A New Concept in Controlled Ventilation of Children with the Bain Anesthetic Circuit

MAJ Robert L. Rayburn, M.C., U. S. Army,* and Shirley A. Graves, M.D.†

The Bain anesthesia circuit was studied as a semi-open or partial rebreathing system during controlled ventilation in 16 children weighing from 7.5 to 48 kg. During anesthesia the lungs were ventilated with a volume ventilator set at three times the calculated alveolar ventilation to provide optimum mixing in the exhalation tube of the Bain circuit. Fresh gas inflow rates initially were set equal to the calculated alveolar ventilation, and after 30 to 45 min, P CO2, P O2, and pH values were measured. At the same time, the fractional concentration of mixed expired carbon dioxide (F E CO2) was recorded from a capnograph inserted between the ventilator and the Bain circuit. After initial readings, the fresh gas inflow was varied over a range of 1,400–3,000 ml/m²/min at 20-min intervals, with the arterial blood-gas values and F E CO2 recorded at each setting. The results indicate that a lower fresh gas inflow than previously recommended can be used safely in children. When the minute ventilation is three times the fresh gas inflow, values for F E CO2 correlate closely with P a CO2; with a fresh gas inflow of 2,500 ml/m²/min, F E CO2 values can be maintained near 40 torr. (Key words: Anesthesia, pediatric; Equipment, circuits, Bain, semi-open; Ventilation, controlled.)

The Bain anesthetic system is used frequently for pediatric patients. Bain showed that with controlled ventilation, a fresh gas inflow of 70 ml/kg/min is adequate for the system to function as a partial rebreathing or semi-open system with arterial carbon dioxide tensions (P a CO2) remaining within normal limits. Since his studies did not include pediatric patients, Bain suggested a minimum fresh gas inflow of 3.5 l/min be used for patients weighing less than 50 kg.† [See addendum.] An inflow of that magnitude converts the circuit into a nonrebreathing system and, thus, minute ventilation, not fresh gas inflow, determines P a CO2. We examined the use of the Bain circuit as a semi-open system during controlled ventilation in children.

* Staff Anesthesiologist, Brooke Army Medical Center.
† Associate Professor of Anesthesiology and Pediatrics, University of Florida College of Medicine.

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Address reprint requests to Major Rayburn: Anesthesia and Operative Service (AFZG-MDS-A), Main Hospital, Brooke Army Medical Center, Fort Sam Houston, Texas 78234.

Methods

We studied 16 healthy children weighing 7.5 to 48 kg who underwent operative procedures not involving the chest or upper abdomen. Premedication consisted of atropine, 0.02 mg/kg (maximum, 0.6 mg), meperidine 1 mg/kg (maximum, 100 mg), and pentobarbital 4 mg/kg (maximum, 100 mg), administered intramuscularly. Blood pressure, pulse, electrocardiogram, and temperature were monitored throughout the study. Patients were anesthetized with nitrous oxide and oxygen (2:1) and halothane, and were paralyzed with d-tubocurarine. The trachea was intubated, and ventilation was controlled using the Bain circuit with a minute ventilation approximately three times the predicted alveolar ventilation. Alveolar ventilation required to maintain P a CO2 at approximately 40 torr during anesthesia was calculated using the following equation:

\[ \dot{V}_A \text{ Standard} = 20 \times \dot{V}_{CO2} \text{ STPD} \]

where \( \dot{V}_A \) = minute alveolar ventilation; \( \dot{V}_{CO2} = \) minute carbon dioxide production (assumed to be 100 ml/m²/min).

Ventilation was provided using a volume ventilator (either the Ohio 300 D/O pediatric ventilator or the Air Shields ventilator—ventilator). The elbow adaptor of the Bain circuit was removed to decrease dead space, and the tidal volume of 15 ml/kg was measured at the endotracheal tube using a Wright respirometer. Fresh gas inflow rates were adjusted with microflowmeters to the calculated values of 1,400–3,000 ml/m²/min. A Godart capnograph was inserted between the Bain circuit and the ventilator to measure continuously the fractional concentration of mixed expired carbon dioxide (F E CO2), which was recorded using a Grass polygraph. After 35 to 45 min at the initial inflow (which was usually equal to the calculated alveolar ventilation), and every 20 min after changing the inflow, an arterial blood sample was drawn for measurement of P a O2, P a CO2, and pH. These blood samples were drawn into heparinized syringes that were tightly capped and transported to the blood-gas laboratory in ice. All values were corrected for body temperature. At the same time blood-gas and pH values were determined, F E CO2 was recorded from the capnograph and was multiplied by 760 torr to obtain the partial pressure of mixed expired carbon dioxide (P E CO2).

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Results

Fifty-two pairs of determinations were performed. Arterial carbon dioxide tension varied inversely with the fresh gas inflow in all 16 children (fig. 1). The least-square regression line indicates that a fresh gas inflow of 2,500 ml/m²/min correlates with a PaCO₂ of 40 torr in these children (r = 0.6, P < 0.001).

Mixed expired carbon dioxide tension was also inversely related to fresh gas inflow (fig. 2). The lines of regression show that PEECO₂ is closely correlated with, but slightly lower than, PaCO₂ at a given inflow rate (r = 0.56, P < 0.01). The means are 38.2 and 42.2 torr, respectively.

Figure 3 is a typical tracing of FEECO₂. When fresh gas inflow was increased, FEECO₂ decreased immediately and within 6 min was almost at the new equilibrium of 0.042. A very slight further decrease occurred over the next 15 min. When inflow was decreased to its initial level, FEECO₂ returned to the baseline value of 0.052 within 6 min.

Discussion

We found that lower fresh gas inflows than those recommended by Bain¹ and other investigators⁶,⁷ could be used to achieve normocapnia in a wide age range of children on the Bain (or Mapleson D-type) circuit with controlled ventilation. The discrepancy between our results and those of Bain, Nightingale, and Mapleson is due to the different amounts of carbon dioxide retention achieved in the exhalation arm of the circuit by the various investigators. This retention, and therefore rebreathing, of carbon dioxide is dependent on the ratio of ventilation to fresh gas inflow. This ratio has ranged from practically no retention of carbon dioxide in Mapleson's theoretical study to essentially maximal retention in our study. The reports by Bain and Nightingale are based on carbon dioxide retention intermediate between that of Mapleson and that in our study. Normal arterial carbon dioxide values have resulted using each of these methods.

Our study was precipitated by the observation that
the percentage of carbon dioxide measured at the end of the Bain circuit near the reservoir bag in Bain's study was approximately 3 per cent. It appeared to us that if a homogeneous mixture of gases could be assured in the exhalation arm of the circuit, then a carbon dioxide concentration more nearly approaching the \( P_{\text{aco}_2} \) value might be allowed to exist in the exhalation arm of the circuit. This would allow lower fresh gas inflows and still maintain normal arterial carbon dioxide tensions.

In an attempt to assure this homogeneity of gases, we used the concept of a time constant, which is defined as the length of time required for the flow through a container to equal the volume of the container. We therefore considered the amount of fresh gas inflow into the lung and circuit to be analogous to volume, and the ventilation of the lung, which passes through the circuit, to be analogous to the flow described in the time-constant definition.

Since three time constants provide a 95 per cent mixing of gases, we postulated that essentially continuous (95 per cent) mixing of the fresh gas inflow with the ventilation gases would occur in the exhalation tube of the Bain circuit if ventilation equalled three times the fresh gas inflow. We recognize that the idea of time constants is based upon exponential washin or washout equations for certain conditions, but for convenience we prefer to use the term "mixing" as if it were synonymous with "washout."

We then assumed that the mean carbon dioxide tension for all the gases contained in each tidal volume of respiratory gas in the lung closely approximated the \( P_{\text{aco}_2} \) value. If we had good mixing we might expect the \( F_{\text{e}CO_2} \) value measured at the ventilator end of the circuit to be similar to the \( P_{\text{aco}_2} \) value. We did find a close correlation between \( P_{\text{aco}_2} \) and \( F_{\text{e}CO_2} \) values, presumably due to this mixing in the circuit (fig. 2).

It was also found that \( F_{\text{e}CO_2} \) values had a relatively constant relationship to \( P_{\text{aco}_2} \) values over various inflow rates. A change in fresh gas inflow was reflected almost immediately in a change in \( F_{\text{e}CO_2} \). This is presumably due to the fact that any change in \( P_{\text{aco}_2} \) values is rapidly reflected at the ventilator end of the circuit due to the mixing of gases.

With our method of ventilation, it simply becomes a matter of using the fresh gas inflow to eliminate continuously from the circuit that volume of gas with a particular \( F_{\text{e}CO_2} \) value equal to the patient's minute carbon dioxide production. For a \( P_{\text{aco}_2} \) of 40 torr, this was calculated to be 2,000 ml/m²/min. In actuality, 2,500 ml/m²/min (fig. 1) was required, probably as a result of a combination of factors, including alveolar dead space, uptake of anesthetic gases by the patient, a respiratory quotient of less than one, and a low assumed value for carbon dioxide production.

The 90 per cent confidence limits for the \( P_{\text{aco}_2} \) values ranged from 32 to 49 torr at a fresh gas inflow of 2,500 ml/m²/min. In our patients, no \( P_{\text{aco}_2} \) value outside a range of 36–47 torr was observed at fresh gas inflow rates between 2,400 and 2,600 ml/m²/min. Subsequent experience with patients using 2,500 ml/m²/min inflow has shown the \( P_{\text{aco}_2} \) values to cluster around 40 torr. Although this fresh gas inflow is recommended, it should be noted that a few patients
may deviate from the normal range for $P_{acO_2}$, as may occur with any method of ventilation.

The correlations of $P_{CO_2}$ values and fresh gas inflow with $P_{acO_2}$ values show some scatter. They are, nevertheless, sufficiently accurate to be clinically useful in estimating $P_{acO_2}$. A number of factors should be considered in assessing the scatter about the lines of regression. The general accuracy of the capnograph (which can be read no more accurately than ± 0.1 per cent), the errors in blood-gas analysis, and the limits of the accuracy with which the microflowmeters may be set may all cause some dispersion. Bain has shown that the depth of halothane anesthesia influences carbon dioxide production. Because of very low vaporizer settings, it was difficult to be certain that the depths of halothane anesthesia were the same in all patients. It is generally agreed that muscle activity increases carbon dioxide production. The adequacy of muscle relaxation was not tested in every case. However, the patient having the highest $P_{acO_2}$ value per inflow rate was observed to be paralyzed inadequately during the study. Data on carbon dioxide production in children are sparse; nevertheless, a value of 100 ml/m²/min was selected on the basis of various sources. The error resulting from this inaccuracy and variability of carbon dioxide production in these children, therefore, cannot be assessed.

The difference between $P_{CO_2}$ and $P_{acO_2}$ values, as shown in figure 2, is relatively small. The difference between the means of the two sets of values, 38.2 and 42.2 torr, respectively, is 4.0 torr. Part of this discrepancy might be explained by the fact that theoretically only 95 per cent mixing was achieved. However, this value is very close to the mean arterial and end-tidal carbon dioxide tension difference of 4.6 torr reported by Nunn, and our difference may be a reflection of the factors mentioned in his paper. It appears that under the conditions of our study, the measurement of $P_{CO_2}$ probably correlates as closely with $P_{acO_2}$ as does the measurement of end-tidal carbon dioxide during conventional ventilation.

In our study, the minute ventilation was set initially at three times the calculated alveolar ventilation, with the initial fresh gas inflow equal to the alveolar ventilation. As the fresh gas inflow was varied, ventilation was not so that the percentages of mixing of the exhaled gases probably varied slightly. For good mixing, ventilation should be at least equal to three times the fresh gas inflow.

In conclusion, fresh gas inflow rates considerably lower than those previously recommended for the Bain circuit can be used safely in children receiving controlled ventilation. Minute ventilation should be three times fresh gas inflow to afford good mixing in the circuit and, thus, provide a close correlation between $P_{CO_2}$ and $P_{acO_2}$. Under these conditions, fresh gas inflows of $2,500$ ml/m²/min will maintain $P_{acO_2}$ near 40 torr.

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**Addendum**

Since acceptance of this paper for publication, Bain has published an article supporting our recommendations, Can Anaesth Soc J 24:533-539, 1977.

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