ANAESTHETIC MANAGEMENT OF THORACOTOMY IN THE PRESENCE OF CHRONIC CARBON DIOXIDE RETENTION

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SUMMARY
A patient suffering from chronic carbon dioxide retention with persistent pneumothorax, following spontaneous rupture of an emphysematous bulla, underwent thoracotomy. The surgical and anaesthetic management are reported.

CASE HISTORY
A male, aged 63 yr, had a long history of chronic productive cough and dyspnoea. He smoked 10–20 cigarettes per day. In 1973 he sustained a spontaneous left pneumothorax which resolved without aspiration or intercostal drainage.

On July 30, 1975, he became more dyspnoeic suddenly and, 2 days later, was admitted to hospital where a chest x-ray showed a large right pneumothorax. An intercostal tube was inserted and the lung re-expanded, but an air leak continued. During the next 6 weeks, despite various patent intercostal tubes, with and without suction, the pneumothorax persisted (fig. 1) and the air leak appeared to be increasing in volume. $P_a^{CO_2}$ was 9.9–10.5 kPa and $P_a^{O_2}$ 4.3–5.3 kPa, when the patient breathed air.

ASSESSMENT
By this time it was clear that intercostal drainage as the sole method of treatment would not succeed. Chemical pleurodesis was contraindicated because the lung was partly collapsed, and thoracotomy with closure of the hole and pleurectomy appeared to be the only feasible alternative.

The following policy was adopted:
(1) IPPV would be kept to a minimum, to reduce the risk of possible fatal pneumothorax on the left side, and to avoid respirator dependence after operation in a patient with chronically impaired pulmonary function.
(2) Surgery would have to be rapid and effective in closing the air leak.
(3) Elective tracheotomy would be performed at the time of operation.
(4) Arterial cannulation would be performed for serial blood-gas analysis and the patient would be admitted to the intensive care unit after operation.
(5) After operation blood-gas abnormalities that were no worse than those that had been tolerated before operation would be accepted and not regarded as an indication for IPPV.

The operation was performed on September 11, 1975. Anaesthesia was induced with halothane vaporized in 100% oxygen. The larynx and trachea were sprayed with 2% lignocaine 10 ml and an oral cuffed endotracheal tube was inserted. Spontaneous
respiration was maintained and anaesthesia continued with halothane and oxygen via a Magill circuit, with a fresh gas flow of 10 litre min⁻¹.

Thoracotomy was commenced, suxamethonium 35 mg was injected i.v. as the pleura was approached, and gentle manual ventilation commenced. The parietal pleura was stripped off the upper half of the chest wall and excised. The apex of the right upper lobe contained a mass of emphysematous bullae about 7 cm in diameter and one of these had a hole at its base 2 mm in diameter, from which air was issuing freely. This was closed with a mattress suture of 2/0 silk reinforced with Teflon felt. The chest was closed in a routine manner with one intercostal drain.

With the patient supine tracheotomy was performed and a cuffed Portex tube was inserted. At the conclusion of operation $P_{\text{aCO}}$ was 14.0 kPa and $P_{\text{aO}}$, 10.9 kPa. Total anaesthesia time was 1 h 15 min.

**Table I. Blood-gas data**

<table>
<thead>
<tr>
<th>Programme for “weaning” from IPPV</th>
<th>Time of sampling (h)</th>
<th>IPPV stopped before sampling (min)</th>
<th>$P_{\text{aCO}}$ (kPa)</th>
<th>$P_{\text{aO}}$ (kPa)</th>
<th>Oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>11.9.75 15 min (once) then 45 min (×3) during day. Controlled IPPV at night</td>
<td>14.15</td>
<td>15</td>
<td>8.7</td>
<td>11.5</td>
<td>5 litre 40% Puritan</td>
</tr>
<tr>
<td>11.9.75 2 h (once) then 15 min. h⁻¹ during day. Controlled IPPV at night</td>
<td>11.45</td>
<td>120</td>
<td>10.1</td>
<td>7.2</td>
<td>3 litre Mistogen + some air</td>
</tr>
<tr>
<td>11.9.75 20 min. h⁻¹ during day. Assisted IPPV at night</td>
<td>10.35</td>
<td>15</td>
<td>7.2</td>
<td>17.3</td>
<td>5 litre 40% Puritan</td>
</tr>
<tr>
<td>11.9.75 30 min. h⁻¹ during day. Assisted IPPV at night</td>
<td>11.45</td>
<td>25</td>
<td>6.8</td>
<td>16.8</td>
<td>5 litre 40% Puritan</td>
</tr>
<tr>
<td>15.9.75 45 min. h⁻¹ and later 2 h (×2) during day. Assisted IPPV at night</td>
<td>11.45</td>
<td>45</td>
<td>7.3</td>
<td>6.8</td>
<td>None</td>
</tr>
<tr>
<td>16.9.75 1 h 50 min/2 h day and night</td>
<td>11.10</td>
<td>120</td>
<td>8.6</td>
<td>6.9</td>
<td>2 litre via Puritan on air</td>
</tr>
<tr>
<td>17.9.75 Off IPPV throughout</td>
<td>11.00</td>
<td>60</td>
<td>8.2</td>
<td>7.2</td>
<td>5 litre 40% Puritan via Negus tube</td>
</tr>
<tr>
<td>29.9.75</td>
<td>—</td>
<td>10.00</td>
<td>—</td>
<td>5.6</td>
<td>9.2</td>
</tr>
<tr>
<td>29.3.76</td>
<td>—</td>
<td>12.00</td>
<td>—</td>
<td>6.1</td>
<td>9.1</td>
</tr>
</tbody>
</table>

11.9.75 = day of operation.
increased to 10.6 kPa despite $\text{PaO}_2$ being 5.3 kPa, and IPPV was recommenced.

On the next day (day 1) 2 h of spontaneous breathing was tolerated by the patient, but at the end of this time he was tired and $\text{PaCO}_2$ was 10.1 kPa. A programme for "weaning" from the ventilator was commenced, starting with 15 min/h of spontaneous breathing on day 1, 20 min on day 2 and 30 min on day 3 using a T-piece with patient-triggered IPPV at other times and at night.

On day 3, a chest x-ray revealed a small left pneumothorax. This did not increase in size and never required evacuation, but it confirmed the risk of IPPV. On day 4 the patient could breathe spontaneously for 1 h 50 min in every 2 h ($\text{PaCO}_2$ 7.9 kPa and $\text{PaO}_2$ 8.7 kPa) and this was continued overnight.

On the next day IPPV was discontinued completely. A Negus tube substituted for the cuffed Portex tube was well tolerated and it was removed and the tracheostomy closed by direct suture so as to restore an effective cough. The patient returned to the ward breathing air.

Pain relief throughout was with morphine sulphate 2.5 mg i.v. as necessary, with gentle "bagging" and tracheo-bronchial suction for the removal of secretions.

Subsequent recovery was uncomplicated and the patient was discharged on the 19th day after surgery; $\text{PaCO}_2$ was 5.6 kPa and $\text{PaO}_2$ was 9.2 kPa breathing air.

Six months later he was ambulant and well. $\text{PaCO}_2$ was 6.2 kPa and $\text{PaO}_2$ was 9.1 kPa on air. Pulmonary function studies show: Forced Vital Capacity 2.5 litre (predicted 3.8 litre) FEV$_1$ 0.8 litre (predicted 2.75 litre), FEV$_1$/VC% 32, Residual Volume 3.5 litre (predicted 2.2 litre), Total Lung Capacity 5.95 litre (predicted 6.2 litre) and RV/TLC 59%, transfer factor 82% of normal, indicating a moderately restrictive and severely obstructive impairment of ventilatory capacity consistent with chronic obstructive airways disease.

DISCUSSION

While various respiratory parameters have been found to correlate broadly with events after operation (Andersen and Ghia, 1970; Stein and Cassara, 1970; Lockwood, 1973) they do not predict survival in specific instances.

Millelde and Nunn (1975) reported four patients, who underwent surgery when $\text{PaCO}_2$ was greater than 5.9 kPa. Two of these had non-incisional surgery and two had intra-abdominal operations. Both of the latter group survived, although one required IPPV for 5 days. Similar precedents for patients undergoing thoracotomy have not been established.

That this patient suffered from chronic carbon dioxide retention is indicated by the continuing increased $\text{PaCO}_2$ 6 months after operation, and other pulmonary function data which indicate a mixed moderately restrictive and severely obstructive impairment of ventilatory capacity.

That such a patient should survive thoracotomy we attribute to the improvement in lung function after operation because of improved expansion of the right lung, and the operative and postoperative regimen.

It is not intended that this case should justify an indiscriminate policy of operating on hopeless respiratory cripples for diseases which in themselves have a bad prognosis, but it does demonstrate that in selected situations, where some acute but reversible complication has occurred, the patient with chronic carbon dioxide retention can in fact survive thoracotomy and be restored to reasonable and enjoyable health.

REFERENCES


NARKOSEDURCHFÜHRUNG BEI BRUSTRAUMERÖFFNUNG BEI VORHANDENSEIN EINER CHRONISCHEN KOHLENSTOFFDIOXYDRETENTION

ZUSAMMENFASSUNG

TRATAMIENTO ANESTÉSICO DE TORACOTOMIA EN PRESENCIA DE RETENCION CRÓNICA DE BIOXIDO DE CARBONO

SUMARIO
Un paciente afectado de retención crónica de dióxido de carbono con neumotórax persistente, consecutivo a la ruptura espontánea de una ampolla enfisematosa, fue sometido a toracotomía. Se comunican los tratamientos quirúrgico y anestésico.