AN EVALUATION OF REBREATHING WITH THE BAIN SYSTEM DURING ANAESTHESIA WITH SPONTANEOUS VENTILATION

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SUMMARY

Data from 12 anaesthetized patients breathing spontaneously from the Bain system were used to calculate the degree of rebreathing occurring when the fresh gas flowrate (VF) was equal to 2, 1 and 0.7 times the estimated normal minute ventilation (V\text{\text{exo}}). Measurements of the expired minute volume (\text{\text{VE}}) and end-tidal carbon dioxide tension (\text{PE'CO}_2) were made to determine the effects of this rebreathing. No rebreathing occurred when VF was equal to twice V\text{\text{exo}}. When VF was equal to V\text{\text{exo}} rebreathing was usually small in amount and produced no changes in \text{\text{VE}} or \text{PE'CO}_2. Changes attributable to rebreathing occurred in only two patients when VF was reduced to 0.7 V\text{\text{exo}}. These results are explained by the presence of anaesthesia-induced ventilatory depression and favourable changes occurring in the respiratory wave forms in the majority of patients studied. In some patients, greater values of \text{\text{VE}} and rebreathing occurred in response to strong surgical stimulation. The net result of increased ventilation in these patients was a decrease in \text{PE'CO}_2. It is concluded that during anaesthesia, when the Bain system is used with VF equal to V\text{\text{exo}}, any increase in \text{PE'CO}_2 which may result from rebreathing is likely to be small and seldom of clinical importance.

The Bain modification of the Mapleson D and E systems (Bain and Spoerel, 1972) has been advocated as a universal anaesthetic breathing system for use with both spontaneous and controlled ventilation (Henville and Adams, 1976). The fresh gas flow rate (VF) recommended in the literature supplied with the Bain circuit (Bain Breathing Circuit, Respiratory Care Inc., Arlington Heights, Illinois, U.S.A.) for use during spontaneous respiration, is 100 ml kg\text{\text{-1}} min\text{\text{-1}}, approximately equal to the normal minute volume of an adult. This is less than is required to prevent rebreathing of expired gas with this system in conscious volunteers (Conway, Seeley and Barnes, 1977; Ungerer, 1978), and has prompted a controversy regarding the possible effects of rebreathing in anaesthetized patients.

In healthy adults under light anaesthesia for dental extractions (VF 100 ml kg\text{\text{-1}} min\text{\text{-1}}) (Spoerel, Aitken and Bain, 1978), the response to rebreathing was hyperventilation with maintenance of PacO2. Similar results were obtained using the Bain circuit with VF equal to the normal minute volume in children (Soliman and Laberge, 1978). Other authors questioned the ability of all subjects to compensate for rebreathing during anaesthesia, when respiratory drive may be depressed. Using a lung model with a sinusoidal respiratory pattern, Rose, Byrick and Froese (1978) showed that, with a normal volume (6 litre min\text{\text{-1}}), rebreathing of expired gas produced an increase in alveolar carbon dioxide when the Bain system was used with the recommended VF of 100 ml kg\text{\text{-1}} min\text{\text{-1}}. Normocapnia only occurred if respiratory minute volume was increased substantially. These findings were later confirmed by Byrick (1980) in a study of lightly anaesthetized adults breathing from the Bain system. To eliminate the risk of potentially dangerous hypercarbia resulting from rebreathing from the Bain system during anaesthesia with spontaneous ventilation, these authors recommended VF of 200–300 ml kg\text{\text{-1}} min\text{\text{-1}} in adults and two to three times normal minute ventilation in children (Steward, 1979). In the present study data from anaesthetized children breathing from the Bain system were used to construct a mathematical model to calculate the degree of rebreathing occurring at various values of VF. Expired minute volume (\text{\text{VE}}) and end-tidal carbon dioxide (\text{PE'CO}_2) were measured to determine the effects of this rebreathing.
PATIENTS AND METHODS

Twelve healthy children aged 6–16 yr were studied. Parental consent was obtained and the study was approved by the Ethics Committee of the institution. The age, weight and height of each child were used to calculate the normal minute ventilation ($V_{\text{to}}$) from a nomogram (Engström and Herzog, 1959) (table I). Six of the patients were premedicated with pentobarbitone, morphine and hyoscine and the remainder were unpremedicated. In all patients anaesthesia was induced with thiopentone followed by suxamethonium to facilitate tracheal intubation. Anaesthesia was maintained with a mixture of 66% nitrous oxide in oxygen with 0.5–1.5% halothane. The inspired halothane concentration was then decreased to twice $V_{\text{to}}$ and finally to 0.7 $V_{\text{to}}$.

Respiratory flow was measured by means of a heated pneumotachograph (Fleisch No. 2) attached to the machine end of the Bain reservoir tube, replacing the breathing bag and pop-off valve. A length of tubing was added distally to prevent less dense room air from entering the pneumotachograph. The signal from the pneumotachograph was biased electronically to eliminate the effects of constant $V_F$ from the anaesthetic machine, and integrated electronically to obtain the tidal volume ($V_T$) and $V'E$. Measurements of $V'E$ were standardized for the size of the child by dividing by $V_{\text{to}}$. The accuracy of the anaesthetic machine flowmeters was assessed by passing gas through a dry gas meter (Parkinson-Cowan). Respiratory gases were sampled continuously at the connector end of the tracheal tube and analysed for carbon dioxide using an infra-red carbon dioxide analyser (Beckman LB II).

The carbon dioxide analyser was calibrated before and after each study with a mixture of carbon dioxide in oxygen, and observed values were adjusted to eliminate the broadening effect of nitrous oxide. Sampling resulted in the loss of 300 ml min$^{-1}$ from the system which was replaced at the flowmeters. All data were recorded on a four-channel pen recorder (Brush). Initially, $V_F$ was set at twice $V_{\text{to}}$, $V_F$ was then decreased to twice $V_{\text{to}}$ and finally to 0.7 $V_{\text{to}}$. Observations were made after 20 min equilibration at each test flowrate.

To calculate the volume of alveolar gas which was rebreathed at each $V_F$, a mathematical model was constructed using a modification of the method previously described by Willis, Pender and Maple son (1975). The model was constructed using data from the patient's pneumotachograph traces. The equations and all of the waveforms used in the model appear in figure 1. In each case inspiration was represented by a sinusoidal function (fig. 1A). To accommodate variations in the expiratory waveform, a two-part model was used. The first part of expiration was represented by a quarter sine wave and the second part was modelled as a sinusoidal, a linear, or an exponential function (fig. 1A, B, C).

Figure 2 illustrates how the respiratory model was used to calculate the degree of rebreathing occurring at different values of $V_F$. Three possible respiratory waveforms are shown. With the Bain system, whenever inspiratory flow exceeds $V_F$ the difference is made up of gas drawn from the reservoir tube. This volume of gas is represented in the model by areas A and B + C. The first part of this gas, represented by area A, is fresh gas that entered the reservoir tube during inspiration when inspiratory

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Sex</th>
<th>$S_A$ (m$^2$)</th>
<th>$V_{\text{to}}$ (litre min$^{-1}$)</th>
<th>Premed.</th>
<th>$F_{\text{hal}}$ (%)</th>
<th>Operation</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>34</td>
<td>137</td>
<td>F</td>
<td>1.15</td>
<td>4.5</td>
<td>+</td>
<td>1.0</td>
<td>Bilateral Mitchell osteotomy</td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>30</td>
<td>129</td>
<td>M</td>
<td>1.03</td>
<td>4.4</td>
<td>–</td>
<td>1.0</td>
<td>Dental restoration and extraction</td>
</tr>
<tr>
<td>3</td>
<td>16</td>
<td>71</td>
<td>178</td>
<td>M</td>
<td>1.9</td>
<td>7.6</td>
<td>–</td>
<td>1.5</td>
<td>Exploration of wrist and forearm</td>
</tr>
<tr>
<td>4</td>
<td>13</td>
<td>36</td>
<td>150</td>
<td>M</td>
<td>1.25</td>
<td>5.1</td>
<td>–</td>
<td>1.5</td>
<td>Extraction of four impacted teeth</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>21</td>
<td>113</td>
<td>F</td>
<td>0.8</td>
<td>3.6</td>
<td>+</td>
<td>0.5</td>
<td>Lt. mastoidectomy</td>
</tr>
<tr>
<td>6</td>
<td>8</td>
<td>27.2</td>
<td>115</td>
<td>F</td>
<td>0.9</td>
<td>4.1</td>
<td>+</td>
<td>0.5</td>
<td>Dental restoration</td>
</tr>
<tr>
<td>7</td>
<td>11</td>
<td>56</td>
<td>146</td>
<td>M</td>
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<td>6.0</td>
<td>–</td>
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<tr>
<td>8</td>
<td>10</td>
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<td>138</td>
<td>F</td>
<td>1.27</td>
<td>5.0</td>
<td>+</td>
<td>1.0</td>
<td>Exc. exostosis rt. lower femur</td>
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<tr>
<td>9</td>
<td>16</td>
<td>42.2</td>
<td>152</td>
<td>F</td>
<td>1.35</td>
<td>5.2</td>
<td>+</td>
<td>0.75</td>
<td>Tymanoplasty and oss. reconstruction</td>
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<tr>
<td>10</td>
<td>6</td>
<td>19.3</td>
<td>118</td>
<td>M</td>
<td>0.83</td>
<td>3.4</td>
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<td>Rt. varus derotation osteotomy</td>
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<tr>
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<td>11</td>
<td>32.0</td>
<td>135</td>
<td>M</td>
<td>1.1</td>
<td>4.7</td>
<td>–</td>
<td>1.0</td>
<td>Rt. tympanomastoidectomy</td>
</tr>
<tr>
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<td>14</td>
<td>73</td>
<td>158</td>
<td>M</td>
<td>1.75</td>
<td>7.0</td>
<td>+</td>
<td>1.5</td>
<td>Hip pinning</td>
</tr>
</tbody>
</table>

$S_A$ = surface area
A Sinusoidal Respiration

\[ V_I = \frac{V_{Ip} \sin \frac{\pi}{2} t}{T_1} \]

\[ V_{E_1} = \frac{V_{Ep} \sin \frac{\pi}{2} t}{2T_{E_1}} \]

\[ V_{E_2} = \frac{V_{Ep} \sin \frac{\pi}{2} (1-t/T_{E_2})}{2T_{E_2}} \]

B Linear Second Part of Expiration

\[ V_{E_1} = V_{Ep} \]

\[ V_{E_2} = V_{Ep} (1-t/T_{E_2}) \]

C Exponential Second Part of Expiration

\[ V_{E_1} = V_{Ep} e^{-0.694t/T_1} \]

\[ V_{E_2} = V_{Ep} e^{-0.694t/T_1} \]

Fig. 1. Equations and waveforms used to model respiration during anaesthesia. \( V_I \) = inspiratory flow at any time \( t \); \( V_{Ip} \) = peak inspiratory flow; \( V_{E_1} \) and \( V_{E_2} \) = the expiratory flow during the first and second parts of expiration respectively; \( V_{Ep} \) = peak expiratory flow. \( T_1 \) = inspiratory time; \( T_{E_1} \) and \( T_{E_2} \) = durations of the first and second parts of expiration respectively; \( T_1 \) = time taken for the exponential flow to decay to one-half of its peak value.

flow was less than \( \dot{V}_F \). This volume of gas is also represented in the model by area \( a \). The second part, represented by area \( B + C \), consists of fresh gas which entered the reservoir tube during expiration together with some expired gas. In the expiratory part of the model these volumes are represented by areas \( b \) and \( c \) respectively. The areas in various parts of the model were obtained by integration starting with area \( a \) which allowed area \( B + C \) and finally area \( c \) to be calculated. The fraction of the tidal volume taken up by rebreathed alveolar gas was obtained by dividing area \( c \) by the total area under the inspiratory curve. This fraction, expressed as a percentage, was referred to as the degree of rebreathing (\( VR \% \) \( VT \)). In order to validate the various assumptions made in constructing the model, two tests were performed. In the first, the equations used to model respiratory flow were mathematically integrated to yield a calculated value of \( VT \). This value of \( VT \) was then compared with the actual \( VT \) of the patient obtained by electronic integration of the pneumotachograph signal. In the second test, the mathematical model of respiration and the meas-
ured PE'CO₂ were used to calculate the maximum carbon dioxide tension at the connector end of the tracheal tube during inspiration (PlpCO₂). This calculated value of PlpCO₂ was compared with the actual PlpCO₂ measured by the carbon dioxide analyser. All the above calculations were performed on a digital computer (Hewlett-Packard 3000).

The effects of rebreathing were assessed by observing changes in Vₑ and PE'CO₂. One or more of the following were considered evidence of carbon dioxide rebreathing at the alveolar level (Kain and Nunn, 1968):

1. An increase in Vₑ of 10% or more not accompanied by a corresponding decrease in PE'CO₂.
2. An increase in PE'CO₂ of 0.7 kPa (5.25 mm Hg) or more not accounted for by a decrease in ventilation.
3. An increase in ventilation of 5% or more accompanied by an increase in PE'CO₂ of 0.3 kPa (2.25 mm Hg) or more.

RESULTS

Figure 3A–B shows simultaneous pneumotachogram and carbon dioxide recordings from three patients when Vₕ was equal to Vₑtot. The observed values of Vₑ and PE'CO₂ are given beneath the flow and carbon dioxide traces respectively. With increasing degrees of ventilatory depression the pneumotachograms demonstrate characteristic changes in respiratory waveform. Respiratory rate and inspiratory and expiratory peak flows are reduced, and the second part of expiration changes from sinusoidal to linear or exponential in form. The corresponding carbon dioxide traces show a progressive increase in PE'CO₂ but rebreathing of alveolar gas, indicated by the presence of carbon dioxide in the inspired gases, is decreased.

The validity of the mathematical model of respiration is shown by the close agreement between the observed and calculated values of Vₑ and PlpCO₂ in figure 4A and B. The complete data of Vₑ, PE'CO₂ and the calculated values of VR % VT for each tested Vₕ appear in table II. In all cases, VR % VT was zero when Vₕ was equal to 2 Vₑtot. No patient satisfied the criteria for rebreathing based on changes in Vₑ and PE'CO₂ when Vₕ was decreased from 2 Vₑtot to Vₑtot and only two patients satisfied these conditions when Vₕ was decreased further to 0.7 Vₑtot (patients Nos 3 and 10, table II). In two patients there was insufficient time to complete the measurements.

![Figure 3. Simultaneous pneumotachogram (A) and carbon dioxide recordings (B) from three patients when Vₕ was equal to Vₑtot. The measured values of Vₑ and PE'CO₂ are given. INSPIR. and EXP. = inspiration and expiration respectively; sine, linear and exponential = waveforms of the second part of expiration. In the carbon dioxide trace of patient 3, substantial rebreathing is indicated by the presence of a large peak of carbon dioxide in the inspired gases. With increasing ventilatory depression in patients 5 and 11, the concentration of inspired carbon dioxide is greatly reduced. Small irregularities in the flow and carbon dioxide traces, most clearly seen in the recordings from patient 11, were caused by cardiac activity.](https://academic.oup.com/bja/article-abstract/55/6/487/259247/259247)
with $V_F$ equal to 0.7 $V_{tot}$ (patients Nos 2 and 4, table II). The mean values ± 1 standard deviation of $V_E$ and $PECO_2$ at each tested $V_F$ appear in table III.

The relationship between $V_F$, $V_E$ and rebreathing is shown in figure 5, where $VR\%VT$ is plotted against $V_F/V_E$ for all values of $V_F$. It will be seen that rebreathing is inversely related to $V_F/V_E$, and that a value of $V_F/V_E$ of at least 2 was required to eliminate rebreathing in any patient studied. An
**Table III.** Mean values of minute ventilation and end-tidal carbon dioxide ± standard deviation

<table>
<thead>
<tr>
<th>Fresh gas flow rate</th>
<th>No. of patients</th>
<th>Mean $\dot{V}E/\dot{V}T$</th>
<th>$P_{E}CO_{2}$ (kPa)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$2\dot{V}<em>{T</em>{ox}}$</td>
<td>12</td>
<td>0.60 ± 0.1</td>
<td>6.8 ± 0.3</td>
</tr>
<tr>
<td>$\dot{V}<em>{T</em>{ox}}$</td>
<td>12</td>
<td>0.70 ± 0.3</td>
<td>6.7 ± 0.5</td>
</tr>
<tr>
<td>$0.7\dot{V}<em>{T</em>{ox}}$</td>
<td>10</td>
<td>0.78 ± 0.6</td>
<td>7.0 ± 0.6</td>
</tr>
</tbody>
</table>

An important corollary to this relationship is that when $VF$ is constant, $VR \% VT$ will be directly related to $VE$. This relationship is shown in figure 6, where $VR \% VT$ and $P_{E}CO_{2}$ are plotted against $VE$ when $VF$ is equal to $V_{T_{ox}}$. $P_{E}CO_{2}$ is inversely related to $VE$ and, paradoxically, an inverse relationship between $P_{E}CO_{2}$ and $VR \% VT$ is demonstrated.

**DISCUSSION**

In the present study, rebreathing was quantified by modelling the respiratory waveforms of patients breathing from the Bain system in a manner similar to that previously described by Willis, Pender and Mapleson (1975) in conscious volunteers breathing from a T-piece (modified Mapleson E). An important difference in the present study was the use of patient data to obtain a more realistic model of respiration during anaesthesia. Another important difference is that no allowance was made for anatomical deadspace in calculating the volume of alveolar gas that was rebreathed. Accordingly, a positive value of $VR \% VT$ indicated that rebreathing had taken place at a level close to the tracheal tube connector, but it did not indicate that rebreathed gas had entered the patient's alveoli.

Only two assumptions were necessary for constructing the mathematical model. First, that breathing during anaesthesia could be represented by a combination of mathematical functions. Clearly, this must lead to discrepancies between the measured respiratory waveform and the model waveform, particularly when the former was distorted by cardiac activity (patient No. 11, fig. 3A). The close agreement between measured $VT$ and calculated $VT$ (fig. 4A) suggests that the difference was small. A second assumption was that no longitudinal mixing of gases occurred in the Bain system reservoir tube, and that all rebreathed gas came from the last part of the previous expiration. The close agreement between the measured $Pl_{PCO_{2}}$ and the value of $Pl_{PCO_{2}}$ calculated from the mathematical model suggests that this assumption was valid (fig. 4B). As both assumptions necessary for the construction of the mathematical model appear to be valid, it is concluded that the model itself is valid.
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The degree of rebreathing (VR % VT) and end-tidal carbon dioxide (PE′CO2) are plotted simultaneously against normalized ventilation (VE/Vtot) when VF was equal to Vtot. The graph demonstrates a paradoxical relationship between VR % VT and PE′CO2.

and the values obtained for VR % VT correspond to the actual percentage of VT taken up by rebreathed alveolar gas.

During spontaneous ventilation the Bain system is normally used with a breathing bag and a pop-off valve attached to the distal end of the reservoir tube. In the present study these were removed in order that the pneumotachograph could be attached at a convenient distance from the patient’s head and the surgical field. Thus, the system was transformed from a modified Mapleson D system to a modified Mapleson E. With the bag and valve in situ expiratory flow would be slightly impeded, and for this reason it has been recommended that the valve should be removed when this system is use in children (Bain and Spoerel, 1972). However, there is no evidence to suggest that the presence or absence of this valve significantly alters the flow requirements of the system. On the contrary, Mapleson (1954) calculated that the flow requirements of systems D and E would be similar, and more recently Rose, Byrick and Froese (1978) could demonstrate no difference in the flow requirements of the Bain system with or without the valve when tested with a mechanical lung model. Accordingly, we believe that the results of the present study can be applied to both forms of the Bain circuit when used under the same conditions of spontaneous ventilation with low fresh gas flow rates.

In the past, different methods have been used to determine the fresh gas flow requirements of the Bain circuit during spontaneous ventilation. The criterion used by Spoerel, Aitken and Bain (1978) has been the maintenance of normal arterial carbon dioxide tension. Other authors have calculated or measured the flow rate required to prevent rebreathing with this system under a variety of conditions. Whichever method is used, the resulting flow rates will ultimately depend upon the carbon dioxide production of the patient. Since this is greater on a weight basis in children than in adults the flow requirements will be correspondingly greater. From a study of carbon dioxide production and elimination in children Bain and Spoerel (1977) calculated that the flow requirements of infants weighing 10 kg or less exceeded 200 ml kg⁻¹ min⁻¹. This gradually decreased to 100 ml kg⁻¹ min⁻¹ for children weighing 30 kg or more. These flow rates, which were recommended regardless of the mode of ventilation, are comparable to the values of Vtot in the present study.

In table II, the broken lines indicate that the criteria for alveolar rebreathing of carbon dioxide (Kain and Nunn, 1968) were satisfied in two patients (Nos 3 and 10) when VF was reduced to 0.7 Vtot. In nine patients (Nos 4–12, table II), the absence of changes in VE and PE′CO2 when VF was reduced to Vtot may be explained by the fact that VR % VT was small. Theoretically, when VF is reduced in patients breathing from a T-piece system, rebreathing starts late in inspiration (Sykes, 1968; Eger, 1974). If the volume of alveolar gas rebreathed is small, it will come to rest in the anatomical deadspace of the patient at end-inspiration, where it is of no significance. Three patients (Nos 1–3, table II) failed to satisfy the criteria for rebreathing based on changes in VE and PE′CO2 when VF was reduced to Vtot, because of an increase in the level of surgical
stimulation. In each case, large increases in $\dot{V}E$ occurred and $V R \% VT$ was high, but $PE'CO_2$ was reduced. Similar changes were observed in patients 1 and 6 (table II) when $VF$ was reduced from $V_{tot}$ to $0.7 \dot{V}_{tot}$.

From figure 5 it is evident that a value of $\dot{V}F/\dot{V}E$ of at least 2 was necessary to eliminate rebreathing in any of the patients in the present study. This result agrees with earlier predictions of the flow requirements of systems D and E (Mapleson, 1954). However, $VR \% VT$ was less than 10% and produced no changes in $VE$ or $PE'CO_2$ until $\dot{V}F/\dot{V}E$ was reduced to less than 1.5. The latter result is in good agreement with the findings of Eger (1974) in a study on anaesthetized patients breathing from a T-piece, but differs slightly from the findings of Willis, Pender and Mapleson (1975) in a similar study using conscious subjects. The larger fresh gas flow rates required to prevent significant rebreathing in the conscious subjects may be accounted for by the fact that respiratory flow in these subjects was sinusoidal in form.

Some patterns of breathing have been shown to be effective in reducing the $\dot{V}F$ required to prevent rebreathing with a T-piece system (Harrison, 1964). In the present study, the finding that $VR \% VT$ was small in the majority of patients when $\dot{V}F$ was reduced to $V_{tot}$ and $0.7 \dot{V}_{tot}$, may be explained by changes in the respiratory waveform when ventilation was reduced during anaesthesia. The effect of these changes on rebreathing in the Bain system is apparent from the model of respiration (fig. 2). When peak respiratory flow is large compared with $\dot{V}F$ and the expiratory waveform is sinusoidal in character (fig. 2A), the volume of gas drawn from the reservoir tube during inspiration will be large and contain a high proportion of alveolar gas (area B + C, fig. 2A). If peak inspiratory flow is reduced and simultaneously expiration becomes prolonged with a period of slower flow towards end-expiration (fig. 2B and C) a smaller volume of gas than previously will be drawn from the reservoir tube, which will contain a lower proportion of alveolar gas concentrated mainly in the latter part of inspiration (areas B + C, fig. 2B and C). This combination of waveform changes where the peak inspiratory and expiratory flow rates were reduced and the last part of expiration was linear or exponential in form, was seen occurring with ventilatory depression in many of our patients (patients 5 and 11, fig. 4, patients 5-12, table II).

The mean $\dot{V}E$ of all patients when $\dot{V}F$ was equal to $2 \dot{V}_{tot}$ (table III) is an indication of the degree of ventilatory depression induced by anaesthetic and pre-anaesthetic drugs. The observed value of $0.6 \dot{V}_{tot}$ is comparable to the data of Nunn (1960) which suggests that during anaesthesia with agents other than diethyl ether, $\dot{V}E$ is typically reduced to 0.65 of its basal value. The mean values of $PE'CO_2$ at all values of $\dot{V}F$ (table III) are comparable to those reported for halothane anaesthesia in the absence of rebreathing (Kain and Nunn, 1968). Individual values of $PE'CO_2$ varied from 5.6 kPa to 7.7 kPa (table II). Although $P_{aCO_2}$ was probably slightly greater (Nunn and Hill, 1960), this is unlikely to be dangerous.

In figure 6, $VR \% VT$ and $PE'CO_2$ are plotted simultaneously against the observed values of $\dot{V}E$ when $\dot{V}F$ was equal to $\dot{V}_{tot}$. It is evident that an increase in $\dot{V}E$ is accompanied by an increase in rebreathing but paradoxically, those patients in whom the degree of rebreathing was greatest had the lowest $PE'CO_2$. To explain this result, it is necessary to recall that with the Bain system any rebreathed alveolar gas is diluted on expiration and on re-inspiration by fresh gas continually entering the system (fig. 2). That part of the increased $\dot{V}E$ which is rebreathed alveolar gas can be regarded as wasted ventilation, but that part of the increase which is fresh gas and enters the alveoli will contribute to an increase in alveolar ventilation and hence reduce $PE'CO_2$.

In the present study, only patients undergoing operations involving strong surgical stimulation had high values of $\dot{V}E$ when $\dot{V}F$ was equal to $\dot{V}_{tot}$ (Nos 1-4, table II). The finding that $PE'CO_2$ of these patients was less than that of the majority of the patients studied is analogous to the findings of previous studies in which the Bain system was used in patients under light anaesthesia (Spoerel, Aitken and Bain, 1978; Byrick, 1980). In these studies rebreathing was accompanied by hyperventilation when $\dot{V}F$ was reduced to $\dot{V}_{tot}$; thus the concentration of carbon dioxide observed was either normal or rarely, moderately increased compared with the value found in awake subjects. The results of the present study indicate that when $\dot{V}E$ is large enough to cause significant rebreathing in patients breathing from the Bain system with $\dot{V}F$ equal to $\dot{V}_{tot}$, the value of $PE'CO_2$ observed will always be less than that observed in those patients exhibiting the usual degree of anaesthetic-induced ventilatory depression (fig. 6).

**Clinical implications**

It is clear that, if rebreathing is to be eliminated or
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reduced to an insignificant level in patients breathing from the Bain circuit, the $\dot{V}_F$ required is dependent upon the actual $V_E$. In practice, fresh gas flow rates are set with reference to estimates of the patient's normal ventilation ($V_{tot}$) which in most cases will be considerably greater than the $V_E$ occurring during anaesthesia. Typically, $V_E$ is reduced to less than 2/3 $V_{tot}$ during halothane anaesthesia and respiratory depression may be even greater when the newer agents enflurane and isoflurane are used (Wade, Wendell and Stevens, 1981). In this case, given the concomitant favourable changes in respiratory flow pattern, it is unlikely that significant rebreathing will occur when $\dot{V}_F$ is equal to $V_{tot}$, since $\dot{V}_F$ will be greater than 1.5 $V_E$. It has been shown that at greater values of $V_E$ substantial volumes of alveolar gas may be rebreathed, but when this occurs the net result of the increased ventilation is a reduction in $P_{E'CO_2}$. We conclude that during anaesthesia when the Bain system is used with $\dot{V}_F$ equal to $V_{tot}$, any increase in $P_{E'CO_2}$ as a result of rebreathing is likely to be small and seldom of clinical importance.

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REFERENCES


UNE ESTIMATION DU RECAPTAGE RESPIRATOIRE AVEC LE CIRCUIT DE BAIN AU COURS D'UNE ANESTHESIE EN VENTILATION SPONTANEE

RESUME

Les données obtenues chez 12 patients anesthésiés respirant spontanément à partir d'un circuit de Bain, ont été utilisées pour calculer le degré de recaptage respiratoire qui survient lorsque le débit de gaz frais ($\dot{V}_F$) est égal à 2 et 1,07 fois la ventilation minute normale estimée ($V_{tot}$). Des mesures du volume expiré par minute ($V_E$) et de la pression partielle de CO₂ en fin d'expiration ($P_{E'CO_2}$) ont été faites pour préciser les effets de ce recaptage. Il n'y a pas de recaptage respiratoire lorsque $\dot{V}_F$ est le double de $V_{tot}$. Lorsque $\dot{V}_F$ égale $V_{tot}$ le recaptage est habituellement faible et n'entraîne pas de modification de $V_E$ ou de $P_{E'CO_2}$. Des modifications que l'on puisse rapporter au recaptage n'ont été observées que chez deux patients lorsque $\dot{V}_F$ était réduit à 0,7 $V_{tot}$. Ces résultats s'expliquent par la présence d'une dépression respiratoire d'origine anesthésique et par des modifications favorables des courbes de cycle ventilatoire chez la majorité des patients étudiés. Chez quelques patients, des valeurs de recaptage et de $V_E$ plus élevées ont été observées en réponse à une stimulation chirurgicale puissante. Le résultat net de l'augmentation de ventilation chez ces patients était une diminution de $P_{E'CO_2}$. Nous en concluons qu'au cours de l'anesthésie, lorsque le circuit de Bain est utilisé avec un $\dot{V}_F$ égal à $V_{tot}$, toute augmentation de $P_{E'CO_2}$ qui survenirait du fait d'un recaptage respiratoire sera probablement faible et aura rarement un retentissement clinique.
EINSCHÄTZUNG DER RÜCKATMUNG IM BAIN-SYSTEM BEI NARKOSE MIT SPONTANATMUNG

ZUSAMMENFASSUNG


UNA EVALUACION DE LA REINHALACION MEDIANTE EL SISTEMA BAIN DURANTE LA ANESTESIA CON VENTILACION ESPONTANEA

RESUMEN

Se utilizaron los datos obtenidos de 12 pacientes anestesiados que respiraban espontáneamente del sistema Bain, para calcular el grado de reinhalación que tuvo lugar cuando el régimen del flujo de gas limpio VF era igual a 2, 1 y 0,7 veces la ventilación normal calculada por minuto Vtot. Se efectuaron mediciones del volumen espirado por minuto (VE) y de la tensión del flujo final de dióxido de carbono (PE'CO₂), para determinar los efectos de esta reinhalación. Cuando VF fue igual a Vtot, la reinhalación fue normalmente de pequeña cantidad y no produjo cambio alguno en el VE ni en la PE'CO₂. Tan sólo en dos de los pacientes tuvieron lugar cambios atribuibles a la reinhalación cuando el VF se redujo a 0,7 Vtot. Estos resultados se explican por la presencia de una depresión ventilatoria inducida por la anestesia y por cambios favorables que tuvieron lugar en las formas de onda respiratorias de la mayoría de los pacientes. En algunos de dichos pacientes se apreciaron valores del VE y de la reinhalación más elevados como respuesta a una fuerte estimulación quirúrgica. El resultado neto del incremento de ventilación en estos pacientes fue una disminución del PE'CO₂. Se concluye que durante la anestesia, cuando se utilizó el sistema Bain con VF igual a Vtot, todo incremento de la PE'CO₂ que pueda ser consecuencia de la reinhalación será con seguridad pequeño y raramente de importancia clínica.