EFFECT OF INSPIRED OXYGEN CONCENTRATION ON THE INCIDENCE OF DESATURATION IN PATIENTS UNDERGOING TOTAL HIP REPLACEMENT

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SUMMARY

The effect of inspired oxygen concentration on the incidence of desaturation was studied in two groups of patients (n = 32 and 31) during total hip replacement. During insertion of acrylic bone cement, those patients receiving an inspired fractional oxygen concentration (FiO₂) of 0.5 had less desaturation (3%) than those receiving an FiO₂ of 0.33 (34%).

KEY WORDS


There have been several reports of complications, including hypoxaemia, associated with the insertion of acrylic bone cement (polymethylmethacrylate) during hip arthroplasty [1-3].

The present study was designed to compare the effects of an FiO₂ of 0.5 and one of 0.33 on the incidence of desaturation during insertion of cement in total hip replacement.

PATIENTS AND METHODS

Sixty-three ASA I or II patients undergoing total hip replacement surgery were studied. The study was approved by the Hospital Ethics Committee and informed written consent was obtained from each patient. All patients were premedicated with papaeveretum 0.3 mg kg⁻¹ and hyoscine 0.006 mg kg⁻¹ i.m. 1 h before operation. Anaesthesia was induced with thiopentone 4–5 mg kg⁻¹. Tracheal intubation was facilitated with vecuronium 0.1 mg kg⁻¹; the lungs were ventilated mechanically, and anaesthesia was maintained with nitrous oxide and 1–1.5% enflurane in oxygen. All patients were monitored using pulse oximetry (SpO₂), non-invasive arterial pressure (SAP) and FiO₂ (Cardiocap, Datex). The patients were positioned for surgery in the lateral position on a horizontal table with the pulse oximeter probe positioned on the non-dependent ear. The patients were allocated randomly to two groups: group I (n = 32) received an FiO₂ of 0.33 throughout the whole procedure, while in group II (n = 31) FiO₂ was increased to 0.5 at least 10 min before insertion of the cement and continued for a further 10 min after the insertion of the cement for each of the acetabular and femoral components (Table I).

The increase in FiO₂ from 0.33 to 0.5 in group II was timed in co-ordination with the surgical and nursing staff. The acetabular component was always inserted first, followed by the femoral component. In group II, after insertion of the acetabular component, FiO₂ was restored to 0.33 and increased to 0.5 10 min before insertion of the femoral component. The femoral cement was inserted using a cement gun, without venting of the shaft. SAP and SpO₂ were measured at 1-min intervals for 10 min before and 10 min after the

| Table I. Patient characteristics (mean (range or sd)) |
|-----------------|------|------------|--------|-------|
| FiO₂            | n    | Age (yr)   | Weight (kg) | Sex (M/F) |
| 0.33            | 32   | 69.4 (65–73) | 72.1 (8.5) | 10/22 |
| 0.5             | 31   | 71.2 (67–74) | 70.8 (9.6) | 12/19 |

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insertion of cement for both components. Measurements were continued beyond 10 min if changes in $SpO_2$ were of longer duration (two patients in group I).

A persistent decrease of 2% or greater in $SpO_2$ and a decrease of 10% or greater in arterial pressure was considered a clinically significant event. Data were analysed using chi-square with Yates’ correction and Mann–Whitney U test as appropriate. $P < 0.05$ was considered statistically significant.

RESULTS

Eleven patients (34%) in group I had a decrease in $SpO_2$, during insertion of the cement. Only one patient (3%) in group II had a decrease in $SpO_2$ during insertion of the cement for a femoral component. Table II shows the incidence of the reduction in $SpO_2$ (2% or more) in both groups during insertion of the cement of the acetabular, femoral or both components of the total hip arthroplasty. The maximum decrease in $SpO_2$ was 10% in group I and 2% in group II.

The decrease in $SpO_2$ occurred within 1–4 min (median 1 (SD 0.93) min) after insertion of the cement and lasted for 5–25 min (median 9.5 (SD 5.92) min). The only episodes of decrease in $SpO_2$ were associated with cement insertion, in both groups throughout the operation.

There was no relation between the incidence or degree of reduction in $SpO_2$, and the general medical condition of the patients ($P = 0.576$) or the habit of smoking ($P = 0.925$) in each group.

In both groups, a decrease in $SpO_2$ did not correlate with a decrease in arterial pressure ($P = 0.85$) (table III). The decreases in SAP lasted for 1–2 min and recovered without treatment.

DISCUSSION

This study has demonstrated a decrease in $SpO_2$, following insertion of cement during total hip replacement in about 30% of patients when an $F_1O_2$ of 0.33 was used. The greatest decrease in $SpO_2$ was 10% (from 96% to 86%) (fig. 1); this occurred during the insertion of cement for the femoral component, but there were no associated changes in arterial pressure in this healthy, non-smoking patient.

The pathophysiological mechanisms responsible for these changes in $SpO_2$ are not clear. Previous work had suggested that cement entering the circulation causes sudden and transient

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**Table II. Number of patients in each group with a decrease in $SpO_2$ of 2% or greater during insertion of the cement of the acetabular, femoral or both components of the total hip replacement. *Changes more frequent ($P < 0.05$) than in group II**

<table>
<thead>
<tr>
<th>Group</th>
<th>$n$</th>
<th>Acetabulum</th>
<th>Femoral</th>
<th>Both</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>32</td>
<td>2</td>
<td>7</td>
<td>2</td>
<td>11 (34.37%)*</td>
</tr>
<tr>
<td>II</td>
<td>31</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1 (3.22%)</td>
</tr>
</tbody>
</table>

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![Graph](https://example.com/graph.png)

**Fig. 1. Decrease in $SpO_2$ as the cement was inserted in the femoral component in a patient in group I.**
TABLE III. Numbers of patients in each group who had a decrease in SAP during the insertion of the cement and a decrease in both SAP and $Sp_{O_2}$

<table>
<thead>
<tr>
<th>Group</th>
<th>Patients with decrease in SAP</th>
<th>Patients with decrease in SAP and $Sp_{O_2}$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Decrease (mm Hg)</td>
</tr>
<tr>
<td>Acetabular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group I</td>
<td>13</td>
<td>9-22</td>
</tr>
<tr>
<td>Group II</td>
<td>4</td>
<td>9-16</td>
</tr>
<tr>
<td>Femoral</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group I</td>
<td>15</td>
<td>15-31</td>
</tr>
<tr>
<td>Group II</td>
<td>7</td>
<td>10-15</td>
</tr>
</tbody>
</table>

Changes in the pulmonary vasculature [2]. Others have suggested that the release of tissue-thromboplast products into the circulation causes intravascular coagulation and respiratory reaction [3]. It has also been suggested that microembolization of air and fat occurs with denaturing of the proteins of the blood draining the area, because of the heat produced during polymerization of the cement, with the release of intracellular components into the circulation [4].

The incidence and severity of the reduction in $Sp_{O_2}$ were greater with the insertion of the femoral component than the acetabular component, as shown by previous workers [1-3]. This could be caused by various factors, including the larger surface area and the higher vascularity of the femoral shaft with the very high pressures generated as the cement sets [1]. Intramedullary pressure may increase to more than 500 mm Hg during cemented implants [5]. Another factor may be that the amount of cement used for the femoral component is about twice that used for the acetabular component.

Our observation that utilization of an $F_{I_{O_2}}$ of 0.5 decreased significantly the incidence of desaturation, implies that desaturation caused by insertion of the cement was caused by ventilation-perfusion inequalities rather than an increase in the true right–left shunt [6]. Changes in arterial pressure after insertion of cement had no effect on changes in $Sp_{O_2}$ and this is in agreement with previous studies [2]. The arterial pressure changes were transient, whereas the duration of desaturation lasted up to 25 min. Similar observations have been described previously [3, 7].

Within the clinical range, arterial oxygen saturation is measured accurately by pulse oximetry [8]. Positioning the pulse oximeter probe on the non-dependent ear is likely to reflect arterial oxygenation more reliably than the fingers, as acute changes in $Sp_{O_2}$ may be delayed by 30–60 s in the finger compared with the ear [9]. Pulse oximeters are also less reliable during transient severe desaturation as their programs average and compute the input in order to reject any artefacts.

These results indicate that increasing $F_{I_{O_2}}$ to 0.5 during the insertion of cement in total hip arthroplasty is to be recommended.

REFERENCES