Respiratory Waveform and Rebreathing in T-Piece Circuits:

A Comparison of Enflurane and Halothane Waveforms

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The effects of respiratory waveform on rebreathing in a modified Mapleson D circuit were studied in 18 healthy adult patients anesthetized with either enflurane or halothane. At high fresh gas flow (FGF) rates, when no rebreathing of CO₂ occurred, the duration of inspiration (T₁) with enflurane was 41 per cent greater than that with halothane. With enflurane there was a characteristic long end-expiratory pause, 0.69 s, whereas with halothane it was only 0.196 s. The mean inspiratory flow rate (V₁/T₁) was higher (224 ml/s) when halothane was used than with enflurane (187 ml/s). When the FGF rate was reduced to 100 ml/kg/min in the modified Mapleson D circuit, patients breathing halothane had increases in minute volumes (Vₑ) in response to increases of 53–75 per cent in inspired volumes of CO₂. The increases in Vₑ resulted from increases in V₁/T₁ of 34–58 per cent. The volume of CO₂ inspired when enflurane was used did not increase until FGF rate was as low as 70 ml/kg/min. The reduced rebreathing was related to the respiratory waveform. The advantage of reduced rebreathing with enflurane is counterbalanced by the more profound respiratory depression it causes. The FGF needed to abolish rebreathing of CO₂ is highly variable, and is dependent on respiratory waveform. (Key words: Anesthetics, volatile; enflurane; halothane. Equipment: circuits; T-piece. Ventilation: anesthetics, effects of; carbon dioxide response; pattern.)

Mapleson¹ recognized the importance of respiratory waveform in determining the rebreathing characteristics of a T-piece breathing system in 1954. His recommendation for a fresh gas flow (FGF) rate of 2–2.5 times minute ventilation to prevent rebreathing of CO₂ assumes a sine-wave pattern of respiration. In this theoretical analysis of the function of his “D system,” Mapleson¹ stated that if, for the same tidal volume, the inspiratory flow rate pattern is “peaky,” entrainment of gas may occur from the expiratory limb of the T-piece.

In a study using a lung model, Harrison² documented the importance of an expiratory pause in the function of a D type of T-piece system. The study confirmed Mapleson’s prediction that a pause would allow a fresh gas “buffer” to exist in the expiratory limb, which could minimize CO₂ rebreathing were entrainment to occur.

The Bain modification³ of the Mapleson D circuit has been shown to be identical to the classic D type of T-piece⁴ in terms of its function. The Bain system permits significant rebreathing of CO₂ during halothane anesthesia⁵ when low FGF rates are used. Clinically, patients anesthetized with enflurane breathe more slowly than those anesthetized with halothane.⁶ Spoerel et al.⁷ have found that, unlike the situation with halothane, the minute volume with enflurane does not increase when FGF is reduced.

The purpose of this investigation was to characterize the differences in respiratory waveforms that occur with these two agents under clinical conditions, and to determine the significance of these waveform differences in a modified Mapleson D circuit during spontaneous respiration.

Methods and Materials

Eighteen healthy patients (ASA I) undergoing general anesthesia in the supine position for exploration and repair of peripheral nerve lesions were studied. These patients (age range 19–65 years) had no clinical evidence of cardiopulmonary disease by history or physical examination. The durations of these procedures allowed time to study the effects of altering FGF rate on respiratory pattern. An institutional review committee agreed that it was not necessary to obtain formal consent from participating patients.

Each unmedicated patient was given thiopental sodium, 3–5 mg/kg, followed by succinylcholine, 1 mg/kg, to facilitate intubation. Either halothane or enflurane, in oxygen, was then administered using a modified Mapleson D (Bain) breathing system. Nitrous oxide was not used in order to avoid interference in the analysis of CO₂ tension and end-tidal anesthetic concentration.

After intubation, the endotracheal tube was attached to a calibrated, heated pneumotachograph, Fleisch #2, which was connected to a differential transducer (Statham PM 5). The pneumotachograph was calibrated with oxygen, and linear accuracy to a flow rate of 20 l/min was demonstrated. The flow signal from the transducer was used to measure T₁, the duration of inspiration; T₂, the total respiratory cycle time; Tₑ, the duration of active expiration, and Tₚa, the duration of zero flow after active expiration. The flow signal was integrated to obtain tidal volume.

The concentration of CO₂ in the respiratory gases
The sequence of FGF rates used for each patient was 100 ml/kg/min, 200 ml/kg/min, and 100 ml/kg/min. The FGF rate was returned to 100 ml/kg/min to ensure that the ventilatory changes were not the result of changing surgical stimulation or anesthetic duration. Additional measurements were made at 70 and/or 50 ml/kg/min in patients receiving enfurane. In patients anesthetized with halothane, FGF rates of less than 100 ml/kg/min were used only if the inspired CO₂ concentration was not significantly increased at the 100 ml/kg/min FGF rate.

After each recording was obtained, end-tidal gas was sampled and analyzed for halothane or enfurane concentration using a calibrated refractometer. Free-flowing peripheral venous blood obtained from a forearm vessel was sampled, and its P$_{CO_2}$ measured, at each FGF rate studied. The P$_{CO_2}$ was compared with the end-tidal CO₂ value measured at the distal end of the endotracheal tube. With similar methodology, the P$_{CO_2}$s of peripheral venous blood have been shown to be within ±1 torr of arterial blood P$_{CO_2}$ values in anesthetized subjects.

Data are presented as means ± 1 SEM. Serial measurements in the same patient were compared using the Student t test for paired data, while differences between enfurane- and halothane-anesthetized subjects were compared using the Student t test for unpaired data. P < 0.05 was considered significant.

Results

Respiratory Waveform

The end-tidal halothane concentration for all patients was 1.05 ± 0.03 per cent, while with enfurane administration, the end-tidal concentration was 1.64 ± 0.04 per cent. No patient responded to the surgical stimulus during the study period. When these figures were converted to MAC multiples, the end-tidal halothane MAC equaled 1.42, while that of enfurane was 0.97. These values were stable throughout anesthesia.

The values found for the measured components of the respiratory cycle, at the FGF rates studied, are summarized in table 1. Even at FGF rates previously shown to prevent rebreathing with T-piece circuits (200 ml/kg/min), there was a significant difference between the phase durations of the respiratory cycle in patients anesthetized with enfurane compared with halothane. The time of inspiration (T$_{I}$) with enfurane was 41 per cent longer than that with halothane, while the fraction of the respiratory cycle devoted to inspiration (T$_{I}$/T$_{Ttot}$) was 15 per cent greater during halothane anesthesia than when enfurane is used. This is primarily a result of a longer expiratory pause (T$_{pause}$ 0.69 s) with enfurane than with
halothane (0.196 s). The duration of active expiration was also longer with enflurane.

The minute volume ($V_e$) in subjects breathing halothane was 40 per cent greater than when enflurane was used, at the highest FGF rate studied. During halothane anesthesia, $V_e$ increased significantly by 49 per cent, when the FGF rate was reduced to 100 ml/kg/min. During enflurane anesthesia, the $V_e$ increased significantly (23 per cent) only at the FGF rate of 50 ml/kg/min. Respiratory frequency was much higher (31–33/min) when halothane was used, compared with enflurane (17–20/min). Tidal volume ($V_t$) increased during halothane anesthesia when the FGF rate was reduced from 200 to 100 ml/kg/min. Patients receiving enflurane had increased $V_t$ only when the FGF rate was reduced to 50 ml/kg/min (table 1).

At the high FGF rate (200 ml/kg/min), the mean inspiratory flow rate ($V_i/T_i$) with halothane anesthesia was 38 per cent higher than when enflurane was used. Patients receiving halothane had significantly increased $V_i/T_i$ (38 per cent) when the FGF rate was reduced to 100 ml/kg/min. With enflurane anesthesia no significant change in mean $V_i/T_i$ was seen until the FGF rate was reduced to 50 ml/kg/min (table 1).

### Table 1. Respiratory Waveform Changes in the Patients Studied, Comparing the Effects of Halothane and Enflurane under Clinical Conditions (Enflurane, n = 9; Halothane, n = 9)

<table>
<thead>
<tr>
<th></th>
<th>Fresh Gas Flow Rate</th>
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<tbody>
<tr>
<td></td>
<td>100 ml/kg/min</td>
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<tr>
<td><strong>Minute volume ($V_e$) (l/min)</strong></td>
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<tr>
<td>Enflurane</td>
<td>4.44 ± 0.28</td>
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<tr>
<td>Halothane</td>
<td>8.93 ± 0.71*</td>
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<tr>
<td><strong>Tidal volume ($V_t$) (ml)</strong></td>
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<tr>
<td>Enflurane</td>
<td>217.2 ± 10.9</td>
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<tr>
<td>Halothane</td>
<td>265.6 ± 15.8*</td>
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<tr>
<td><strong>Respiratory rate (f) (min⁻¹)</strong></td>
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<tr>
<td>Enflurane</td>
<td>19.9 ± 1.13</td>
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<tr>
<td>Halothane</td>
<td>33.3 ± 2.96</td>
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<tr>
<td><strong>Mean inspiratory flow rate ($V_i/T_i$) (ml/s)</strong></td>
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</tr>
<tr>
<td>Enflurane</td>
<td>171.15 ± 8.4</td>
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<tr>
<td>Halothane</td>
<td>310.82 ± 26.1*</td>
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<tr>
<td><strong>Fraction of total respiratory cycle time devoted to inspiration ($T_i/T_{tot}$)</strong></td>
<td></td>
</tr>
<tr>
<td>Enflurane</td>
<td>.413 ± .012</td>
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<tr>
<td>Halothane</td>
<td>.487 ± .009</td>
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<tr>
<td><strong>Time of inspiration ($T_i$) (s)</strong></td>
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<tr>
<td>Enflurane</td>
<td>1.28 ± .06</td>
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<tr>
<td>Halothane</td>
<td>.9 ± 0.04</td>
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<tr>
<td><strong>Total respiratory cycle time ($T_{tot}$) (s)</strong></td>
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<tr>
<td>Enflurane</td>
<td>3.11 ± .14</td>
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<tr>
<td>Halothane</td>
<td>1.85 ± .09</td>
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<tr>
<td><strong>Duration of end-expiratory pause ($T_{pause}$) (s)</strong></td>
<td></td>
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<tr>
<td>Enflurane</td>
<td>.64 ± .06</td>
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<tr>
<td>Halothane</td>
<td>.04 ± .02*</td>
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<tr>
<td><strong>Duration of active expiration ($T_{active}$) (s)</strong></td>
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<tr>
<td>Enflurane</td>
<td>1.18 ± .08</td>
</tr>
<tr>
<td>Halothane</td>
<td>.91 ± .05</td>
</tr>
<tr>
<td><strong>CO₂ volume per breath (ml)</strong></td>
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</tr>
<tr>
<td>Enflurane</td>
<td>10.04 ± .28</td>
</tr>
<tr>
<td>Halothane</td>
<td>9.86 ± .61*</td>
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<tr>
<td><strong>End-tidal CO₂ (FECO₂) (per cent)</strong></td>
<td></td>
</tr>
<tr>
<td>Enflurane</td>
<td>6.35 ± .2</td>
</tr>
<tr>
<td>Halothane</td>
<td>5.43 ± .2</td>
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*Significantly different from value obtained at high gas flow rate in the same patients (P < 0.05) by t test for paired data.
enflurane was used (8.3 ± 1.0 torr) was not significantly different from the gradient (7.0 ± 1.3 torr) when halothane was administered. Although rebreathing of CO₂ in patients anesthetized with enflurane did not occur until FGF rates were as low as 70 ml/kg/min (table 1), the venous blood $P_{CO_2}$s achieved in this non-rebreathing state ranged from 46 to 71 torr (mean 53.8 ± 2.2), whereas the average $P_{CO_2}$ in the subjects receiving halothane was 45.4 ± 1.8 torr (range 41 to 50 torr) at the FGF rate of 100 ml/kg/min.

The increase in the volume of CO₂ inspired per min (fig. 2) when halothane was used at a FGF rate of 100 ml/kg/min was magnified by the high respiratory frequency (table 1) in this group. In a typical trace of the inspired flow of CO₂ in a patient who received halothane (fig. 1), the increase in CO₂ inspired was found to occur after the dead-space gas was inhaled. This confirmed the assumption that the increase in measured volume of CO₂ inspired as FGF rate decreased with halothane anesthesia was the result of rebreathing CO₂, and not solely due to an increase in end-tidal CO₂ in dead-space gas. In another typical trace for a subject breathing enflurane (fig. 1), the inspired CO₂ was found to increase slightly when the FGF rate decreased. However, the increase in inspired CO₂ occurred early in the breathing, showing that this increase resulted from the increase in CO₂ concentration ($F_{ET,CO_2}$) in the endotracheal tube and connector, and not from rebreathing when the FGF was reduced.

There was considerable variation in the absolute values of mean inspiratory flow rates ($V_i/T_i$) and inspired CO₂ volumes per breath among patients (table 1). When the absolute values of $V_i/T_i$ were plotted (fig. 3d) against the simultaneous inspired CO₂ volumes of those breaths for patients receiving halothane anesthesia, a significant positive correlation was found ($r = 0.78; P < 0.001$). When a similar plot for patients breathing enflurane was graphed, the line of best fit was not a straight line, but rather a modified exponential (fig. 3B).

The interrelationship of these two variables was then evaluated by plotting the change in $V_i/T_i$ (Δ$V_i/T_i$) versus the change in inspired volume of CO₂ that resulted from a given change in FGF rate (fig. 4A and B). During halothane anesthesia the mean inspiratory flow rate increased as the FGF rate was reduced (table 1). Simultaneously, the inspired volume of CO₂ increased. Figure 4A illustrates that for a given change in $V_i/T_i$ at each FGF rate studied, there was a linear increase in CO₂ volume inspired, over the range examined in this study.

When enflurane was used, there was no increase in $V_i/T_i$ or inspired CO₂ load as the FGF rate was reduced, until FGF rates of less than 100 ml/kg/min were reached (figs. 3B and 4B). When the changes in $V_i/T_i$s (Δ$V_i/T_i$) in enflurane-anesthetized patients

**Inspired Volume of CO₂**

The measured inspired volume of CO₂ per breath is composed of two parts. The initial component results from reinhalation of dead-space gas proximal to the sampling catheter (in the endotracheal tube and connectors), and the second part is the CO₂ inspired as a result of rebreathing from the circuit.

At a FGF rate of 200 ml/kg/min, the inspired volume of CO₂ was 84 per cent higher in subjects anesthetized with enflurane than in those given halothane (table 1). This difference at the high FGF rate, hence no rebreathing, was due in part to a higher end-tidal CO₂ level ($F_{ET,CO_2} = 6.24$ per cent) when enflurane was used, compared with halothane ($F_{ET,CO_2} = 5.37$ per cent). Although the absolute volumes of dead space above the CO₂ catheter sampling site at the distal end of the endotracheal tube varied with individual patients, the position of the sampling catheter in any given patient remained constant throughout the study period. Thus, any change in the volume of CO₂ inspired from that recorded at the high FGF rate represented an increase in the volume of CO₂ rebreathed.

The inspired volume of CO₂ per breath increased significantly (53–75 per cent) in patients anesthetized with halothane when the FGF rate was reduced from 200 to 100 ml/kg/min. In patients anesthetized with enflurane, no change in the inspired CO₂ was detectable until the FGF rate was reduced to 70 ml/kg/min (table 1). At a FGF rate of 50 ml/kg/min, when enflurane was used, the inspired CO₂ volume increased by 60 per cent over that present in the nonrebreathing state (200 ml/kg/min).

The end-tidal-to-venous blood $P_{CO_2}$ gradient when
were plotted against the changes in inspired volumes
of CO₂, no significant linear relationship (fig. 4B)
was found (r = 0.2). With small increases in Vᵢ/T₁ in
this group of patients, no increase in the volumes of
CO₂ inspired was found (figs. 3B and 4B). This finding
was attributed to the much longer end-expiratory
pause (table 1) in this group, resulting in no further
CO₂ entrainment with small increases in Vᵢ/T₁. The
levelling out of this inspired CO₂ volume (fig. 3B)
supported the contention that at the high FGF rate the
inspired CO₂ volume was reduced to an irreducible
minimum value. For halothane-anesthetized patients
the variability of CO₂ inspired can be well explained
(y = −0.20 + 0.42 x) (r = 0.90) by changes in Vᵢ/T₁
(fig. 4A). The reference value for each point in this
relationship was the value measured immediately
prior to each change in the FGF rate. Using a variable
reference point throughout the study period allowed
us to assess the relationship between the changes in
mean Vᵢ/T₁ and the resulting changes in inspired CO₂
load throughout the range examined in these patients.
The linear regression when halothane was used (fig.
4A) illustrated that for this agent, with a brief expiratory
pause (table 1), there was a linear increase in CO₂
volume inspired with an increase in inspiratory flow
rate in the clinical situation studied. The increases
in Vᵢ in response to reduced FGF rates resulted from
increases in Vᵢ/T₁, since T₁/Ttot was constant (table 1)
with both agents. In terms of the functional alveolar
ventilation (hence CO₂ elimination) achieved by a
given increase in Vₑ, the net effectiveness of an increase
in tidal volume will depend on the fraction of tidal
volume containing CO₂. The fraction of CO₂ inspired
in a given tidal volume (CO₂ volume/tidal volume)
at each FGF rate was calculated. This fraction is the
average inspired volume of CO₂ per breath, or FCO₂.
Figure 5A shows that during halothane anesthesia,
subjects had increases in FCO₂ and Vᵢ/T₁ as FGF was
reduced. The FCO₂ resulting from a change in Vᵢ/T₁
was significantly related in a linear fashion to the

![Graph](image)

**Fig. 3.** (above), calculated inspired volumes of
CO₂ of nine patients breathing halothane and
oxygen, plotted against mean inspiratory flow rates
(Vᵢ/T₁) measured at various fresh gas flow (FGF) rates.
The significant linear correlation suggests
that the variability in the inspired CO₂ volume can
be well explained by a change in the Vᵢ/T₁ measured.

**Fig. 4.** (below), when the inspired volumes of CO₂
in nine patients breathing enflurane were plotted
against the simultaneous Vᵢ/T₁'s measured, no
significant linear correlation was found. The line of
best fit, drawn by hand, suggests that there was
no change in CO₂ inspired until Vᵢ/T₁ increases
were large.
magnitudes of the change in inspiratory flow rate (fig. 5A). When enflurane was used (fig. 5B), the fraction of CO₂ inspired (F_{CO₂}) decreased as V₀/T₁ increased at the low FGF rates studied. The difference between the slopes of these regression lines (fig. 5A and B) is significant (P < 0.001).

**Discussion**

**Respiratory Waveform**

The major differences in respiratory waveforms between halothane anesthesia and enflurane anesthesia were a longer inspiratory time (T₁) and a longer respiratory cycle time (T₀/T₁) when enflurane was used, as well as a significant pause at end-expiration with this agent. The mean inspiratory flow rate (V₀/T₁) at high FGF rates was lower with enflurane than with halothane. The importance of these waveform differences to the clinical anesthetist was apparent only when their influence on rebreathing of CO₂ in an anesthetic breathing system was considered.

During halothane anesthesia, all patients rebreathed significantly increased volumes of CO₂ at a FGF rate of 100 ml/kg/min. With enflurane anesthesia, the inspired CO₂ volume was not significantly greater at 100 ml/kg/min than at high FGF rates. With both agents, the increases in V₀ in response to an inspired CO₂ load resulted from increasing V₀/T₁ with a constant T₁/T₀.

In the presence of a very brief expiratory pause during halothane anesthesia, this increase in V₀/T₁ resulted in increased CO₂ entrainment per breath from the expiratory limb (figs. 1 and 3A). If the fraction of CO₂ inspired (F_{CO₂}) increased, then, in terms of gas exchange, the effectiveness of increasing V₀ (by increasing V₀/T₁) would be altered, because the desired increase in functional alveolar ventilation would not be fully achieved due to the increase in inspired CO₂. Figure 5A shows that when halothane anesthesia was used with a low FGF rate, the fraction of CO₂ in the inspired volume increased as the patient increased V₀/T₁ in an attempt to compensate for the inspired CO₂ load imposed by low FGF rates. However, if the ratio of inspired volume of CO₂ to tidal volume (F_{CO₂}) were to decrease as V₀/T₁ increased, then the increase in minute ventilation would result in the
desired increase in effective alveolar ventilation. With enflurane anesthesia, our patients could increase \( V_t/T_1 \) at low FGF rates, and when this occurred, the fraction of CO\(_2\) in each breath (\( F_{\text{CO}_2} \)) tended to decrease (fig. 5A).

The change in the fraction of CO\(_2\) in each tidal volume (\( F_{\text{CO}_2} \)) reflected not only the effect of altering FGF, causing an inspired CO\(_2\) load, but also the ability of the patient to compensate for this load by increasing \( V_E \). Therefore, in spite of their ability to increase \( V_E \) at a FGF rate of 100 ml/kg/min, patients breathing halothane inspired higher CO\(_2\) concentrations per tidal volume as they increased \( V_t/T_1 \), and subjects breathing enflurane did the opposite (figs. 5A and B).

Although the respiratory waveforms varied somewhat among patients, the differences between agents described (table 1) were large and predictable. These differences in waveform did not appear to be secondary to variations in depth of anesthesia or surgical stimuli. The end-tidal enflurane concentration, when expressed as a MAC multiple (0.97), was less than the comparable end-tidal halothane value (1.4). This may reflect an error in our assumed value of 1.68 per cent for enflurane MAC in our healthy subjects, as suggested by Knill et al. Such a discrepancy would not alter the clinical implications of this study, since all patients in both groups were lightly anesthetized and undergoing similar surgical stimuli. Some of the increase in \( V_E \) in patients breathing enflurane at low FGF rates late in the study period (two hours) may have been due to attenuation of the ventilatory depression produced by this agent over time.

The mechanisms by which anesthetic agents alter ventilatory responsiveness and timing in man are not understood. It is clear that inhalational anesthetics have different and selective actions on various aspects

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**Fig. 5A (above)**, when the changes in the fractions of CO\(_2\) inspired (inspired volume CO\(_2\)), or \( F_{\text{CO}_2} \), in patients breathing halothane were plotted against the changes in \( V_t/T_1 \) between measurements, a significant positive correlation was found. This suggests that as \( V_t/T_1 \) increased during halothane anesthesia, the fraction of tidal volume inspired containing CO\(_2\) actually increased; thus, as these patients respond to low fresh gas flow (FGF) rates with greater \( V_E \), they inspire progressively increasing concentrations of CO\(_2\).

**B (below)**, when the changes in the fractions of CO\(_2\) inspired (\( F_{\text{CO}_2} \)) in patients anesthetized with enflurane were plotted against the simultaneous changes in \( V_t/T_1 \), a significant negative correlation was found. This suggests that as \( V_t/T_1 \) increased during enflurane anesthesia, patients inspired progressively lower concentrations of CO\(_2\); thus, as these patients respond to low FGF rates with greater \( V_t \), they inspire decreasing concentrations of CO\(_2\).
of respiratory control. They cannot be regarded as nonspecific respiratory depressants, and by analyzing a given response of minute ventilation in terms of flow \((V/T_f)\) and timing \((T_f/T_{tot})\), as proposed by Motoyama et al., the large difference between agents was readily apparent.

This study shows that under clinical conditions the halothane waveform encourages rebreathing of \(CO_2\) with a modified Mapleson D breathing system at low FGF rates. These data explain some of the confusion in the earlier literature on spontaneous ventilation with low FGF rates using T-pieces. Many variables, including circuit structure, FGF rate, and respiratory waveform, determine the extent of rebreathing. The potential for predictability of the inspired \(CO_2\) load is limited by these variables. Spoerl et al. have reported that in patients breathing enflurane, \(V_E\) did not increase as FGF was reduced, whereas when halothane was used, increases in \(V_E\) comparable to those reported in this study were found. Our results indicate that these differences were due to the effects of respiratory waveform changes caused by the anesthetic agents used.

Is enflurane, then, a better agent to use with this type of circuit at low FGF rates? Although increased rebreathing of \(CO_2\) (\(F_{CO_2}\)) at low FGF rates (e.g., 100 ml/kg/min) is minimized by using enflurane, the \(P_{CO_2}\) remains high. No circuit can compensate for the hyperventilation resulting from anesthesia. Enflurane has been shown to be a more potent respiratory depressant than halothane. The \(P_{CO_2}\) values we observed are similar to those reported elsewhere for spontaneously breathing anesthetized patients.

The benefits of nonrebreathing at low FGF rates with enflurane anesthesia, due to an advantageous waveform, seem to be counterbalanced by the depressed ventilatory drive found even during light anesthesia with surgical stimulation. We previously demonstrated that when patients rebreathe \(CO_2\) at low FGF rates during halothane anesthesia, hypercapnia results. This hypercapnia is partially prevented by increasing the FGF rate. The best the anesthesiologist can do to prevent \(P_{CO_2}\) from increasing is to guarantee a minimal inspired \(CO_2\) load with any agent. This study shows that a prime determinant of the \(CO_2\) rebreathed in a Mapleson D breathing system is the patient's respiratory waveform. The FGF rates necessary to abolish rebreathing in individual patients vary considerably, depending on this waveform, which is uncontrollable during spontaneous respiration.

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