO₂ on bicarbonate showed, during acetate dialysis, a decreasing SpO₂ with a wide variation of the pattern between successive time intervals. For example patient 5, with stable SpO₂ during bicarbonate dialysis, had consistently reduced arterial oxygen throughout acetate dialysis with prolonged periods of desaturation less than 90% (Figure 2) (Table 1). One hour after the start of dialysis patient 5 showed a large reduction in oxygen saturation to less than 90% SpO₂ for 1 h and then recovered during the remainder of the dialysis. This was not seen during bicarbonate dialysis. The study was discontinued at this time and the patient switched back to bicarbonate dialysis.

Three patients (nos 6–8) showed a very unstable pattern of SpO₂ during both bicarbonate and acetate dialysis. Patient no. 8 showed a reasonably good pattern between successive time intervals. For example patient 5, with stable SpO₂ during bicarbonate dialysis, had consistently reduced arterial oxygen throughout acetate dialysis with prolonged periods of desaturation less than 90% (Figure 2) (Table 1). One hour after the start of dialysis patient 5 showed a large reduction in oxygen saturation to less than 90% SpO₂ for 1 h and then recovered during the remainder of the dialysis. This was not seen during bicarbonate dialysis. The study was discontinued at this time and the patient switched back to bicarbonate dialysis.

Subjects and methods

Eight patients with chronic renal failure were studied. Their ages ranged from 23 to 65 years and they had been undergoing haemodialysis routinely for 8–25 months. Informed consent was obtained and the study had the approval of the hospital ethical committee. All patients received a 4 h haemodialysis with a cuprophane membrane three times a week. Each patient was studied on two occasions: once using a bicarbonate-containing dialysate and once with an acetate-containing dialysate. Vascular access was by an arteriovenous fistula and anticoagulation was maintained with heparin.

Using a pulse oximeter (Ohmeda Biox 3700) and a disposable finger probe, oxygen saturations were measured continuously for 30 min predialysis and during the 4 h dialysis period. The pulse oximeter was interfaced with an IBM-compatible portable computer and a 20 Mb Winchester hard disk. Every 10 s the oximeter sent a 3-character string to the computer which displayed it on the monitor screen. The display included SpO₂, heart rate, oximeter and alarm status, and the time of recording. The data was automatically written to a file on the hard disc, which was used to show if there was any significant difference between the two dialyses. In this regard the median of the lower 20th centile of SpO₂ was analysed because of the clinical implications of the duration of low oxygen saturations. We also analysed the duration of time spent below an oxygen saturation of 90% because this corresponds to a dangerously low PaO₂ in patients with anaemia and cardiovascular disease.

Arterial blood samples were taken at 0, 30, 60, 120, 180, and 240 min from the start of haemodialysis and assayed for pH, arterial oxygen tension (PaO₂), arterial carbon dioxide tension (PaCO₂) and base excess, using a blood gas analyser (Instrument Lab 1306 pH, blood gas analyser).

Oxygen due to the effect of CO₂ washout on the gas exchange R value.

Previous work has relied on measurements of oxygen tension using repeated arterial blood samples, which is invasive, inconvenient and intermittent. Continuous measurement of arterial oxygen saturation has recently become commonplace using non-invasive pulse oximetry and its use has previously been documented during haemodialysis [10]. The aim of this study was to use pulse oximetry to examine oxygen saturation (SpO₂) continuously during haemodialysis and to compare the effects of acetate and bicarbonate dialysates.

One of the problems with continuous oximetry is the difficulty in displaying the mass of data produced by this approach. One solution is to produce arrays of SpO₂ distribution in consecutive time intervals [11]. The advantage of this approach is that it compresses a large amount of data into a small, clear summary of the pattern of oxygen saturation during any time period.

Distribution of SpO₂ in each 5 min interval was displayed as a spectrum of SpO₂ values against time for each patient during acetate and bicarbonate dialysis. The first six curves in each assay represent the predialysis period and provide information about baseline oxygen saturation over a 30 min period.

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Table 1. Changes in oxygen saturation during dialysis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Dialysate</th>
<th>Pattern of SpO₂</th>
<th>Lower median SpO₂ 20th centile</th>
<th>Time below a SpO₂ of 90%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>Stable</td>
<td>96</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>Stable</td>
<td>97*</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>Stable</td>
<td>95</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>Stable</td>
<td>97*</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>A</td>
<td>Stable</td>
<td>96</td>
<td>-</td>
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<td></td>
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<td>-</td>
</tr>
<tr>
<td>5</td>
<td>A</td>
<td>Unstable</td>
<td>91</td>
<td>62 m 14 s</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>Stable</td>
<td>96*</td>
<td>1 m 50 s</td>
</tr>
<tr>
<td>6</td>
<td>A</td>
<td>Unstable</td>
<td>94</td>
<td>6 m 4 s</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>Unstable</td>
<td>95*</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>A</td>
<td>Unstable</td>
<td>96</td>
<td>12 m 24 s</td>
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<td>95*</td>
<td>2 m 56 s</td>
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<td>94</td>
<td>4 m 56 s</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>Unstable</td>
<td>95 NS</td>
<td>7 m 30 s</td>
</tr>
</tbody>
</table>

* Significant difference between the two values; in each case P < 0.001.
A, acetate; B, bicarbonate.

Blood gas analysis

The oxygen saturation was measured in the arterial samples using a co-oximeter and this value together with the PaO₂ used to calculate the position of the oxygen dissociation curve. The episodes of desaturation measured by the pulse oximeter showed no correlation with shifts in the oxygen dissociation curve.

Arterial oxygen tension (PaO₂) decreased during haemodialysis using an acetate dialysate and this change compared to control reached significance at 60, 120, and 180 min (P < 0.05). There was no significant reduction in PaO₂ during bicarbonate dialysis (Table 2).

Discussion

The computer-derived curves, compressed into arrays of oxygen saturation during haemodialysis, showed two different patterns in this study. The first pattern showed tall well-defined peaks of SpO₂ values with a small variation from interval to interval, indicating a stable recording which is compatible with normal ventilatory control. In the second pattern the curves were broad, showing very large variation between sampling intervals often falling to low values of oxygen saturation for long periods. This is indicative of impaired ventilatory control [11]. A third pattern, seen in postoperative studies where atelectasis complicated thoracic surgery, is a closely superimposed set of curves all at a reduced oxygen saturation, indicative of venous admixture with normal ventil-
Continuous measurements of $O_2$ saturation during haemodialysis

Fig. 2. Patient 5 shows good ventilatory control during bicarbonate dialysis but wide variability in the pattern of $SpO_2$ on acetate. This is indicative of impaired ventilatory control. Note that the $SpO_2$ is below 90% for a considerable time on dialysis.

Fig. 3. Patient 8 shows very unstable ventilatory control during both acetate and bicarbonate dialysis.

When acetate and bicarbonate dialysates were compared six of the eight patients showed a significantly reduced $SpO_2$ during acetate but not bicarbonate dialysis. However, oxygen saturation decreased to less than 90% for prolonged periods in four patients. A $SpO_2$ of 90% is equivalent to a $PaO_2$ of 8 kPa if there has been no shift in the position of the oxygen dissociation curve, and this level of saturation represents a serious degree of hypoxaemia, particularly in anaemic patients, those with cardiovascular disease, or those who have cardiovascular instability during dialysis. This may lead to a severe reduction of oxygen delivery to the brain, myocardium, and other organs and is an indication for oxygen therapy during haemodialysis in these easily identified patients. $PaO_2$ readings are of limited clinical usefulness because of...
Previous authors have shown that during haemodialysis the worst combination of dialysate and membrane in the production of hypoxaemia is acetate with cuprophane [9]. These authors also claimed that the use of acetate instead of bicarbonate was consistently associated with hypoxaemia, although not all patients in our study developed greater falls in oxygen saturation with acetate than with bicarbonate dialysis.

Several mechanisms have been proposed to account for the hypoxaemia. One proposal is that there is a decrease in granulocyte count during the first 15 min of dialysis [12]. These granulocytes adhere to pulmonary vessels, release free radicals [13], and cause an increase in interstitial and alveolar water. This reduction in white cell count is only seen when bioincompatible membranes such as cuprophane are used and not during dialysis with PAN [9]. It has been suggested that this is due to activation of the alternative path-
and it has been shown recently caused not by hypoxaemia but by the effects of pre-existing V/Q inequality on hypoxaemia. Symptomatic hypotension is the most frequent problem seen during routine haemodialysis and is substantiated by other authors.

However, this was only partially due to the effects of CO₂ washout on ventilatory control and on the gas exchange R value, and cannot be explained by shifts in the oxygen dissociation curve. Some patients tolerate acetate dialysis without any hypoxaemia and were equally happy with either dialysate. But patients who develop significant periods of desaturation (<90% SpO₂) or cardiovascular instability felt better and preferred bicarbonate rather than acetate dialysis. The clinical implications of the study are as follows:

1. Severe hypoxaemia, a SpO₂ less than 90%, occurred in about half of the patients.
2. Prolonged hypoxaemia (<90% SpO₂) may be associated with severe impairment of tissue oxygenation in anaemic patients with impaired cardiac output or regional blood flow.

These patients may well be typical of others on chronic haemodialysis programmes; thus pulse oximetry should be used to identify patients who become hypoxaemic. Such patients, particularly if they are anaemic and have cardiovascular instability, should not only be monitored but also be given supplemental oxygen therapy.

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References

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PaO_2 = P_1O_2 - \frac{PaCO_2}{R}
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It has also been shown that if the loss of carbon dioxide through the dialysis membrane causes hypoventilation and periods of apnoea due to impaired ventilatory drive [6-8] and it has been shown that it is the carbon dioxide load that determines ventilatory rate [14]. However a very important mechanisms is the effect on the respiratory exchange ratio (R). The PaCO₂ in venous blood returning from the machine is diminished and this reduced production of carbon dioxide is reflected in a fall in R and therefore a decrease in PaO₂.

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