CONTINUOUS POSITIVE PRESSURE VENTILATION IN THE MANAGEMENT OF EIGHT CASES OF ACUTE PULMONARY OEDEMA

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SUMMARY

The blood-gas and circulatory effects of continuous positive pressure ventilation (CPPV) were studied in eight patients with acute pulmonary oedema. In four cases, an end-expiratory pressure of +10 cm H₂O led to a marked reduction of the alveolar/arterial oxygen tension difference. In two further cases, it was necessary to increase the end-expiratory pressure to +15 cm H₂O and +20 cm H₂O respectively to obtain a similar improvement. In the remaining two cases, CPPV was ineffective.

Continuous positive pressure ventilation appears to be of value in the treatment of acute pulmonary oedema (Uzawa and Ashbaugh, 1969; Cheney and Martin, 1971) but the risk of adverse circulatory effects limits its use (Kumar et al., 1970). We have studied eight cases of acute pulmonary oedema of different aetiology. The different responses of the alveolar/arterial oxygen tension difference ((A-a)PO₂) to continuous positive pressure ventilation are analysed.

PATIENTS AND METHODS

Eight patients diagnosed as having acute pulmonary oedema were studied (table I). The diagnosis was based on clinical findings (history, cyanosis, dyspnoea, pulmonary rales, expectoration of blood, and gallop rhythm) and on the radiographic appearances of the lung fields. In addition to treatment with digitalis and diuretics, an endotracheal tube was passed and the patients were mechanically ventilated with a volume-controlled ventilator (Engström Model 300). The concentration of the inspired oxygen (P吸入) was measured with a Teledyne Beckman oxygen analyser. A floating No. 7 Swan-Ganz catheter was inserted and the patients monitored with an electronic monitor (Statham P231B).

TABLE I. Relevant data for patients studied.

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Pulmonary complication</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52</td>
<td>Mixed mitral lesion. Cardiac failure. Atricular fibrillation in the last 5 years</td>
<td>APO</td>
<td>Acute renal failure. Pulmonary embolism. Ventricular fibrillation and death. Autopsy performed</td>
</tr>
<tr>
<td>2</td>
<td>79</td>
<td>AMI</td>
<td></td>
<td>APO</td>
</tr>
<tr>
<td>3</td>
<td>60</td>
<td>Chronic bronchitis. In the last year angina with exercise. AMI 4 days before admission</td>
<td>APO</td>
<td>12 hours after admission complete radiological remission. Transferred to a general ward 15 days later</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>Cirrhosis of the liver. Anasarca. Congestive heart failure. Initial diagnosis: APO</td>
<td>Bilateral pneumonia</td>
<td>Cardiac arrest and death 7 hours after admission. Bilateral pneumonia demonstrated at autopsy</td>
</tr>
<tr>
<td>5</td>
<td>32</td>
<td>Chronic alcoholism. During an acute alcoholism episode fell into a mixture of water and alkali. Aspiration</td>
<td>APO</td>
<td>Improvement in the first 48 hours. Respiratory failure and death 34 days later. Microscopic pulmonary lesions compatible with oxygen toxicity</td>
</tr>
<tr>
<td>6</td>
<td>50</td>
<td>AMI shock</td>
<td>APO</td>
<td>Cardiac arrest with recovery 4 hours after admission. Anuria. Death 10 hours later. Autopsy. Two coronary arteries occluded</td>
</tr>
<tr>
<td>7</td>
<td>17</td>
<td>Subarachnoid haemorrhage. Unconsciousness. Aspiration of gastric juice</td>
<td>APO</td>
<td>Partial remission of APO during the first 24 hours. At 33 hours CPPV stopped. At 41 hours shock followed by ventricular fibrillation and death</td>
</tr>
<tr>
<td>8</td>
<td>36</td>
<td>Mixed mitral lesion. Mixed aortic lesion</td>
<td>APO</td>
<td></td>
</tr>
</tbody>
</table>

APO = acute pulmonary oedema  AMI = acute myocardial infarction
catheter was placed via an antecubital vein in the pulmonary artery under X-ray control. A Courand BD, G18 needle was placed in the brachial artery by percutaneous puncture. The systemic and pulmonary artery pressures were measured directly using the Elema Schonander pressure transducers. The pressure wave-forms were inscribed on a Mingograf 81 recorder. The catheters were used also for blood sampling for gas analysis. These measurements were made with an Eschweiler Combianalizator. The alveolar/arterial oxygen tension difference, the arterial/venous oxygen content difference and the shunt ratio \((Q_s/Q_t)\) were calculated from these data. Measurements were made as follows:

1. During intermittent positive pressure ventilation (IPPV) with an end-expiratory pressure equal to the atmospheric pressure (average for Madrid 706 mm Hg).

2. After increasing the end-expiratory pressure by 10 cm H\(_2\)O, serial samples were taken from each patient at 15-min intervals.

3. In the last two cases, the end-expiratory pressure was increased to 15 and 20 cm H\(_2\)O respectively. Serial samples of blood were taken at 30-min intervals.

4. In four cases, the end-expiratory pressure was returned to the atmospheric level and an additional blood sample was taken 15 min later. In these cases, the full sequence was repeated after recommencing CPPV.

Controlled ventilation was facilitated by the use of intravenous pancuronium 2–4 mg, repeated if necessary.

The fraction of cardiac output passing through a shunt \((Q_s/Q_t)\) was calculated using the formula (Bartels et al., 1963):

\[
\frac{Q_s}{Q_t} = \frac{(C'O_2 - C_AO_2)}{(C'CO_2 - CVO_2)}
\]

Alveolar oxygen tension \((P_{AO_2})\) was calculated as follows:

\[
P_{AO_2} = P_{O_2} - (P_{A0_2}/R)(1 - F_{L2O}(1 - R))
\]

(Riley et al., 1946).

The alveolar/arterial oxygen tension difference \(((A-a)PO_2)\) is the difference between the alveolar oxygen tension and the arterial oxygen tension. The arterial/venous oxygen content difference was calculated from \(C_AO_2 - CVO_2\), where \(C_AO_2\) and \(CVO_2\) are the content of oxygen in arterial and venous blood respectively.

**RESULTS**

The effect of CPPV was not uniform in all eight cases. Four patients (1, 2, 3 and 5; fig. 1) showed a decrease in alveolar/arterial oxygen content difference in response to an end-expiratory pressure of +10 cm H\(_2\)O. The best reduction was 42% (average 13% at 15 min, 19% at 30 min and 22% at 60 min). In patients 4, 6, 7 and 8, the decrease was negligible (average 0.6% at 30 min and 2% at 60 min). In patients 7 and 8, increasing the end-expiratory pressure to +15 cm H\(_2\)O and +20 cm H\(_2\)O respectively was associated with decreases in alveolar/arterial oxygen tension differences of 16 and 26%. In four patients in whom the end-expiratory pressure of +10 cm H\(_2\)O was stopped, the improvement in the alveolar/arterial oxygen tension difference was lost but could be restored by recommencing CPPV. The effect of CPPV on the radiological appearances was not checked in every case. However, in cases where X-ray control was available CPPV did not cause any

![Fig. 1](https://academic.oup.com/bja/article-abstract/45/10/1070/320121/45101070)
significant improvement in the appearances of the lung fields. Arterial blood pressure was not significantly affected by any of the manoeuvres. In all patients except one, the pulmonary rales disappeared 15 minutes or so after CPPV was commenced.

Table II shows haemodynamic findings in patients 7 and 8.

**DISCUSSION**

When CPPV improves the alveolar/arterial oxygen tension difference, several factors are thought to be important, notably the increase in functional residual capacity and the greater degree of alveolar distension in the expiratory phase, which avoids alveolar collapse and improves gas exchange.

In acute pulmonary oedema, an increase in the intra-alveolar pressure is likely to oppose the passage of fluid from the capillaries into the alveoli and this reduction of the alveolar capillary barrier will lead to an improved transfer of oxygen (Russell, Morgan and Lumley, 1971).

Increasing the end-expiratory pressure will increase mean alveolar pressure which will increase alveolar oxygen tension directly. In terms of the alveolar/arterial oxygen tension difference, this effect is more marked when the concentrations of oxygen supplied in the inspired gas are high.

Two groups can be distinguished among the patients in this study. The first group consists of the patients 1, 2, 3 and 5 in whom the alveolar/arterial oxygen tension gradient decreased markedly with an applied end-expiratory pressure of $+10 \text{ cm H}_2\text{O}$. In three of these patients there was a primary cardiac cause for their pulmonary oedema although the best result (patient 5) was obtained in a patient who had inhaled toxic material. The second group, consisting of patients 4, 6, 7 and 8, showed no improvement with the provision of an end-expiratory pressure of $+10 \text{ cm H}_2\text{O}$. Patient 4 was considered on clinical grounds to have acute pulmonary oedema but had massive confluent pneumonia at postmortem examination. Patient 6 was suffering from acute myocardial infarction associated with severe shock. It is possible that he had a highly elevated left atrial pressure and that the end-expiratory pressure of $+10 \text{ cm H}_2\text{O}$ was insufficient to counterbalance it. However, a very poor haemodynamic condition made us reluctant to elevate the end-expiratory pressure further. Postmortem examination revealed total occlusion of both main coronary arteries. In cases 7 and 8, the alveolar/arterial oxygen tension difference responded to higher end-expiratory pressures. However, the response of these patients to the increased pressures was different. Patient 7, suffering from acute pulmonary oedema secondary to aspiration of gastric juice, showed a striking reduction in $Q_s/Q_t$ (fig. 2), the arterial/venous oxygen content difference remaining stable throughout the study period. Patient 8, suffering from acute pulmonary oedema secondary to mitral and aortic valve disease and in whom the left atrial pressure was 80 mm Hg, showed a reduction in $Q_s/Q_t$ but a 23% increase in arterial/venous oxygen content difference. In cases 1 to 6, the cardiovascular effects of CPPV were assessed only in terms of the arterial blood pressure measured with the sphygmomanometer. No striking changes were observed. In case 8, in whom the arterial/venous oxygen content difference increased, arterial pressure was measured directly, although the measurements showed no important change. We consider that there was a reduction in cardiac output of which arterial blood pressure measurement gave no indication (Philbin, Patterson and Baratz, 1972).

This small series shows that the effect of CPPV in the management of pulmonary oedema is variable and that it is not easy to predict which patients will
Fig. 2. Changes in shunt ratio (Qs/Qt) and arterial/venous oxygen content difference (vol/100 ml) during CPPV using different end-expiratory pressures (EP). PAP=pulmonary artery pressure. (A) data for patient 7; (B) data for patient 8.
benefit from the therapy. Moreover, it underlines the importance of careful measurement of pulmonary gas exchange together with the effect of the manoeuvre on the cardiac output (Colgan, Barrow and Fanning, 1971; Colgan and Marocco, 1972).

REFERENCES

NORTH-EAST OF SCOTLAND SOCIETY OF ANAESTHETISTS

Syllabus 1973-74

1973

THURSDAY, NOVEMBER 29 (Aberdeen)
“A New Look Re-visited”, Professor J. Parkhouse.

1974

THURSDAY, MARCH 21 (Dundee)
“Factors affecting drug action with special reference to the muscle relaxants”, Dr Stanley Feldman.

THURSDAY, MAY 16 (Stracathro)
ANNUAL GENERAL MEETING. Presidential Address: Dr G. S. Robertson.

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Hon. Secretary: Dr EDITH BEVERIDGE, Department of Anaesthesia, Royal Infirmary, Aberdeen.