Rebreathing and end-tidal CO₂ during spontaneous breathing with the Bain circuit

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In order to examine the relationship between end-tidal CO₂ (F_{ET}CO₂) and inspired CO₂ (F_ICO₂) in anaesthetized patients breathing spontaneously with a Bain breathing circuit and a fresh gas volume (V_F) of 100 ml·kg⁻¹·min⁻¹, the respiratory rate (f) and minute ventilation (V_E) was changed in two groups of six patients each by the induction or reversal of narcotic respiratory depression. During light nitrous oxide-halothane anaesthesia (Group I), the intravenous injection of 0.1 mg·kg⁻¹ of alphaprodine caused a rapid fall in F_ICO₂ from 2.3 ± 0.5 per cent to 0.7 ± 0.1 per cent concomitant with the reduction in f (37 ± 5 to 16 ± 4) breath·min⁻¹ and V_E (137 ± 29 to 55 ± 13 ml·kg⁻¹·min⁻¹), while the F_{ET}CO₂ rose gradually from 5.2 ± 0.9 per cent to 6.4 ± 0.9 per cent over a ten-minute period. During light nitrous oxide-halothane anaesthesia supplemented by alphaprodine (Group II), 0.2 mg of naloxone intravenously caused a rise in F_ICO₂ from 0.5 ± 0.3 per cent to 2.9 ± 0.6 per cent simultaneous with a rise in f (11 ± 2 to 25 ± 7) breath·min⁻¹ and V_E (70 ± 25 to 133 ± 34 ml·kg⁻¹·min⁻¹), while the F_{ET}CO₂ declined gradually over a ten-minute period from 7.6 ± 0.7 per cent to 6.4 ± 0.4 per cent.

The change in F_ICO₂ always occurred exactly at the same time as the drug-induced change in respiration. It was associated with a corresponding change in the degree of mixing of fresh gas and expired gas within the breathing system and appeared to correlate with the

change in the ratio V_E/V_F. There was no indication that the F_ICO₂ or the distribution of CO₂ within the system had any effect on F_{ET}CO₂ or CO₂ elimination. Under these conditions the F_ICO₂ and the volume of rebreathed CO₂ can not be the cause but must be regarded as a passive change consequent to the altered pattern of breathing.

Key words

ANAESTHETIC TECHNIQUES, NON REBREATHING: Bain circuit.

The safe use of the Bain circuit¹ (as a modified Mapleson D system) for spontaneous breathing under anaesthesia has been questioned²⁻⁵ because of the presence of carbon dioxide in the inspired gas, indicating rebreathing. Strong recommendations were made that rebreathing must be avoided by using much higher fresh gas volumes (V_F) than the suggested 100 ml/kg⁻¹·min⁻¹.⁶ An attempt to express the inspired CO₂ concentration (F_ICO₂) as a CO₂ load was recently presented by Byrick,^{7,8} this was based on the commonly held assumption that the patient is responding to the re-inhaled CO₂ with hyperventilation, thus compensating for the load imposed by the inhaled CO₂. An inability to achieve such compensation, e.g., due to anaesthetic depression of respiration, would then cause hypercapnia. This concept assumes that there is a direct relationship between F_ICO₂ and alveolar or end-tidal CO₂ (F_{ET}CO₂) and that hypercapnia can be avoided by keeping the F_ICO₂ below 1 per cent.⁹

During controlled ventilation, a direct relationship between F_ICO₂ and F_{ET}CO₂ could not be established;¹⁰ the F_ICO₂ could be manipulated independently of F_{ET}CO₂ by changing tidal volume, respiratory rate and the ratio between inspiratory and expiratory time. It was concluded that the F_ICO₂ reflected the degree of rebreathing or mixing of

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fresh gas and expired gas within the breathing tube, but the F_{ETCO_2} and CO_2 elimination were not affected by these changes.

Although the situation is more complex during spontaneous breathing, the distribution of gas within the breathing circuit should in principle not be different from controlled ventilation. The F_{ICO_2} , as indicator of rebreathing, should change unrelated to the F_{ETCO_2} and vary passively with changes in respiratory rate (f) and minute ventilation ($\dot{V}_E$). In order to demonstrate that in the spontaneously breathing patient the F_{ICO_2} can change passively as the result of a pharmacologically induced change in breathing, the induction and withdrawal of narcotic depression of the respiration was studied in anaesthetized patients.

Methods

In 12 healthy adult patients (7 female, 5 male, mean age 46 years, range 19–74 years), who were undergoing surgical procedures suitable for spontaneous breathing during anaesthesia, a specially prepared Bain breathing system was used that allowed sampling at intervals along the circuit.¹⁰ In addition, gas was sampled from a small catheter introduced through the connecting elbow to the tip of the endotracheal tube (F_{ETCO_2} and F_{ICO_2}) and from a port near the venting valve to determine the CO_2 concentration of vented gas. F_{ETCO_2} and F_{ICO_2} were recorded continuously with a mass spectrometer (Elmer-Perkins); the sampling volume of about $80 \text{ ml} \cdot \text{min}^{-1}$ was low enough not to interfere with the function of the circuit. Sampling from the other ports was done sequentially starting at the port nearest the endotracheal tube. These determinations were done shortly before and ten minutes after the induced change in respiration.

The study was approved by the Committee on Human Research of the University of Western Ontario. All patients were unpremedicated. Anaes-

thesia was induced with a sleep-dose of thiopental and the trachea was intubated following a single dose of succinylcholine. After return of spontaneous breathing, a $\dot{V}_F$ of $100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ was set with a $N_2O:O_2$ ratio of about 5:2. The flow set on the flow meters was later verified with a Collins recording spirometer. The expired minute volume ($\dot{V}_E$) was determined by a Wright respirometer interposed between the Bain circuit and tracheal tube.

In six patients (Group I) anaesthesia was maintained with supplementation of 1 per cent halothane. After 45 minutes of stable anaesthesia, $\dot{V}_E$ and respiratory rate (f) were determined before and after the sampling sequence described above. The gas concentration in the trachea (tip of tracheal tube) was then continuously monitored while the patient was given 0.1 mg/kg^{-1} alphaprodine intravenously. Ten minutes after the injection $\dot{V}_E$ and f were again estimated and the sampling sequence repeated. In another six patients (Group II) the anaesthesia was supplemented with halothane 0.5 per cent and intravenous alphaprodine in a dosage sufficient to keep f near 10 breaths/min⁻¹. After 45 minutes of stable anaesthesia, $\dot{V}_E$ and CO_2 distribution were determined as described above and with continuous recording of the F_{ETCO_2} and F_{ICO_2} , 0.2 mg naloxone was then given intravenously. $\dot{V}_E$, f and CO_2 distribution were again determined ten minutes after this injection. All reported results represent the mean $\pm$ S.D. of six patients.

Results

The six patients in Group I had a $\dot{V}_E$ of $137 \pm 29 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and an f of $37 \pm 5 \text{ breath} \cdot \text{min}^{-1}$, 45 minutes after induction of anaesthesia; F_{ETCO_2} was 5.2 ± 0.9 per cent, F_{ICO_2} 2.3 ± 0.5 per cent. Alphaprodine injection was followed after about 30 seconds by a rapid decline in F_{ICO_2} to values below 1 per cent, exactly coinciding with a decrease in f and in $\dot{V}_E$. The F_{ETCO_2} rose gradually during the ten-minute observation period (Figure 1). The distribution of CO_2 within the breathing tube changed from a marked degree of mixing to a wide separation between maximum and minimum CO_2 concentration (Figure 2). The changes in F_{ETCO_2} and F_{ICO_2} occurred at a different rate and in the opposite direction in all six patients (Figure 3). Ten minutes after the injection the $\dot{V}_E$ was $55 \pm 30 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ at an f of 16 ± 4 per minute; the F_{ETCO_2} was 6.4 ± 0.9 per cent and the F_{ICO_2} $0.7 \pm$

Abbreviations

$\dot{V}_F$	= Minute volume of fresh gas supplied by anaesthetic apparatus.
$\dot{V}_E$	= Expired minute volume.
F_{ETCO_2}	= End tidal CO_2 concentration.
F_{ICO_2}	= Lowest CO_2 concentration recorded during inspiration.
f	= Respiratory rate.

FIGURE 1 CO_2 concentration in trachea: example of changes of FET_{CO_2} and FI_{CO_2} in response to alphaprodine (0.1 mg/kg^{-1}) during light $\text{N}_2\text{O}-\text{O}_2$ halothane anaesthesia.

FIGURE 2 Change in maximum and minimum CO_2 concentration recorded in the trachea and along the Bain circuit. Recordings are from the same patient during light N_2O -halothane anaesthesia before (upper part) and after alphaprodine (lower part). The maximum value in the trachea represents FET_{CO_2} , the minimum FI_{CO_2} . The CO_2 concentration in the breathing tubes were sampled at 30 cm intervals from the point of fresh gas inflow (0) to the connector with the anaesthetic machine (6). End represents the per cent CO_2 at the venting valve.

0.1 per cent. The $\dot{V}_E/\dot{V}_F$ ratio as an indicator of rebreathing¹¹ had changed from 1.37 before alphaprodine to 0.55 ten minutes later.

Patients in Group II had, after 45 minutes of anaesthesia, a $\dot{V}_E$ of $70 \pm 25 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ with an f of $11 \pm 2 \text{ breath}\cdot\text{min}^{-1}$. The FET_{CO_2} was 7.6 ± 0.7 per cent and the FI_{CO_2} , 0.5 ± 0.3 per cent. In response to naloxone 0.2 mg intravenously, f and $\dot{V}_E$ increased simultaneously with a rise in FI_{CO_2} while the FET_{CO_2} was gradually falling (Figure 4). The CO_2 distribution within the breathing tube changed from a wide separation between maximum and minimum values to a marked degree of mixing (Figure 5). Again the response was consistent in all patients (Figure 6). Ten minutes after the injection the $\dot{V}_E$ was $133 \pm 34 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$, the f $25 \pm 7 \text{ breath}\cdot\text{min}^{-1}$, the FET_{CO_2} had fallen to 6.4 ± 0.4 per cent while the FI_{CO_2} had risen to 2.9 ± 0.6 per cent. An initial $\dot{V}_E/\dot{V}_F$ ratio of 0.70 had changed to 1.33 ten minutes after naloxone.

Discussion

The FI_{CO_2} is generally regarded as an indicator of rebreathing. In the Mapleson D system, rebreathing is considered undesirable, presumably because it leads to CO_2 accumulation and forces the patient to hyperventilate. It is recommended that the FI_{CO_2} be kept as low as possible, by increasing $\dot{V}_F$ to at least twice the level of $\dot{V}_E$.

The observations presented here are not compatible with these assumptions. It could be shown that the induction and reversal of opiate depression of the respiration was associated with a simultaneous change in FI_{CO_2} . Under these conditions the respective fall or rise in FI_{CO_2} could not be the cause, but

FIGURE 3 Response of F_{ETCO_2} and F_{ICO_2} to alphaprodine in six patients (mean values $\pm$ S.D.) during anaesthesia with nitrous oxide and 1 per cent halothane ($\dot{V}_F = 100 \text{ ml/kg}^{-1}/\text{min}^{-1}$). The values for f , $\dot{V}_E$ and $\dot{V}_E/\dot{V}_F$ are listed before and ten minutes after alphaprodine. (See also Figure 1.)

had to be the consequence of the change in breathing.

Light halothane anaesthesia was associated with mild hyperventilation and a raised F_{ICO_2} , indicating considerable rebreathing; the F_{ETCO_2} was in a normal range. A pharmacological reduction of ventilation led to hypercarbia, while the F_{ICO_2} fell concomitant with the decline of f and $\dot{V}_E$ (Figures 1 and 3). Reversal of the opiate depression by naloxone restored the initial pattern of breathing and caused a return of the elevated F_{ETCO_2} towards normal, in spite of a raised F_{ICO_2} (Figures 4 and 6). These changes were consistent in all patients.

A $\dot{V}_E$ in excess of $\dot{V}_F$ was always associated with considerable mixing of expired gas and fresh gas within the breathing system (Figures 2 and 5). A $\dot{V}_E$ below $\dot{V}_F$, particularly with a low f , resulted in a

wide separation between minimum and maximum CO_2 levels inside the breathing circuit. It would appear that mixing of gases or rebreathing was related to the ratio $\dot{V}_E/\dot{V}_F$; ¹¹ a ratio >1.0 would show an elevated F_{ICO_2} as evidence of rebreathing. With opiate depression this ratio fell to 0.55, indicating a virtual absence of rebreathing. This demonstrates that functional non-rebreathing can not only be achieved, as often recommended^{2-5,9} by increasing $\dot{V}_F$ to twice $\dot{V}_E$ ($\dot{V}_E/\dot{V}_F = 0.5$), but also by a corresponding reduction of $\dot{V}_E$ below $\dot{V}_F$.

In controlled ventilation it could be shown¹⁰ that the F_{ICO_2} correlated with the degree of mixing of fresh gas and exhaled gas within the circuit. With a fixed $\dot{V}_F$, the F_{ICO_2} could be raised by increasing $\dot{V}_F$, or f , or both and by increasing the duration of inspiration. All these manipulations did not affect

FIGURE 4 CO_2 concentration in trachea: example of changes in response to 0.2 mg naloxone given during anaesthesia with N_2O -halothane supplemented with alphaprodine.

FIGURE 5 Changes in CO_2 concentration throughout breathing circuit during light N_2O -halothane anaesthesia supplemented with alphaprodine before (upper part) and after alphaprodine (lower part). (For details see Figure 2.)

FET_{CO_2} as long as $\dot{V}_E$ remained greater than $\dot{V}_F$, nor did they influence the CO_2 content of vented gas. It was concluded that rebreathing or the mixing of gases within the circuit did not affect CO_2 elimination nor the alveolar CO_2 concentration.

In spontaneous breathing the same relationships

should exist, except that $\dot{V}_E$ is determined by the patient. With a fixed $\dot{V}_F$ the $\dot{V}_E$ will be either greater or lower than $\dot{V}_F$ depending on the degree of pharmacological depression of breathing. According to the $\dot{V}_E$, the breathing system will fluctuate between a functional state of non-rebreathing (if $\dot{V}_E/\dot{V}_F = <0.5$) to various degrees of partial rebreathing (if $\dot{V}_E > \dot{V}_F$). This functional state is indicated by the level of F_{ICO_2} .

The FET_{CO_2} is not influenced by the ratio $\dot{V}_E/\dot{V}_F$ but depends on the absolute values of $\dot{V}_F$ and $\dot{V}_E$. The relationships of FET_{CO_2} to $\dot{V}_E$ and $\dot{V}_F$ has been demonstrated by Rose *et al.* in model experiments.⁴ If $\dot{V}_E > \dot{V}_F$ then $\dot{V}_F$ will mainly determine FET_{CO_2} ; if $\dot{V}_E < \dot{V}_F$, then $\dot{V}_E$ will mainly control FET_{CO_2} . Our observations provide no evidence that the relationship between $\dot{V}_E$, $\dot{V}_F$ and FET_{CO_2} is affected by rebreathing or the degree of mixing of fresh gas and alveolar gas inside the breathing system. The F_{ICO_2} only reflects the mixing as determined by $\dot{V}_E$, $\dot{V}_F$ and by the patient's breathing pattern.

Byrick^{7,8} has made an attempt to quantify the inspired CO_2 by calculating its volume from F_{ICO_2} and inspiratory flow rate. Assuming that his patients were reacting to the rebreathed CO_2 with hyperventilation or, in the absence of a compensatory response, with hypercapnia, he introduced the concept of the inspired " CO_2 load." In a comparison of halothane and enflurane in spontaneously breathing patients,⁸ he found lower CO_2 loads, i.e., less breathing with enflurane but then stated that "The advantage of reduced rebreathing with enflurane is counterbalanced by the more profound respiratory depression it causes."

FIGURE 6 Response of $F_{ET}CO_2$, $F_{I}CO_2$ and f to 20.2 mg naloxone intravenously in six patients (mean values $\pm$ S.D.) during nitrous oxide-halothane anaesthesia supplemented with alphaprodine. Inserted are values for V_E and V_E/V_F before and ten minutes after naloxone. (See also Figure 4.)

This apparent contradiction disappears if rebreathing is seen as a passive phenomenon. Enflurane causes greater respiratory depression resulting in elevated $F_{ET}CO_2$ values; according to the findings presented here, this must be associated with a low $F_{I}CO_2$. Halothane causes less respiratory depression resulting in near normal $F_{ET}CO_2$ values; however, the higher V_E and the higher f raise passively the $F_{I}CO_2$ as an indication of greater rebreathing.

The fact that rebreathing in the Mapleson D system is passive and does not influence the CO_2 homeostasis is further supported by the lack of any correlation between the volume of CO_2 re-inhaled and the alveolar CO_2 level.⁷ And since a direct effect of the re-inhaled CO_2 on the patient's breathing can not be demonstrated, the volume of re-inhaled CO_2 should not be interpreted as a "load" to the patient.

It must be emphasized that the conditions discussed here pertain only to the normally functioning breathing system of the Mapleson D configuration, particularly in regard to an adequate volume of fresh gas delivered close to the patient's airway. These relationships are not applicable if the $F_{I}CO_2$ is increased due to the interposition of deadspace between the patient's airway and the point of fresh gas inflow.

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Résumé

Afin d'analyser le rapport entre la concentration du CO_2 à la fin de l'expiration (F_{ETCO_2}) et le CO_2 inspiré (F_{ICO_2}) chez des patients anesthésiés et respirant par eux-mêmes avec un circuit de Bain et d'un contenu de gaz frais ($\dot{V}_F$) de $100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ le taux de la respiration (f) et la ventilation minute ($\dot{V}_E$) changèrent chez deux groupes de six patients avec l'induction ou l'inversion de la dépression causée par le narcotique. Durant l'anesthésie avec le protoxyde d'azote-halothane (Groupe I) une injection intraveineuse de $0.1 \text{ mg} \cdot \text{kg}^{-1}$ d'alphaprodine causa une baisse de F_{ICO_2} de 2.3 ± 0.5 pour cent à 0.7 ± 0.1 pour cent, concomitant avec la réduction de f (37 ± 5 à 16 ± 4) respiration $\cdot \text{min}^{-1}$ et de $\dot{V}_E$ (137 ± 29 à $55 \pm 13 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), alors que le F_{ETCO_2} monta graduellement de 5.2 ± 0.9 pour cent à 6.4 ± 0.9 pour cent au cours d'une période de dix minutes. Au cours de l'anesthésie avec le protoxyde d'azote halothane auquel on ajouta de l'alphaprodine (Groupe II), l'administration intraveineuse de 0.2 mg de naloxone causa une élévation de F_{ICO_2} de 0.5 ± 0.3 pour cent de 2.9 ± 0.6 pour cent simultanément avec une élévation de f (11 ± 2 à 25 ± 7 respiration $\cdot \text{min}^{-1}$) et de $\dot{V}_E$ (70 ± 25 à $133 \pm 35 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), alors que le F_{ETCO_2} baissa graduellement au cours d'une période de dix minutes de 7.6 ± 0.7 pour cent à 6.4 ± 0.4 pour cent. Le changement du F_{ICO_2} se manifesta toujours exactement en même temps que les variations en respirations déclenchées par les drogues. Ce changement était en rapport avec les variations qui correspondent avec le taux du mélange de gaz frais et de gaz expiré dans le système respiratoire et semble être en corrélation avec la variation du ratio $\dot{V}_E/\dot{V}_F$. Ceci n'indique pas que le F_{ICO_2} ou la distribution du CO_2 dans le système ait aucun effet dans le F_{ETCO_2} ou dans l'élimination du CO_2 . Dans ces circonstances, le F_{ICO_2} et le volume du CO_2 réinspiré doivent être interprétés comme des changements passifs résultant du modèle altéré de la respiration mais ne peuvent pas en être la cause.