THE EFFECT OF POSTOPERATIVE ARTIFICIAL VENTILATION ON ARTERIAL BLOOD OXYGENATION

D. R. BEVAN, N. O. JONES, JEAN LUMLEY AND J. NORMAN

SUMMARY

Arterial blood samples were taken at hourly intervals for three hours from ten patients who were artificially ventilated in the immediate postoperative period following mitral valve replacement. Constant minute volume ventilation was used without periodic hyperinflation. Over the three-hour period the arterial blood oxygen tension rose slightly. It was concluded that constant volume ventilation was not associated with a progressive fall in the arterial blood oxygen tension in these circumstances.

Bendixen, Hedley-Whyte and Laver (1963) reported that controlled ventilation during anaesthesia led to a progressive fall in the arterial oxygen tension (PaO₂) and that this fall could be abolished by periodic hyperinflations of the lungs. Other workers have not confirmed these observations (Askrog et al., 1964; Sykes, Young and Robinson, 1965; Lumley, Morgan and Sykes 1969; and Morgan, Lumley and Sykes, 1970). Nevertheless the hypothesis proposed by Bendixen, that periodic "sighs" are necessary to prevent progressive hypoxaemia, has been extended to patients requiring prolonged intermittent positive pressure ventilation (IPPV). In order to provide this facility, more complex ventilators have been introduced which incorporate "sighing" mechanisms.

It has been our impression in patients needing IPPV that a fall in PaO₂ in the absence of demonstrable cardiorespiratory changes is unusual. The study which we report was designed to determine the effect of IPPV with a constant minute volume on the PaO₂ of patient who were not anaesthetized. No "sighs" were used.

METHODS

The studies were carried out on ten patients (3 male and 7 female) who had undergone cardiopulmonary bypass for mitral valve replacement with Starr-Edwards prostheses. Their ages ranged from 18 to 67 years. They had varying amounts of preoperative lung involvement associated with the rheumatic valve lesions (Arnott, 1963) but no other significant lung pathology. Anaesthesia for the operation consisted of a thiopentone, nitrous oxide, oxygen, relaxant sequence with incremental doses of an analgesic as required. A radial artery cannula was inserted percutaneously after induction of anaesthesia and was kept patent for 24-48 hours postoperatively.

All patients were rousable on leaving the operating theatre. Studies were commenced when the arterial blood pressure, the heart rate and the central venous pressure appeared stable, usually within one hour of leaving theatre. The patients were disturbed as little as possible: aspiration of tracheal secretions and other manoeuvres being performed only when necessary. Analgesia was provided by papaveretum (4-8 mg i.v.) when needed.

Each patient was intubated and ventilated by means of machines set to deliver a constant minute volume (Cape or Barnett Mk II ventilators) at a fixed rate of between 14 and 20 breaths/min. The circuit delivering the inspired gas—approximately 100 per cent oxygen—to the patient was modified by including a pressure-operated non-rebreathing collect valve to separate the expired gases from the inspired gases compressed in the ventilator tubing (Sykes, 1969). The expired gases were passed through a low resistance mixing unit (Sykes, 1968) to allow collection of mixed expired gas samples. The resistance to expiration did not exceed 2 cm of water.

The tidal volume was adjusted initially to maintain an end-tidal carbon dioxide concentration of between 5 and 6 per cent. This volume was not changed subsequently during the study. An initial set of samples was obtained and further samples were obtained at hourly intervals for the next three hours.

Inspired gas samples were collected from a sampling port in the inspiratory line before the collect valve and mixed expired gas samples were taken from the mixing unit. 100-ml oiled glass-and-metal syringes were used, each syringe being washed out with two 100-ml samples before the third 100-ml sample was retained for analysis. The oxygen concentrations were measured using a Servomex paramagnetic oxygen analyser* (model DCL 101 Mk II). The mixed expired carbon dioxide concentration was measured using an infrared analyser† (Hartman-Braun model URAS 4). The expired gas temperature was recorded as was the barometric pressure.

An arterial blood sample was taken during the period when the expired gas was being sampled. The oxygen tension was measured using an oxygen electrode‡ (Radiometer model E5406) and the output read from a pH meter (Radiometer model pH 27). Both the electrode and the paramagnetic analyser were calibrated with "white spot" nitrogen, room air and 100 per cent oxygen.

The blood carbon dioxide tension was determined using the micro-equilibration technique of Astrup (1959), employing the Radiometer micro pH electrode equipment standardised with buffers prepared to the National Bureau of Standards’ specifications at pH values of 6.840 and 7.382. The infrared carbon dioxide analyser was calibrated with mixtures of carbon dioxide in oxygen whose carbon dioxide concentrations were determined by analysis using the Haldane apparatus.

The accuracies for these analyses were (±1 SD) gas oxygen concentrations, ±0.1 %/v/v; gas carbon dioxide concentrations, ±0.1 %/v/v; blood oxygen tension, ±3 mm Hg and blood carbon dioxide tension, ±1 mm Hg.

The factors of Kelman and Nunn (1966) were used to calculate the blood oxygen and carbon dioxide at the patient's body temperature.

**CALCULATIONS**

The partial pressure of oxygen and carbon dioxide in the inspired and expired gases were calculated:

\[
\begin{align*}
\text{P}_{1O2} &= \text{Fi}_{O2} \times \left( \text{PB} - \text{P}_{H2O} \right) \\
\text{PE}_{O2} &= \text{FE}_{CO2} \times \left( \text{PB} - \text{P}_{H2O} \right) \\
\text{PE}_{CO2} &= \text{FE}_{CO2} \times \left( \text{PB} - \text{P}_{H2O} \right)
\end{align*}
\]

when \( \text{PB} \) = Barometric Pressure

and \( \text{P}_{H2O} \) = saturated water vapour pressure at body temperature.

The ideal alveolar oxygen partial pressure was calculated from:

\[
\text{P}_{A02} = \text{P}_{O2} - \frac{\text{Pa}_{O2} - \text{PE}_{O2}}{\text{PE}_{CO2}}
\]

where \( \text{Pa}_{CO2} = \) arterial carbon dioxide tension.

The alveolar to arterial oxygen tension difference ((A-a)Po2_diff) was calculated by subtraction. The physiological dead space/tidal volume ratio (VD/VT) was calculated:

\[
\text{VD/VT} = \frac{\text{Pa}_{CO2} - \text{PE}_{CO2}}{\text{Pa}_{CO2}} \times 100\%
\]

No correction was made for the dead-space of the apparatus used.

**RESULTS**

The results of the studies in the ten patients are summarised in table 1.

The inspired oxygen tension, the arterial carbon dioxide tension and the calculated ideal alveolar oxygen tension did not change significantly during the course of the study. The dead space/tidal volume ratio was high with a mean initial value of 49.7 per cent but again did not change significantly.

### Table 1. Respiratory parameters in 10 patients receiving IPPV after open-heart surgery. (mean values ± one standard deviation)

<table>
<thead>
<tr>
<th>Sample</th>
<th>( \text{P}_{1O2} ) (mm Hg)</th>
<th>( \text{PE}_{O2} ) (mm Hg)</th>
<th>( \text{Pa}_{O2} ) (mm Hg)</th>
<th>( \text{A-a} \text{P}_{O2} \text{diff} ) (mm Hg)</th>
<th>( \text{Pa}_{CO2} ) (mm Hg)</th>
<th>( \text{PE}_{CO2} ) (mm Hg)</th>
<th>( \text{VD/VT} ) %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>689.9</td>
<td>665.0</td>
<td>639.2</td>
<td>368.6</td>
<td>267.6</td>
<td>44.3</td>
<td>21.4</td>
</tr>
<tr>
<td>1 hour</td>
<td>689.6 ± 32.5</td>
<td>664.1</td>
<td>642.7</td>
<td>392.1</td>
<td>250.6</td>
<td>39.6</td>
<td>21.2</td>
</tr>
<tr>
<td>2 hour</td>
<td>689.0 ± 30.5</td>
<td>664.4</td>
<td>642.0</td>
<td>397.4</td>
<td>244.6</td>
<td>37.8</td>
<td>20.1</td>
</tr>
<tr>
<td>3 hour</td>
<td>686.0 ± 30.5</td>
<td>664.9</td>
<td>640.8</td>
<td>402.9</td>
<td>237.9</td>
<td>39.0</td>
<td>19.6</td>
</tr>
</tbody>
</table>

*Servomex Controls Ltd., Crowborough, Sussex.
† Godart C.P.I. Ltd., Rochester, Kent.
‡ Radiometer Ltd., Copenhagen, Denmark.
EFFECT OF POSTOPERATIVE ARTIFICIAL VENTILATION

The relation between duration of ventilation and \( P_{aO_2} \) in each patient.

The relation between duration of ventilation and \((A-a)P_{O_2}\)diff in each patient.

\[
P_{aO_2} \text{ mm Hg}
\]

\[
A-a \text{ DO}_2 \text{ mm Hg}
\]

0 1 2 3

Time (hr)

0 1 2 3

Time (hr)

Fig. 1. The relation between duration of ventilation and \( P_{aO_2} \) in each patient.

Fig. 2. Relation between duration of ventilation and \((A-a)P_{O_2}\)diff in each patient.

DISCUSSION

It is well recognized that during and after surgery and anaesthesia there is some impairment of the oxygenation of arterial blood (Gordh, Linderholm and Norlander, 1958; Stark and Smith, 1960; Nunn and Payne, 1962). The defect has been attributed to atelectasis which produces ventilation-perfusion imbalance and intrapulmonary shunting (Nunn and Payne, 1962). Following the work of Mead and Collier (1959), Bendixen, Hedley-Whyte and Laver (1963) showed that anaesthesia and artificial ventilation led to a progressive hypoxaemia which could be reversed by intermittent hyperinflation of the lungs. The progressive fall in \( P_{aO_2} \) with anaesthesia and artificial ventilation could not be found by Askrog and his colleagues (1964), Sykes, Young and Robinson (1965) and Lumley, Morgan and Sykes (1969). In addition, Morgan, Lumley and Sykes...
(1970) did not find any significant increase in $PaO_2$ after passive hyperinflation of the lungs.

The studies reported here were designed to determine the changes in arterial oxygenation with artificial ventilation in patients in an intensive care unit following open-heart surgery. In these patients it is usual to find a considerable $(A-a)Po_2$ diff and a large dead space/tidal volume ratio (Sykes et al., 1970). The results we found were similar. We chose to use a high inspired oxygen concentration to eliminate ventilation-perfusion inequalities as a cause of hypoxaemia. We were unable to obtain true mixed-venous blood samples in these patients and are thus unable to state if the large $(A-a) Po_2$ diff still present was due to true shunting or to a low cardiac output or a combination of these two factors.

Nevertheless the results show that constant volume ventilation in this group of patients led to a small rise in the arterial oxygen tension and a fall in the $(A-a)Po_2$ diff gradient. We cannot assess exactly the mechanisms leading to this rise but we doubt if any large increase in cardiac output were the cause, for we waited until a reasonably stable cardiovascular system was present, before beginning the studies. The studies were completed before the effects of the post-perfusion lung syndrome would be seen (Sykes et al., 1970).

To us the implication of the study is that in these patients following open-heart surgery, artificial ventilation with a constant volume ventilator and without “sighs” does not lead to a progressive fall in the oxygenation of arterial blood. It is possible that with a “sighing” mechanism arterial oxygenation would further improve but without direct evidence for this we do not see the need for such equipment in these patients.

ACKNOWLEDGEMENTS

The authors thank Mr W. P. Cleland and Professor H. H. Bentall for permission to study their patients. We also wish to thank Miss B. D. Bird and Miss F. Henderson for technical assistance, Miss J. Green for secretarial help, and the members of the Department of Medical Illustration.

REFERENCES


L’EFFET DE LA VENTILATION ARTIFICIELLE POSTOPERATOIRE SUR L’OXYGENATION DU SANG ARTERIEL

SOMMAIRE

Des échantillons de sang artériel ont été prélevés à intervalles d’une heure durant trois heures chez dix patients, artificiellement ventilés au cours de la période postopératoire immédiate après remplacement de la valve mitrale. On utilisa une ventilation à volume-minute constant sans hyperinflation périodique. La pression d’oxygène dans le sang artériel augmenta légèrement au cours de la période de trois heures. On conclut que la ventilation à volume constant ne s’associa pas dans ces circonstances à une réduction progressive de la pression d’oxygène dans le sang artériel.
EFFECT OF POSTOPERATIVE ARTIFICIAL VENTILATION

ZUSAMMENFASSUNG

INTERNATIONAL SYMPOSIUM ON
CEREBRAL CIRCULATION AND METABOLISM

An International Symposium on Cerebral Circulation and Metabolism will be held in Philadelphia, Pa. at the Marriott Hotel, June 6-9, 1973. General topics include the control of cerebral blood flow and metabolism, and the pathophysiological and chemical substrates of abnormal function in experimental and clinical pathology. The deadline for submission of abstracts is January 15, 1973.

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