THE REGULATION OF Pa_{CO_2} DURING CONTROLLED VENTILATION OF CHILDREN WITH A T-PIECE

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ALTHOUGH the T-piece was introduced over 40 years ago, the optimal fresh gas flow rate for the use of the Mapleson D or E modifications during controlled ventilation is still controversial. Many of the formulae proposed appear to be contradictory and confusing. Suggested fresh gas flow requirements vary from 2.6 to 6.6 litres per minute for a 30 kg child.[1,2]

Recently the desirability of accurately regulating the arterial carbon dioxide tension during anaesthesia to prevent untoward changes in cerebral blood flow, cardiac output, respiratory control, blood H^+ concentration (pH) and electrolytes has been recognized. Bain and Spoerel have focussed attention on this issue in adults using their modification of the Mapleson D system deliberately converted to a partial rebreathing circuit by the use of low fresh gas flows.[3] Rayburn and Graves[1] and Nightingale, *et al.*[2] utilized the same principle in children during controlled ventilation and achieved acceptable arterial carbon dioxide levels with low fresh gas flows delivered either by a modified Mapleson D or the classical Ayre's T-piece.

There are two problems with these studies. First, the limitations of the statement that the arterial carbon dioxide tension is controlled by the regulation of fresh gas flow rate have not been defined. There are many factors affecting gas exchange in any patient during controlled ventilation with a T-piece (Table I). To achieve predictability of the carbon dioxide tension achieved in a given patient, one would need to eliminate or to minimize as many of these factors as possible. Secondly, fresh gas flow requirements must be expressed in a formula that is both convenient and useable over a wide range of patient sizes. In adults a convenient formula is available because the carbon dioxide production is directly proportional to body weight. In children there are no such simple formulae because of the curvilinearity of carbon dioxide production when related to body weights. Rayburn's solution, flow requirements related to body surface area (2500 ml·m^2) is relatively inconvenient.[1] The fresh gas flow proposed by Nightingale, a single value based on body weight (220 ml·kg^{-1}) underventilated the small infants but produced hypocarbia in older children.[2] Bain and Spoerel have modified their previous recommendation of a minimum 3.5 litres per minute fresh gas flow and now recommend that fresh gas flow be varied with changes in body weight in children as well as adults.[4]

TABLE I

VARIABLES DETERMINING Pa_{CO_2} DURING CONTROLLED VENTILATION WITH A T-PIECE SYSTEM

1*	FGF	fresh gas flow
2*	$\dot{V}E$	minute ventilation
3*	V_D/V_T	the ratio of apparatus and physiological dead space to tidal volume
4*	Waveform	delivered by the ventilator
5	$\dot{V}co_2$	carbon dioxide production
6	$a\text{-}AD_{co_2}$	arterial to alveolar carbon dioxide gradient

*Denotes variables evaluated in this investigation.

The theoretical basis for the approach used in this present study was developed by Conway, Seeley and Barnes in their mathematical analysis of the interrelationships of carbon dioxide tension and fresh gas flow in partial rebreathing systems.[5] They showed that an important parameter is the fraction of the fresh gas flow that actually participates in gas exchange, but did not determine this value in clinical practice.

The aims of this study of controlled ventilation with a T-piece were:

(a) to examine the influence of physiological and circuit variables (Table I) on carbon dioxide tension using a lung model to define the circumstances which produce maximal predictability of carbon dioxide tension.

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(b) to determine the fractional utilization of fresh gas in actual clinical practice in order to derive a practical formula for the calculation of fresh gas flow requirements in children.

(c) to validate the formula in a heterogeneous group of anaesthetized patients and to determine its limitations.

METHODS

(a) *Lung model study: influence of variables on carbon dioxide tension*

The lung model was a simple bag in box system[6] (Figure 1). A five-litre anaesthetic reservoir bag impermeable to carbon dioxide served as the "lung" when it expanded and contracted and the bottle, an airtight glass container, represented the thorax. Carbon dioxide was accurately metered into the bottom of the bag and mixed by a fan. Plastic tubing of variable lengths was used to add on a dead space volume equivalent in size to the sum of the physiological plus apparatus dead space of anaesthetized patients. When tidal volume was varied, this dead space volume was also adjusted so that it remained a constant fraction of the tidal volume over the whole range of minute ventilations investigated.[7] Fresh gas (air) was supplied to the T-piece circuit (a Bain circuit) from an accurately calibrated flow meter. Ventilation was controlled using an Air Shields constant volume ventilator. Complete mixing within the "lung" resulted in a flat end-expiratory plateau of carbon dioxide concentration which was measured with a mass spectrometer. Flow and volume were measured with a pneumotachograph and recorded along with the carbon dioxide concentration on a polygraph. Carbon dioxide production (180 ml· min^{-1}),[8] dead space to tidal volume ratio (0.4)[9] minute ventilation (frequency 12, tidal volume 700 ml), and waveform, (inspiratory to expiratory ratio 1:2 with a long expiratory pause[10]) were all appropriate for a 70 kg adult male.

The effect on alveolar carbon dioxide of different fresh gas flows, from 30 to 240 ml· kg^{-1}· min^{-1} was determined.

The influence of ventilation was isolated using a fixed fresh gas flow of 70 ml· kg^{-1}· min^{-1} and levels of alveolar carbon dioxide were measured as minute ventilation increased from 3 to 17 l· min^{-1}.

To isolate the effect of changing V_D/V_T on gas exchange, the alveolar carbon dioxide was determined for 3 V_D/V_T ratios (0.3, 0.4, 0.5) over a range of minute ventilations (4 to 12 l· min^{-1}) using a constant fresh gas flow of 70 ml· kg^{-1}· min^{-1}.

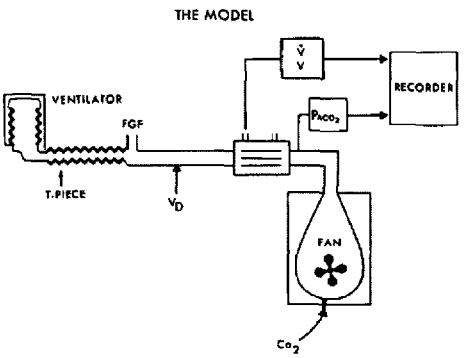

FIGURE 1 A compliant bag (the alveolar compartment) in a box (the thorax) and plastic tubing for deadspace (V_D) served as the model lung. Carbon dioxide was metered into the "alveoli" and mixed with a fan. Ventilation was controlled using an Air Shields Ventimeter ventilator attached to a T-piece circuit, with a source of fresh gas, air. Gas for carbon dioxide analysis by mass spectrometer was sampled near the junction of deadspace and alveolar compartments. A pneumotachograph was used for flow and volume measurements. All values were recorded on a polygraph.

Using different waveforms with I:E ratios from 2.5:1 to 1:4 the P_{ACO_2} was measured with a constant fresh gas flow (70 ml· kg^{-1}· min^{-1}) and minute ventilation of 8.4 l· min^{-1}.

To define a relationship which could give a constant and predictable P_{ACO_2} we determined many different combinations of fresh gas flow and minute ventilation all of which achieved the same level of alveolar carbon dioxide. This information was plotted on a graph of FGF vs $\dot{V}E$ so that an isopleth of constant alveolar carbon dioxide tension was described.

All results are expressed at ATPD.

(b) *Patient study*

Eight healthy supine patients (ASA I) age 1–16 (wt 11–64 kg) undergoing general anaesthesia for either cerebral angiography or plastic facial repairs were studied. Each was induced with sodium thiopentone (4 mg· kg^{-1}) atropine (0.02 mg· kg^{-1}) and succinylcholine (1 mg· kg^{-1}) and the trachea was intubated with a cuffed tracheal tube. Ventilation was controlled using an Air Shields Ventimeter ventilator, with inspiratory:expiratory ratio set at 1:2. Either Bain circuits or Ayre's T-pieces were used. These have been shown to function in an identical manner in the lung model.[6] Maintenance anaesthesia was achieved using muscle relaxants, meperidine or

halothane and a 50:50 nitrous oxide and oxygen mixture. Flows and volumes of ventilation were measured using a pneumotachograph of appropriate size and recorded on a polygraph. Arterial blood samples were stored on ice, analyzed in duplicate for pH, Pa_{CO_2} and Pa_{O_2} by Corning blood gas electrodes and corrected for body temperature, which was monitored using a rectal probe. For each patient minute ventilation and fresh gas flows were varied. At least 30 minutes after induction or after a change in the ventilation and fresh gas flows, an arterial blood sample was obtained and analysed. Carbon dioxide production using a T-piece circuit during steady state anaesthesia was calculated from the equation: $\dot{V}CO_2 = FGF \times Fv_{(CO_2)}$ [Ref 11, equation (1) where Fv_{CO_2} is the fraction of carbon dioxide in the expiratory limb – i.e. vented carbon dioxide fraction].

The vented carbon dioxide fraction was measured by a mass spectrometer with a catheter sampling at the end of the expiratory tubing. There was no interference with nitrous oxide or halothane using this instrument. From these measurements of Pa_{CO_2} FGF, Fv_{CO_2} and $\dot{V}E$, an optimized formula was derived for fresh gas flow and ventilation to achieve two levels of arterial carbon dioxide, 4.0 kPa (30 torr) and 4.9 kPa (37 torr).

To determine the accuracy of the performance of the ventilator, the volume set on the Air Shields Ventimeter (200–1000 ml) was compared with the actual volume delivered to the patient as measured from the integrated flow trace of the pneumotachograph. Observations included patients weighing from 11 to 64 kg.

The protocol of this study was reviewed by an institutional committee which concluded that specific consent was not required.

(c) Evaluation of formula for FGF requirements

Twenty-six patients were chosen at random from an operating-room list. In order to study a heterogeneous population, no attempt was made to standardize patient selection, anaesthetic agents or surgical procedures. Following intravenous induction with sodium thiopentone (4 $mg \cdot kg^{-1}$) atropine (0.02 $mg \cdot kg^{-1}$) and succinylcholine (1 $mg \cdot kg^{-1}$) each patient was intubated with a cuffed or uncuffed tracheal tube depending on the procedures involved. A mixture of 2:1 nitrous oxide and oxygen was delivered by either an Ayre's T-piece or a Bain circuit and ventilation controlled with an Air Shields constant volume ventilator. Maintenance of anaesthesia was achieved with halothane, or narcotics and muscle relaxants (d-tubocurare or pancuronium bromide) when necessary. Rectal temperatures were measured. Patient position was variable depending upon the surgical procedure. Each anaesthetist set the fresh gas flow and ventilation according to the formula recommended for either a Pa_{CO_2} of 4 kPa (30 torr) or 4.9 kPa (37 torr). The pattern of ventilation was set at will by the individual anaesthetist. Generally the tidal volume was greater than 10 $ml \cdot kg^{-1}$. The I:E ratio was approximately 1:2. In order to make the anaesthetic as routine as possible no attempt was made to measure accurate VE or flow patterns. Arterial blood samples taken at least 30 minutes after positioning the patient and controlling his ventilation were accurately analyzed by Corning blood gas electrodes for Pa_{CO_2}, Pa_{O_2} and pH and corrected for body temperature.

The accuracy of the anaesthetic machine flowmeters at 1, 3 and 8 litres per minute was determined using both a microflowmeter calibrated for nitrous oxide and oxygen and a water displacement spirometer.

Results and Discussion

(a) Lung model study: influence of variables on carbon dioxide tension

Fresh gas flow: Steady state levels of alveolar carbon dioxide are shown at FGF from 30 to 240 $ml \cdot kg^{-1} \cdot min^{-1}$ in Figure 2. At very high fresh gas flows, values for the alveolar carbon dioxide equalled those that would be achieved with a

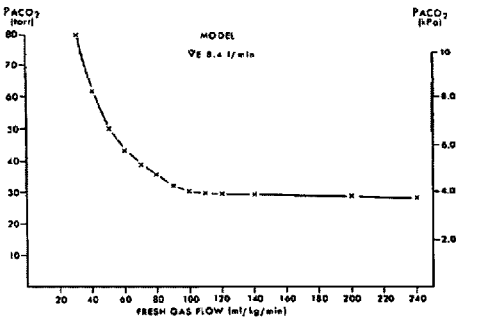

FIGURE 2 Relationship of alveolar carbon dioxide tension to fresh gas flow rates at a constant level of minute ventilation. ($\dot{V}CO_2$ = 180 $ml \cdot min^{-1}$, V_D/V_T = 0.4). At very high fresh gas flows the alveolar carbon dioxide values approach those that would be achieved with a non-rebreathing circuit, i.e. the alveolar carbon dioxide is ventilation-limited. At lower fresh gas flows the increases in carbon dioxide tension reflect rebreathing of expired gases.

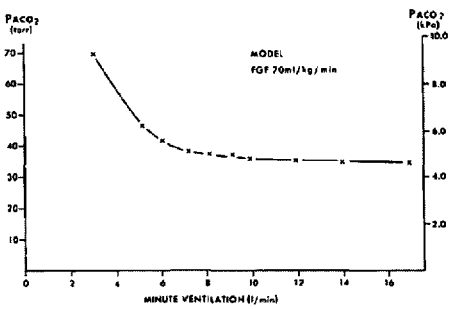

FIGURE 3 Relationship of alveolar carbon dioxide tension to minute ventilation at a fixed fresh gas flow ($\dot{V}CO_2$ = 180 ml·min^{-1}, VD/VT = 0.4). Initially, increasing $\dot{V}E$ caused the carbon dioxide tension to fall, but at high levels of ventilation a plateau was reached. At this point, further increases in ventilation served only to increase rebreathing and alveolar carbon dioxide became fresh gas flow limited.

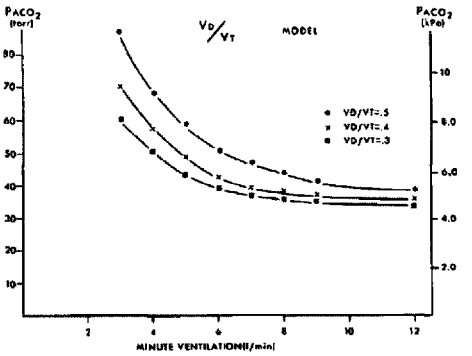

FIGURE 4 Alveolar carbon dioxide, minute ventilation relationships for three ratios of physiological dead space to tidal volume. ($\dot{V}CO_2$ = 180 ml·min^{-1}, FGF = 70 ml·kg^{-1}·min^{-1}. Large differences in carbon dioxide tension resulted from alterations in VD/VT at low levels of ventilation. These differences were minimized as minute ventilation increased.

non-rebreathing circuit. At this point the alveolar carbon dioxide was essentially ventilation limited. The increase in alveolar carbon dioxide at low fresh gas flows was due to the rebreathing of expired gases.

$\dot{V}E$: Alveolar carbon dioxide levels, measured as minute ventilation increased from 3 to 17 l·min^{-1} are shown in Figure 3. Increasing the minute ventilation initially caused the alveolar carbon dioxide to fall, but at high levels of minute ventilation a pleateau occurred and PA_{CO_2} was fresh gas flow limited. This limit was reached at the point where further increases in minute ventilation served only to increase rebreathing.

VD/VT: The dead space to tidal volume ratio is variable under anaesthesia, ranging from 0.3 in healthy children to 0.7 or more in older individuals with signs of obstructive lung disease.[12] Three VD/VT ratios (0.3, 0.4, 0.5) were selected and the alveolar carbon dioxide was measured over a range of minute ventilations (Figure 4). At low levels of minute ventilation changes in VD/VT caused marked changes in PA_{CO_2} similar in magnitude to the changes one would predict with a non-rebreathing circuit. The influence of VD/VT on alveolar carbon dioxide was minimized by high levels of minute ventilation and the rebreathing of carbon dioxide.

Waveform: Respiratory waveform, the flow pattern of gases during inspiration and expiration, affects the carbon dioxide tension achieved for any given fresh gas flow.[13] During the expiratory pause fresh gas clears carbon dioxide from the expiratory limb. A common pattern for controlled ventilation utilizes an inspiratory to expiratory ratio of 1:2, with a long expiratory pause. In the model (Figure 5) when the I:E ratio fell below 1:1, the PA_{CO_2} rose. Any pattern of ventilation which eliminates the expiratory pause will result in a higher carbon dioxide tension.

$\dot{V}CO_2$: This study has included only one level of carbon dioxide production, which is that appropriate for a 70 kg male adult. Carbon dioxide production in anaesthetized patients can be vari-

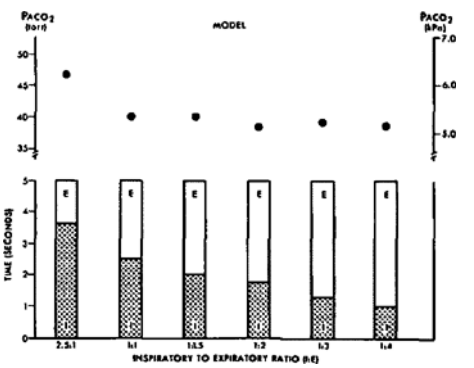

FIGURE 5 Influence of the ratio of inspiratory and expiratory period (I:E) on alveolar carbon dioxide tension when minute ventilation, FGF and $\dot{V}CO_2$ were all held constant. The total breath period of 5 sec was divided as indicated: stippled bars = inspiration, open bars = expiration. Resultant PA_{CO_2} tensions are plotted in the top panel for each I:E ratio. No increase in PA_{CO_2} occurred until the inspiratory period exceeded the expiratory period.

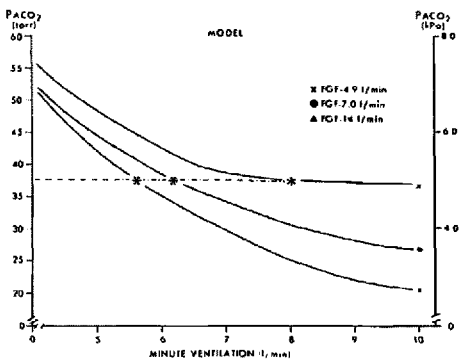

FIGURE 6 Relationship of alveolar carbon dioxide tension to minute ventilation ($\dot{V}CO_2$ = 180 ml·min^{-1}, VD/VT = 0.4) at FGF rates of 4.9, 7.0 and 14.0 l·min^{-1}.
*Indicates points where identical carbon dioxide tensions were achieved by three markedly different combinations of $\dot{V}E$ and FGF.

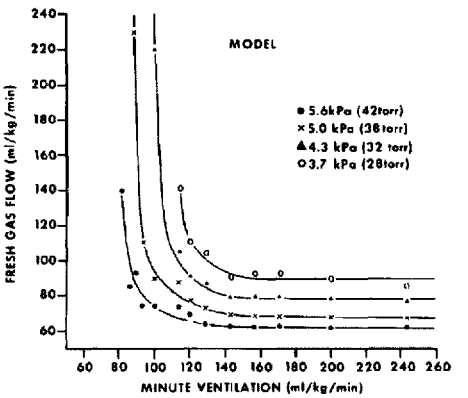

FIGURE 7 The interrelationship of fresh gas flow and minute ventilation is shown by four isopleths, each joining points of equal carbon dioxide tension. Each isopleth describes the multiple different combinations of $\dot{V}E$ and FGF that all produce the same net gas exchange. On the left, carbon dioxide tensions are influenced strongly by changes in $\dot{V}E$, but are insensitive to alterations in FGF. Towards the right-hand side changes in $\dot{V}E$ have little effect with carbon dioxide tensions, depending strongly on the fresh gas flow rate.

able.[14] Any increase in this level would result in a simple proportional increase in PA_{CO_2} as demonstrated in a model by Ramanathan, *et al.*[15]

a-AD_{CO_2}: This sixth factor could not be isolated in the model where all values of carbon dioxide tension are expressed as alveolar carbon dioxide. In patients a significant but variable a-AD_{CO_2} can exist under anaesthesia.[7] From the principles of ventilation and perfusion distributions one can predict that this a-AD_{CO_2} would be minimized if one deliberately chose to use large minute ventilations with rebreathing in which case ventilation to low $\dot{V}/\dot{Q}$ areas would be improved, but rebreathing would limit the effect of this increased ventilation in high $\dot{V}/\dot{Q}$ zones. Recommendations to achieve a particular carbon dioxide level will be more predictable under these circumstances.

Interrelationship of FGF and $\dot{V}E$: To approach this issue the alveolar carbon dioxide tension was plotted for three fresh gas flows (70, 100, 200 ml·kg^{-1}·min^{-1} over a range of minute ventilations from 4 to 10 litres·min^{-1} in Figure 6. On each of the three lines there was a point of identical PA_{CO_2}, 4.9 kPa (37 torr) achieved by three different combinations of minute ventilation and fresh gas flow. There are in fact many combinations of FGF and $\dot{V}E$ which result in the same PA_{CO_2} such that on a graph of FGF vs $\dot{V}E$ (Figure 7) isopleths of constant alveolar carbon dioxide tension can be delineated. Here isopleths of 3.9, 4.1, 4.5, 4.9 kPa (28.5, 32.1, 35.7, 39.2 torr) are shown. The very high fresh gas flows and low minute ventilations of a non-rebreathing circuit suggested by Mapleson[16] on the left side achieved the same alveolar carbon dioxide level as the low fresh gas flow and high minute ventilation proposed by Bain and Spoerel on the right. On the left, the region of high flow of dry gases, the PA_{CO_2} was determined by $\dot{V}E$, $\dot{V}CO_2$, and VD/VT ratio. On the right where controlled rebreathing of humidified gases existed, the PA_{CO_2} was determined by the fresh gas flow and $\dot{V}CO_2$ but changes in VD/VT were minimized.

(b) Patient study

Table II shows an example of carbon dioxide tensions obtained when different combinations of fresh gas flow and minute ventilation were used in a 12-year-old male weighing 40 kg anaesthetized for a cerebral arteriogram.

During the study the $\dot{V}CO_2$ measured was relatively constant but the ratio of ventilation to fresh gas flow changed fourfold. The minute ventilation was doubled from 5.5 to 11 litres per minute and the fresh gas flow halved from 11 to 5.5 l·min^{-1}. There was essentially no change in the arterial carbon dioxide tension (4.1 kPa, 31 torr). When these three experimentally determined points (Table II) are replotted on the graph of FGF vs $\dot{V}E$ (Figure 8) they fit the line described in the model (Figure 7). Both the FGF of 275

TABLE II

ARTERIAL CARBON DIOXIDE TENSIONS WITH VARYING COMBINATIONS OF $\dot{V}E$ AND FRESH GAS FLOW

$\dot{V}E$(l·min^{-1})	FGF(l·min^{-1})	$\dot{V}E$/FGF	$\dot{V}CO_2$(ml·min^{-1})	Pa_{CO_2}(kPa)
5.5	11.0	0.5	140	4.0 (29.4 torr)
8.5	5.5	1.55	165	4.1 (31.2 torr)
11.0	5.5	2.0	170	4.1 (31.4 torr)

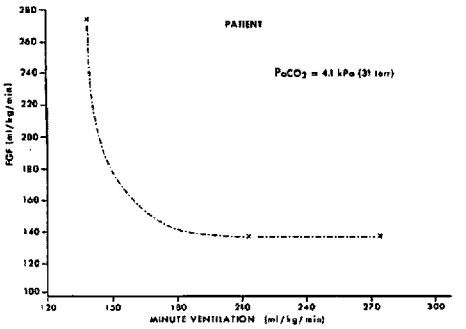

FIGURE 8 Plot of values of $\dot{V}E$ and FGF, all of which resulted in an arterial carbon dioxide tension of 4.1 kPa (31 torr) during anaesthesia in a 40 kg, 12 yr male patient with relatively constant carbon dioxide production. The shape of the smooth curve joining the points was based on the relationship established in detail in the lung model (Figure 7).

ml·min^{-1}·kg^{-1} on the left side and a FGF of 135 ml·min^{-1}·kg^{-1} on the right side achieved the same arterial carbon dioxide level. This indicates an obvious difference in the proportion of fresh gas supplied by the flow meters which actually ended up in the patient's alveoli. This concept that only a certain fraction of the fresh gas is available for gas exchange has been derived by Conway in his analysis of rebreathing circuits where

$Fa_{CO_2} = \dot{V}CO_2/(f \times FGF)$ ref 5
$= \dot{V}CO_2/\dot{V}A$ equation (2)
Fa_{CO_2} = arterial CO_2 fraction
FGF = fresh gas flow in ml·min^{-1} (ATPD)
f = fractional utilization of fresh gas flow
$\dot{V}A$ = alveolar ventilation ml·min^{-1} (ATPD)

The value $f \times$ FGF is simply that portion of the fresh gas which is available for gas exchange. It is equivalent to the effective alveolar ventilation in which case equation 2 becomes the familiar gas exchange equation. The remaining fresh gas ($1 - f$) is wasted down the expiratory limb and cannot contribute to gas exchange.

The magnitude and variability of the fractional utilization of fresh gas (f) could be calculated by dividing the vented carbon dioxide fraction by the arterial carbon dioxide fraction (from the combination of equations 1 and 2). Twenty-seven measurements of "f" were obtained from the eight children in the study, at different minute ventilation and fresh gas flow ratios (Figure 9). At low $\dot{V}E$/FGF ratios, less than 1.5, fractional utilization was small and unpredictable. When the $\dot{V}E$/FGF was greater than 1.5, "f" was relatively constant. Sixteen of the measurements were done above this ratio, and the mean value of "f" was 0.72 with a standard deviation of 0.046. The standard deviation in this measurement resulted from changes in a-AD_{CO_2}, VD/VT and waveform which existed not only between patients, but between individual measurements on the same patient.

There was no significant difference in the fraction of fresh gas utilized when delivered by the Bain circuit or the Ayre's T-piece (n = 4 each group, unpaired t-test). This confirmed in patients the work done earlier in the lung model.

Although the VD/VT ratio can increase markedly during hypotensive anaesthesia[17] the mean value of fractional utilization after the infusion of either pentolinium or nitroprusside in 11 of these patients ($f = 0.70 \pm 0.053$) was not significantly

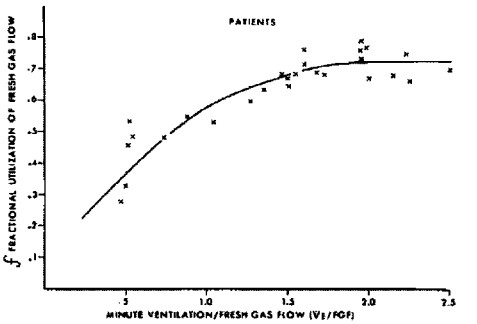

FIGURE 9 Variation of the fractional utilization of fresh gas (f) with the ratio of minute ventilation to fresh gas flow rate. Fractional utilization is low and variable at levels of $\dot{V}E$/FGF less than 1. Above a ratio of 1.5 f becomes virtually constant (mean 0.72 ± 0.05).

different ($p > 0.05$) from the value obtained during normotension. This confirms the conclusion from Figure 4 that changes in V_D/V_T are minimized by rebreathing at a high $\dot{V}_E$/FGF ratio (greater than 1.5).

Our fractional utilization is less than the value of 0.95 suggested by Rayburn's data.[1] This apparent conflict can possibly be explained by Rayburn's use of an infrared carbon dioxide analyzer in the presence of nitrous oxide and the absence of any allowance for water vapor in the conversion of fractional concentration of carbon dioxide to partial pressure. Both of these factors would tend to elevate measurements of expired carbon dioxide levels and lead to an overestimation of their utilization of fresh gas. To obtain a value of 0.95 one would require virtually instantaneous mixing of the carbon dioxide produced by the body with the total fresh gas flow. This did not occur in our study despite the use of ratios of $\dot{V}_E$/FGF similar to those of Rayburn. This limit to the degree of mixing is inherent in the use of cyclic ventilators. Patient studies are limited as to the maximum minute ventilation attainable but in our model when we increased the $\dot{V}_E$/FGF to 4:1 we were unable to show any improvement in the fractional utilization. The value "f" reflects the variability of $\dot{V}_E$/FGF, waveform and a-AD_{CO_2}. Because it was relatively constant (0.72 when $\dot{V}_E$/FGF was greater than 1.5 in the patient study) it was possible to predict the fresh gas flow necessary to achieve a particular arterial carbon dioxide level. The values of carbon dioxide production were taken from three studies; awake sedated children from 10–50 kg[18], anaesthetized children from 45–65 kg,[4] anaesthetized adults 60–100 kg.[19] For simplicity we have used the mean value of carbon dioxide production in males and females. These rates are within the 95 per cent confidence limits for carbon dioxide production of anaesthetized children from 10 to 60 kg described in a recent study by Nightingale and Lambert.[20] The fresh gas flow requirements have been determined for two

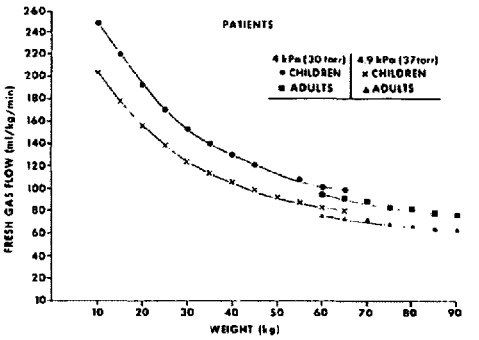

FIGURE 10 The predicted fresh gas flow requirements for two desired levels of carbon dioxide tension, plotted against body weight for children and adults. Calculations are based on values for carbon dioxide production from the literature[4,18,19] and f as determined in this study. This relationship is valid only if $\dot{V}_E$ is at least 1.5 × the FGF rate.

levels of arterial carbon dioxide, 4.9 kPa (37 torr) and 4 kPa (30 torr) in both children (> 10 kg) and adults (Figure 10). For adults fresh gas flows of 70 $ml \cdot kg^{-1} \cdot min^{-1}$ and 90 $ml \cdot kg^{-1} \cdot min^{-1}$ are adequate for these Pa_{CO_2} levels. These fresh gas flow levels agree well with data presented by Bain and Spoerel.[4] In children a marked curvilinearity arises because carbon dioxide production is disproportionately high in small children when related to body weight. However, this FGF requirement curve can be reasonably approximated by two straight lines divided at the 30 kg point. A two-part formula can then be derived for fresh gas flow requirements based on body weight (Table III). Accurate minute ventilation must be set at least 1.5 times the fresh gas flow where "f", the fractional utilization is constant or optimum predictability will not be achieved.

Discrepancies between the volume set on the bellows calibration and that delivered to the patient can arise from expansion of the ventilator tubing, any leak occurring around the tracheal tube and from the addition of fresh gas delivered

TABLE III

FRESH GAS FLOW REQUIREMENTS (CONTROLLED VENTILATION)

Weight	Predicted	Predicted
	Pa_{CO_2} 4.9 kPa (37 torr)	Pa_{CO_2} 4.0 kPa (30 torr)
10–30 kg	1000 ml + 100 $ml \cdot kg^{-1}$	1600 ml + 100 $ml \cdot kg^{-1}$
> 30 kg	2000 ml + 50 $ml \cdot kg^{-1}$	3200 ml ± 50 $ml \cdot kg^{-1}$

$\dot{V}_E$ (on the ventilator) must be twice this FGF.

from the anaesthetic machine during inspiration. With a non-rebreathing system, when carbon dioxide tension is ventilation limited, these discrepancies will make marked changes in the Pa_{CO_2} achieved. When the low fresh gas flows of the optimized formula were used and the Ventimeter was set to deliver a minute ventilation of twice the fresh gas flow (tidal volumes from 200–1000 ml) it was found that in all the patients (11–64 kg) $\dot{V}E$ was at least 1.5 times the FGF. Therefore, it is recommended that the minute ventilation set on a constant volume ventilator be at least twice the recommended fresh gas flow requirements (Table III). At this ratio maximum utilization of fresh gas is achieved and any further increases in $\dot{V}E$ will not change the Pa_{CO_2}.

(c) Assessment of the formula

Twenty-six patients selected at random were distributed as follows: 17 boys, 9 girls; 10< 20 kg, 10 between 20 and 40 kg, 6 > 40 kg; 14 ASA I, 9 ASA II, 3 ASA III. Seventeen were in the supine position, six prone, two in the lateral decubitus. Nine had cuffed tracheal tubes and the remainder uncuffed tubes.

The results verified the usefulness of the optimized formula (Figure 11). In 11 patients when a Pa_{CO_2} of 4 kPa (30 torr) was desired the actual Pa_{CO_2} was 4.5 ± 0.6 kPa (33.7 ± 4.3 torr). When the predicted Pa_{CO_2} was 4.9 kPa (37 torr) the measured Pa_{CO_2} in 15 patients was 5.3 kPa (39.9 torr ± 5.4 torr). Actual carbon dioxide tensions were slightly higher than the predicted level. This was due to a systematic error in the flowmeters on the anaesthetic machines used. The 11 machines used in the study delivered an average of 8.1 per cent less flow than that recorded on the flowmeters. The error, which ranged from +3 to −20 per cent was not dependent on the flow rate. These discrepancies were due to inaccurate rotameters or leaks within the anaesthetic machine.

No changes in heart rate or blood pressures were noted during the surgical procedures due to large volume ventilation. However, certain patients with hypovolaemia or cardiac dysfunction can have a significant fall in venous return with large tidal volumes. In one patient with gross splenomegaly, excessive airway pressures prevented the delivery of the required minute volume until the spleen was removed. A large leak around the tracheal tube of another patient caused the ventilator bellows to collapse and higher fresh gas flows were required to compensate. Total flows and the exact composition of

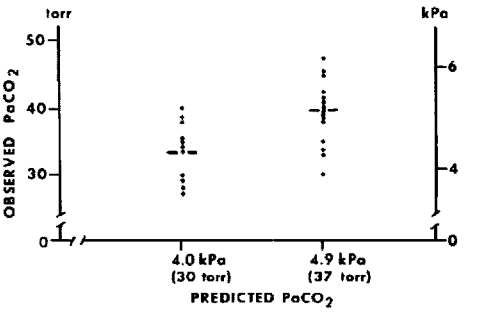

FIGURE 11 Arterial carbon dioxide tensions observed in a heterogeneous patient population when fresh gas flow was calculated from the formula derived in this study. Both mean values (horizontal line) and individual points are shown for predicted Pa_{CO_2} levels of 4.0 and 4.9 kPa (30 and 37 torr).

nitrous oxide and oxygen mixtures below 2 litres·min^{-1} are unreliable on standard anaesthetic machines; therefore children weighing less than 10 kg are not included in the formula. When it is not possible to use the optimized formula we suggest switching to a totally non-rebreathing system where Pa_{CO_2} is ventilation limited. If one chooses instead an intermediate combination of FGF and $\dot{V}E$ where fractional utilization becomes variable and Pa_{CO_2} depends on both these factors, then predictability will be lost.

The fresh gas flows required for Pa_{CO_2} 5.3 kPa (40 torr) in this study of children are higher than those suggested by Rayburn. Variations in the circuit, patient VD/VT, $\dot{V}E/FGF$ and fractional utilization cannot account for this. However patients in Rayburn's study were heavily premedicated with meperidine and pentobarbitone. Since patients in our study received no premedication it is highly probable that differences in carbon dioxide production explain this discrepancy. Total fresh gas flows required for normocarbia in our children are similar to those recommended by Bain and Spoerel with their theoretical calculations and graph of flow requirements based on body weight.[4] Our study emphasizes that these recommendations will only be valid when ventilation is controlled at twice the fresh gas flow, and are not applicable to spontaneous ventilation.

In this study of a heterogeneous population of 26 patients variations in carbon dioxide production, fresh gas flow and "*f*", the fractional utilization of FGF all account for the rather large standard deviation in the measured Pa_{CO_2}. No practical formula can eliminate this error. The

estimate of carbon dioxide production was based on measurements in healthy individuals. Drugs, patient sex, fever, sepsis, movement and environmental temperatures can all cause wide variation in the normal value. Errors in FGF as high as 20 per cent have been shown to occur even in well serviced anaesthetic machines. Only with controlled rebreathing and a ratio of $\dot{V}E/FGF$ greater than 1.5 are deviations in fractional utilization minimized.

Therefore there is an unavoidable limit on the accuracy of predicting the Pa_{CO_2} during anaesthesia. Although one may set the flowmeter and ventilation according to the optimized formula, it will only be a guide. For very precise control of Pa_{CO_2}, there is no alternative but to measure it directly.

Summary

A lung model study described the factors determining the carbon dioxide tension during controlled ventilation with a T-piece system (either a Bain circuit or the classical Ayre's T-piece). When minute ventilation was large with respect to fresh gas flow and a standard ventilatory pattern was utilized the important variables were fresh gas flow (FGF) and carbon dioxide production ($\dot{V}CO_2$). Under these circumstances the influence of minute ventilation ($\dot{V}E$), dead space to tidal volume ratio (VD/VT), waveform and $a\text{-}AD_{CO_2}$ on carbon dioxide tension were minimized.

Eight healthy patients (11–64 kg) were studied during controlled ventilation to determine how much of the fresh gas coming from the flowmeters actually participates in gas exchange. With a T-piece system when minute ventilation to fresh gas flow ratio is at least 1.5 the patient's effective alveolar ventilation equals 72 per cent of the total fresh gas flow. This value and levels of carbon dioxide production from the literature were used to derive a simple two part formula of fresh gas flow requirements for two levels of arterial carbon dioxide in children. Children weighing between 10 and 30 kg require fresh gas flows of $1000 + 100\ ml \cdot kg^{-1} \cdot min^{-1}$ and $1600 + 100\ ml \cdot kg^{-1} \cdot min^{-1}$ to achieve arterial carbon dioxide tensions of 4.9 kPa (37 torr) and 4.0 kPa (30 torr) respectively. When body weight is between 30 and 70 kg the requirements increased to $2000 + 50\ ml \cdot kg^{-1} \cdot min^{-1}$ to achieve a Pa_{CO_2} of 4.9 kPa (37 torr) and $3200 + 50\ ml \cdot kg^{-1} \cdot min^{-1}$ to achieve a Pa_{CO_2} of 4.0 kPa (30 torr). In all situations $\dot{V}E$ must be at least twice the fresh gas flow requirements.

The formula was validated in a heterogeneous group of 26 patients. Mean values of Pa_{CO_2} were very close to those predicted, although individual values differed considerably from the mean. These deviations cannot be eliminated because of patient and equipment variability. Therefore blood gas analysis must be performed whenever the exact regulation of Pa_{CO_2} is required.

Résumé

Les facteurs modifiant la Pco_2 durant la ventilation contrôlée avec pièce en T (conventionnelle ou circuit de Bain) ont été étudiés sur un modèle mécanique de poumon. Avec un rapport ventilation-minute sur apport de gaz frais élevé et une ventilation normalisée pour ce type de montage, les facteurs importants étaient le volume de gaz frais et la production de CO_2. Dans ces conditions, l'influence de la ventilation-minute, du rapport espace mort sur air courant, du rapport inspiration-expiration et du gradient alvéolo-artériel en CO_2, étaient minimisés.

Huit patients en bonne santé (d'un poids de 11 à 64 kg), ont été étudiés en respiration contrôlée pour établir la participation des gaz frais aux échanges gazeux. Avec un système en T lorsque le rapport ventilation-minute / apport de gaz frais est d'au moins 1.5, 72 pour cent des gaz frais participent chaque minute à la ventilation alvéolaire. Nous avons utilisé ce résultat ainsi que les taux de production de CO_2 rapportés dans la littérature pour formuler une équation simple permettant de connaître les débits de gaz frais nécessaires pour maintenir deux taux de CO_2 chez l'enfant.

Les enfants pesant de 10 à 30 kg ont besoin d'un apport de $1000 + 100\ ml \cdot kg^{-1} \cdot min^{-1}$ en gaz frais pour maintenir une Pa_{CO_2} de 4.9 kPa (37 torr), et de $1600 + 100\ ml \cdot kg^{-1} \cdot min^{-1}$ pour maintenir celle-ci à 4 kPa (30 torr). Lorsque le poids est de 30 à 70 kg, l'apport de gaz frais doit être de $2000 + 50\ ml \cdot kg^{-1} \cdot min^{-1}$ pour maintenir une Pa_{CO_2} de 4.9 kPa et de $3200 + 50\ ml \cdot kg^{-1} \cdot min^{-1}$ pour la maintenir à 4 kPa. Dans tous les cas, la ventilation-minute doit être au moins deux fois plus grande que l'apport de gaz frais.

Nous avons fait la vérification de cette formule chez 26 patients. Les Pa_{CO_2} moyennes observées étaient proches des valeurs prédites, bien que l'on ait observé des variations individuelles importantes. Ces variations ne peuvent être éliminées à cause de la diversité des patients ainsi que de l'équipement. Par conséquent, il demeure nécessaire de faire l'analyse des gaz artériels

lorsque le contrôle précis de la Pa_{CO_2} est important.

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