VENTILATORY RESPONSES TO REBREATHING AND CARBON DIOXIDE INHALATION DURING ANAESTHESIA IN CHILDREN

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Rebreathing may occur at low fresh gas flows in anaesthetic breathing systems such as the T-piece and its modifications, and to prevent this, it has been suggested that fresh gas flows of at least 2.5 to 3 times the actual minute ventilation be used (Conway, Seeley and Barnes, 1977; Ungerer, 1978; Byrick, 1980; Nott, Walters and Norman, 1982; Lindahl, Charlton and Hatch, 1984). However, there is debate as to whether a moderate degree of rebreathing need be avoided (Dean and Keenan, 1982; Spoerel, 1983). In 1968, Kain and Nunn reported criteria for rebreathing based upon the view that carbon dioxide rebreathing was not clinically important until it occurred within alveolar ventilation. More recently, Meakin and Coates (1983) concluded, from calculations of the degree of rebreathing using a mathematical model, that fresh gas flows equal to minute ventilation would not result in clinically significant rebreathing in Mapleson D systems, according to the Kain and Nunn criteria. These criteria are given below and one of the three has to be fulfilled:

I. Minute ventilation increased by more than 10% while end-tidal carbon dioxide tension remained unchanged or increased.

II. Minute ventilation was unchanged and end-tidal CO₂ tension increased by more than 0.7 kPa.

III. Minute ventilation increased by more than

SUMMARY

In 12 spontaneously breathing intubated children (9.3–25 kg), ventilatory responses to rebreathing and to the inhalation of carbon dioxide (CO₂) were investigated during halothane anaesthesia for minor surgical procedures. A T-piece (Mapleson F system) was used, modified by the insertion of a pneumotachograph and a paediatric airway adaptor of an in-line capnograph in the patient limb. Exhaled gas was collected for determination of expired CO₂ content. Measurements were made when the fresh gas flow (FGF) was at the borderline for rebreathing (FGF₁) and during 10 min with a mean FGF 44% lower, producing a maximal inspired CO₂ (% × CO_max) of 1.45 ± 0.38% (mean ± 1 SD). Measurements were repeated 5 min after returning to a flow exceeding FGF₁ and then during CO₂ inhalation for 10 min after the addition of 1.24 ± 0.32% CO₂ (mean ± 1 SD) to this flow. During both rebreathing and CO₂ inhalation end-tidal CO₂ (E_CO₂) was unchanged and VE did not increase significantly (18%), but during CO₂ inhalation alveolar ventilation increased (P < 0.05), indicating an adequate and intact response to this level of CO₂ inhalation. Estimations of I_CO_max could be made from the expression: I_CO_max (%) = -0.7 x FGF/VE + 2.5 and FGF to minute ventilation (VE) ratios lower than 1 were found to produce I_CO_max of 1.8% or higher. Such low FGF are likely to result in rebreathing within the alveolar ventilation and are thus of clinical importance. We believe that to increase the margin of safety in anaesthetized spontaneously breathing children, FGF of at least 1.5 to 2 times VE should be used.
In the present study the ventilatory responses to rebreathing caused by low fresh gas flows and by the addition of CO₂ to inspired gas were studied during halothane anaesthesia in spontaneously breathing children. Relationships between actual minute ventilation, fresh gas flow and maximal inspired CO₂ concentrations were determined and the clinical importance of rebreathing evaluated.

PATIENTS AND METHODS

The ventilatory responses to rebreathing at low fresh gas flows (FGF) and to the inhalation of CO₂ as reflected by minute ventilation ($V_e$), tidal volume ($V_T$), respiratory rate ($f$) and end-tidal CO₂ concentration ($\epsilon'_{CO₂}$) were followed during halothane anaesthesia and surgery in 12 intubated, spontaneously breathing children. Body weights ranged from 9.3 to 25 kg and ages from 1 to 5 yr. All patients were free from cardiorespiratory disease. Further patient data are presented in table I.

Anaesthesia was induced with cyclopropane in oxygen ($F_{O_2}$ 0.5) and tracheal intubation was facilitated by the injection of suxamethonium chloride 1–1.5 mg kg⁻¹ i.v. Respiration was spontaneous throughout and anaesthesia maintained with nitrous oxide and 0.5–2.0% halothane in oxygen. For genital procedures, caudal analgesia was established after the induction of anaesthesia with 0.25% bupivacaine 0.5 ml kg⁻¹. The anaesthetic system was a modified T-piece (Mapleson F system) (fig. 1). Arterial pressure and heart rate were measured automatically at regular intervals (Dinamap 850, Applied Medical Research, Tampa, Florida, U.S.A.). A heated pneumotachograph (Fleisch No. 0) and the paediatric airway adaptor of an in-line capnograph (Hewlett-

![Fig. 1. The anaesthetic system used. CO₂ indicates the position of the in-line capnograph and $\dot{V}$ the position of the pneumotachograph.](image-url)
Packard, 14360 A) were placed in the patient limb of the T-piece. $V_E$ was measured by electrical integration of the flow signal from a differential pressure manometer (Validyne MP45-1-871, range $\pm 2$ cm H$_2$O). $\dot{V}CO_2$ was measured from the recorded CO$_2$ signal and an easily identified plateau phase was required in all cases. The response time of the capnograph was 0.05 s which, from a technical point of view, allowed registration of end-tidal CO$_2$ values from a plateau phase at respiratory rates well above those seen in this study. Expired gas passed to a dry gas meter (Standard Gas Meter, AB Nordgas, Stockholm, Sweden) and then to a three-way valve from which a timed collection (over 5 min) of a measured volume of gas could be made into a Douglas bag. The percentage of CO$_2$ in expired gas ($F_{ECO_2}$) was measured by the capnograph. Flow, volume and CO$_2$ signals were recorded using a u.v. recorder (S.E. Labs (EMI) Ltd, S.E. 3006). Volume was calibrated with a 50-ml syringe. The capnograph was calibrated using certified gas mixtures containing CO$_2$ within the measuring range. The deadspace of the system was 6 ml and the respiratory resistance of the system, pneumotachograph and in-line paediatric airway adaptor of the capnograph was 10 cm H$_2$O litre$^{-1}$ s$^{-1}$ with continuous flow rates up to 10 litre min$^{-1}$.

**Calculations**

$V_E$, $V_T$, alveolar ventilation ($V_A$), deadspace volume per breath ($V_D$) and carbon dioxide elimination ($\dot{V}CO_2$) were corrected to body temperature and pressure saturated (BTPS). The following formulae were used:

$$\dot{V}CO_2 (ml \; min^{-1}) = \frac{gas \; collection \; \dot{V}E \times (F_{ECO_2} - F_{ICO_2})}{100}$$

where $P_{ICO_2}$ is the fraction of CO$_2$ in inspired gas and $ICO_2$ is the mean inspired CO$_2$ concentration. During rebreathing the mean inspired CO$_2$ concentration was not known, therefore calculations of $VA$ and $VD$ could not be made.

Evaluation of the clinical significance of rebreathing was made according to the criteria detailed by Kain and Nunn (1968) for evidence of alveolar rebreathing (see introduction and table III).

**Plan of investigation**

The anaesthetist in charge of the patient was not involved in the study, and was at liberty to control the depth of anaesthesia. No measurements were made until anaesthesia was considered clinically stable, the halothane concentration constant and at least 20 min had elapsed since induction.

Measurements of $\dot{V}E$, $V_T$, $FE_{CO_2}$, $ICO_2$, $E'_{CO_2}$, arterial pressure and heart rate were then made with no rebreathing using a fresh gas flow (FGF) which was just great enough to avoid rebreathing (FGF$_T$). This borderline for rebreathing of CO$_2$ was determined in each patient by the use of the capnograph at gradually decreasing FGF. The FGF setting before the one during which CO$_2$ in inspired gas was first detected (FGF$_T$) was used during measurements at “no rebreathing”.

Then, FGF was decreased to achieve a maximal inspired CO$_2$ concentration per breath ($ICO_2_{max}$)(%) of about 1.5%. Measurements of $\dot{V}E$, $V_T$, $f$, $ICO_2$, $E'_{CO_2}$, arterial pressure and heart rate were obtained just before the change of FGF setting and then every minute during a 5-min.

**Table II.** Mean values ± 1 SD for arterial pressure, heart rate and respiratory rate before (0), 2, 5 and 10 min of rebreathing or CO$_2$ inhalation

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<td><strong>Arterial pressure (mm Hg)</strong></td>
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<tr>
<td>Systolic</td>
<td>101±18</td>
<td>96±9</td>
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<td>96±12</td>
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<td>Diastolic</td>
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<td>48±7</td>
<td>49±9</td>
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<td><strong>Heart rate (beat min$^{-1}$)</strong></td>
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<td>118±21</td>
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<td><strong>Respiratory rate (b.p.m.)</strong></td>
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<td><strong>CO$_2$ inhalation (min)</strong></td>
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<tr>
<td>0</td>
<td>97±19</td>
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<td>95±11</td>
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<td>49±7</td>
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<td>5</td>
<td>116±20</td>
<td>114±18</td>
<td>116±16</td>
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<td>10</td>
<td>27±9</td>
<td>28±9</td>
<td>28±9</td>
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</table>
REBREATHEING AND CO₂ INHALATION IN CHILDREN

**Fig. 2.** The pattern of CO₂ during inspiration investigated in this study. Values are mean ± 1 SD I_{CO₂ max} and mean I_{CO₂}.

Statistics

Mean values and standard deviation (SD) were calculated. The results were evaluated by the use of paired t test applied to mean data.

RESULTS

Arterial pressure, and heart and respiratory rates were virtually unchanged during periods of rebreathing and CO₂ inhalation (table II). The two CO₂ exposures during inspiration are presented schematically in figure 2. The mean values (±1 SD) of I_{CO₂ max} and of I_{CO₂} were 1.45 ± 0.38% and 1.24 ± 0.32% respectively.

**Rebreathing**

The I_{CO₂ max} (%) given in figure 2 was achieved after the FGF had been decreased by 44% from (mean ± 1 SD) 5.25 ± 1.58 litre min⁻¹ to 2.96 ± 0.59 litre min⁻¹ (P < 0.001). Individual decreases in FGF are illustrated in figure 3 and ranged from 0.7 to 2.5 times the measured minute ventilation. The relationship between the I_{CO₂ max} (%) and the FGF/V̇E ratio was described by the regression equation: I_{CO₂ max} = -0.71 . FGF/V̇E + 2.49 with a coefficient of correlation of -0.79 (fig. 4).

There was considerable variation in response to this level of rebreathing. In no child did V̇E increase significantly and in several no increases were seen in V̇E and V̇T (fig. 5). After 10 min of rebreathing mean V̇E and V̇T had increased by 18 and 24%, respectively (ns). Mean respiratory rate and V̇E were unchanged (fig. 6).

Only one child fulfilled the criteria of Kain and Nunn for significant rebreathing (table III, fig. 10).
Inhalation of carbon dioxide

The ventilatory response to the inhalation of 1.24 ± 0.32% carbon dioxide (mean ± 1 SD, fig. 2) was similar to that found during rebreathing at low FGF. The seven patients who responded to CO₂ did so within 2 min. In five patients there were no increases in \( \dot{V}E \) and \( VT \) (fig. 7). \( e'_\text{CO}_2 \) concentrations remained unchanged at this inspired CO₂ concentration. After 10 min of CO₂ inhalation mean values of \( \dot{V}E \) and \( VT \) were increased by 18 and 19%, respectively. Respiratory rate and \( e'_\text{CO}_2 \) were unchanged (fig. 8).

Carbon dioxide elimination (\( \dot{V}_{CO_2} \)) was unchanged during the inhalation of carbon dioxide.
This was achieved by a higher $\dot{V}A$ which was increased by 32% (mean value ± 1 SD) from 1212 ± 231 ml min⁻¹ before to 1603 ± 450 ml min⁻¹ during CO₂ inhalation ($P < 0.05$)(fig. 9). $Vd/VT$ ratios (mean ± 1 SD) decreased by 23% from 0.31 ± 0.13 before to 0.24 ± 0.12 during CO₂ inhalation (ns) (fig. 9).

In spite of the inhalation of carbon dioxide (that is CO₂ present also early during inspiration), the criteria of Kain and Nunn for clinically significant rebreathing were fulfilled in only six of the 12 children (fig. 10).

**DISCUSSION**

At low fresh gas flows in anaesthetic systems such as Mapleson D, E and F (Willis, Pender and Mapleson, 1975) rebreathing appears late during inspiration. Therefore, apparatus and patient deadspaces could be the only parts exposed to inspired CO₂. Under these circumstances rebreathing would not create any CO₂ load as far as the patient is concerned. There are, however, difficulties in calculating the amount of CO₂ that reaches the alveoli so the clinical importance of

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**TABLE III. Evaluation of rebreathing according to criteria for rebreathing set by Kain and Nunn (1968): I. $\dot{V}E > 10\%$, $PE_{CO_2}$ unchanged; II. $\dot{V}E$ unchanged, $PE_{CO_2} > 0.7$ kPa; III. $\dot{V}E > 3\%$, $PE_{CO_2} > 0.3$ kPa. $PE_{CO_2} = \text{end-tidal CO}_2 \text{ tension (kPa).}$

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>FGFᵢ</th>
<th>FGFᵢ₀ (°)</th>
<th>$ICO_{max}$ (%)</th>
<th>$\dot{V}E$ change (%)</th>
<th>$PE_{CO_2}$ change (kPa)</th>
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<td>3.5</td>
<td>1.67</td>
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<td>2</td>
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<td>3</td>
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<td>-0.13</td>
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<td>1.45</td>
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<td>-0.98</td>
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<td>6</td>
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<td>+0.20</td>
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<td>±0</td>
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<tr>
<td>12</td>
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<td>1.25</td>
<td>+6</td>
<td>+0.17</td>
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FGFᵢ = the fresh gas flow borderline for rebreathing and FGFᵢ₀ = the low fresh gas flow at which rebreathing of about 1.5% occurred. + Indicates criterion fulfilled and 0 not fulfilled.
rebreathing has still to be judged from ventilatory responses in patients, and criteria for the evaluation of ventilatory changes have been described by Kain and Nunn (1968).

At FGF/VE ratios of 2 with spontaneous breathing in Mapleson D, E and F systems rebreathing is known to occur in most patients (Lindahl, Charlton and Hatch, 1984). According to the regression equation for $i_{CO_2 \text{max}}$ in this study the level of rebreathing at 2 FGF/VE would be 1.1% CO$_2$. In the study by Meakins and Coates (1983) however, rebreathing did not occur at 2 FGF/VE. This discrepancy was somewhat surprising since their theoretical mathematical calculations were correctly based on measurements in anaesthetized young patients; but their FGF to VE ratios were derived from the Engström and Herzog (1959) nomogram for VE estimations—published in 1959 for the setting of artificial ventilation which gave minute ventilations which were 30–40% higher compared with calculations from a recently published regression equation for VE in anaesthetized spontaneously breathing children (Lindahl, Hulse and Hatch, 1984). If lower VE had been used by Meakins and Coates, it is likely that rebreathing would have occurred at higher FGF/VE ratios.

When FGF was reduced by more than 40% from that precisely required to avoid rebreathing, statistically significant ventilatory changes were not found. One individual demonstrated a marked ventilatory response with an increase in VE of over 50% at an $i_{CO_2 \text{max}}$ of 2.15% CO$_2$, which was the highest seen in the sample, but, as the end-tidal CO$_2$ tension decreased by almost 1 kPa (patient
Fig. 9. Carbon dioxide output ($V_{\text{CO}_2}$), alveolar ventilation ($V_A$), deadspace volume ($V_D$) and $V_D/V_T$ ratio before (unfilled columns) and after 10 min of $1.24 \pm 0.32\%$ CO\textsubscript{2} inhalation (hatched columns).

Fig. 10. Relations between measured changes of minute ventilation ($\Delta V_E$) in per cent and of end-tidal CO\textsubscript{2} tensions ($\Delta P_{e\text{CO}_2}$) in kPa during rebreathing ($1\text{CO}_2\max : 1.45 \pm 0.38\%$, left diagram) and CO\textsubscript{2} inhalation ($t_{\text{CO}_2} : 1.24 \pm 0.32\%$, right diagram). Filled circles represent children who did fulfil the criteria of rebreathing set by Kain and Nunn (1968), and open circles, those patients who did not.

No. 5, table III) Kain and Nunn's criteria for clinically important rebreathing were not fulfilled. Thus, it was difficult to find evidence of rebreathing within alveolar ventilation. As rebreathing occurs late in inspiration, the volume of rebreathed alveolar gas may stay within the deadspace and hence, not effect alveolar ventilation. Even so, rebreathing within alveolar gas has to be at least in the order of 1-1.25\% CO\textsubscript{2} if a uniform ventilatory response was to be expected, since the ventilatory responses during CO\textsubscript{2} inhalation in this study were as variable as the responses during rebreathing. The six patients fulfilling the criteria during CO\textsubscript{2} inhalation were surprisingly few, as inhaled CO\textsubscript{2} always reaches the alveoli but were, on the other hand, sufficient to indicate that higher levels of alveolar CO\textsubscript{2} rebreathing cannot be accepted clinically.
The similarity in end-tidal carbon dioxide concentrations after 10 min of CO₂-free breathing, rebreathing or CO₂ inhalation suggests the presence of adequate compensatory respiratory mechanisms balancing CO₂ homeostasis in the body. The compensatory mechanisms during CO₂ inhalation did not primarily result in a uniform increase in minute ventilation. Alveolar ventilation was, however, increased (P < 0.05), indicating an adequate adaptation during CO₂ breathing at the depth of anaesthesia used in this study. An increased \( V_A \) that was explained most probably by the higher \( \dot{V}_T \) noted during CO₂ breathing (which is known to improve ventilation/perfusion ratio (Rose and Froese, 1980), that is to increase efficiency of ventilation) explained the unchanged \( \dot{C}O_2 \) concentration before and during CO₂ inhalation found in this study.

To achieve a consistent response in \( \dot{V}_E \) during rebreathing \( \dot{C}O_2 \max \) of 1.8% had to be reached, while lower concentrations resulted in variable responses (table III). According to the regression equation for the relationship between \( \dot{C}O_2 \max \) and \( FGF/\dot{V}_E \), FGF settings lower than \( \dot{V}_E \) are likely to result in rebreathing within the alveolar ventilation. Such low fresh gas flows are at the utmost limit which can be tolerated in clinical practice using these breathing systems. During anaesthesia many things can go wrong; one of them being insufficient CO₂ elimination. Since, CO₂ is a factor which we know how to eliminate from inspired gas, fresh gas flows of at least 1.5 to 2 times \( \dot{V}_E \) must be used to keep the maximal inspired CO₂ concentration within acceptable limits to achieve a necessary margin of safety in anaesthetized spontaneously breathing children. If the total elimination of rebreathing is desired, a fresh gas flow of 3.5 times \( \dot{V}_E \) is required, according to the regression equation calculated in this study.

It was concluded that at an \( I_{CO_2 \max} \) of 1.45 ± 0.38% resulting from low fresh gas flows, \( \dot{V}_E \) increased by 18% (ns) and \( \dot{C}O_2 \) was unchanged. Criteria for clinically important CO₂ rebreathing were only fulfilled in one patient. During the inhalation of 1.24 ± 0.32% CO₂, \( \dot{V}_E \) was also increased by 18% (ns) and \( \dot{C}O_2 \) was unchanged. Although, \( \dot{V}_E \) did not increase significantly during the inhalation of 1.24 ± 0.32% CO₂, \( V_A \) did, indicating an intact and adequate ventilatory response to this level of CO₂ breathing during halothane anaesthesia. Estimations of \( I_{CO_2 \max} \) could be made from the expression: \( I_{CO_2 \max} = -0.7 \times \dot{F}GF/\dot{V}_E + 2.5 \) and FGF to \( \dot{V}_E \) ratios lower than 1 are likely to result in clinically significant rebreathing with \( I_{CO_2 \max} \) concentrations of more than 1.8%. To avoid alveolar rebreathing, FGF settings of at least 1.5 to 2 times \( \dot{V}_E \) must be used in these anaesthetic systems.

ACKNOWLEDGEMENTS

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