

LETTERS TO THE EDITOR

SIR,

During the past few years a series of articles have been published concerning the performance of a modified Mapleson D system (Bain, J.A. & Spoerel, W.E., *Canad. Anaesth. Soc. J.*, 19: 426 (1972); 20: 629 (1972); 22: 34 (1975). As interest in this circuit is now being shown on this side of the Atlantic, it is of interest to point out that there appears to be a considerable fallacy in the theory on which Bain and Spoerel have based their treatment of this circuit.

Bain and Spoerel repeatedly claim that in a system which allows complete mixing of fresh gas inflow with alveolar gas, alveolar carbon dioxide concentration (F_{ACO_2}) will be determined by the ratio of carbon dioxide production (\dot{V}_{CO_2}) and fresh gas inflow (\dot{V}_F), so that

$$F_{ACO_2} = \dot{V}_{CO_2} / \dot{V}_F$$

This equation was first postulated by Mapleson (1958) for the Magill (Mapleson A) circuit during spontaneous ventilation with a fresh gas flow equal to alveolar ventilation. In this circuit gas vented from the circuit is specifically alveolar gas in a volume equal to V_F . Thus $\dot{V}_{CO_2} = \dot{V}_F \times F_{ACO_2}$. Nunn and Newman (1964) confirmed this postulate and further showed that in all circuits which did not selectively vent alveolar gas (F_{ACO_2}) would exceed $\dot{V}_{CO_2} / \dot{V}_F$.

If complete gas mixing occurs within any circuit then the gas vented will have a composition equal to inspired (not alveolar) gas. A simplified analysis, assuming an equilibrium state and assuming inspired and expired alveolar volumes to be equal, shows in fact that

$$F_{ACO_2} = \dot{V}_{CO_2} (1/\dot{V}_F + 1/\dot{V}_A)$$

Thus if V_F here is made equal to predicted alveolar ventilation, actual alveolar ventilation must be raised to infinite levels to maintain a normal F_{ACO_2} . Bain and Spoerel claim that their original equation only holds if ventilatory volume exceeds fresh gas flow. However, it appears from their articles that total ventilation would rarely be greater than twice the fresh gas flow.

Whilst not doubting that normocarbica can be achieved with the Mapleson D circuit and many other circuits at low fresh gas flows if controlled ventilation is increased to adequate levels, it is certain that neither the postulate of total gas mixing nor the oft quoted equation of Bain and Spoerel have any validity. The economy of the Mapleson D system during controlled ventilation resides in the ability of the tubing to act as a reservoir for *unmixed* fresh gas. The performance of the circuit can only be assessed mathematically if ventilatory pattern and flows are known. Unfortunately, we in this department have found these authors' descriptions of their methodology so ambiguous as to defy analysis.

C.M. CONWAY

Professor of Anaesthetics

Magill Department of Anaesthetics,
Westminster Medical School,

London S.W.1, England.

REFERENCES

- MAPLESON, W.W. Theoretical considerations of the effects of rebreathing in two semi-closed anaesthetic systems. *Brit. med. Bull.*, 14: 64 (1958).
NUNN, J.F. & NEWMAN, H.C. Inspired gas, rebreathing, and apparatus dead space. *Br. J. Anaesth.*, 36: 5 (1964).

SIR:

We appreciate the opportunity to reply to Dr. C.M. Conway's remarks concerning the theoretical assumptions which we made in regard to the mode of carbon dioxide elimination of our modified Mapleson D. system.

In our evaluation of this breathing circuit we were able to show that, with controlled ventilation in excess of the patient's calculated minute volume, the P_{aCO_2} could be changed predictably by altering the fresh gas inflow; normocapnia could be maintained with a fresh gas inflow of the same magnitude which Kain and Nunn (*Anesthesiology* 20: 964, 1968) reported for the Magill system and patients with an increased CO_2 production showed an elevated P_{aCO_2} with our recommended fresh gas inflow of 70 ml/kg/min which returned to normal with a higher fresh gas flow. Since our modified circuit behaved similar to the Magill circuit we felt justified in assuming that the relationship between fresh gas inflow, CO_2 output and alveolar or arterial P_{CO_2} should be the same.

The formula $P_{aCO_2} = V_{CO_2}/VF$ established only the general relationship between the three parameters involved and does not consider the pattern of flow and distribution of gases within the circuit. Although Mapleson applied this formula to the Magill circuit in spontaneously breathing patients, this relationship should pertain to all rebreathing circuits and we don't consider it a fallacy to make this assumption on the basis of our findings.

However, we have also shown that the P_{aCO_2} calculated on the basis of this formula coincides only with observed values within a range of near normal CO_2 tension; an increased fresh gas inflow produced a less than predicted fall in P_{aCO_2} and a reduction of the inflow led to less CO_2 accumulation than calculated (*Can. Anaesth. Soc. J.* 22: 34, 1975). This observation was reported earlier by Baraka (*Brit. J. of Anaesth.* 41: 527, 1969); unfortunately his interesting paper came only to our attention after our report was printed and we must apologise to Dr. Baraka for this oversight. It would appear that there is a modifying factor to the formula for fresh gas inflows below or in excess of the volume of alveolar ventilation. We are not aware of a corresponding observation with the Magill circuit in controlled ventilation but would suspect similar results; Scholfield and Williams (*Brit. J. Anaes.* 46: 442, 1974) reported the same finding with a circle system without CO_2 absorption.

We have stated that the volume of fresh gas inflow will determine the P_{aCO_2} only if the minute volume of ventilation is greater than the fresh gas inflow; we have generally ventilated our patients with a tidal volume of about 10 ml/kg/min at a rate of 12-16 per minute. If the volume of ventilation falls below the volume of fresh gas inflow, the volume of ventilation will determine CO_2 elimination. A fresh gas inflow above the patient's minute volume of ventilation does not significantly affect the P_{aCO_2} .

Our assumption of complete mixing within the circuit was based on our observation, that the CO_2 content of gas vented from the expiratory valve was nearly constant i.e. there was virtually no difference between inspiration and expiration. The CO_2 concentration in vented gas was higher than the lowest inspired concentration measured in the endotracheal tube, and appeared to represent a mean between inspired and expired (alveolar) CO_2 levels.

Dr. Conway believes that the performance of our circuit can only be assessed mathematically if ventilatory flows and pattern are known; this must be questioned in view of our observation that the same fresh gas inflow which will maintain normocarbia in patients on controlled ventilation will also maintain a normal Pa_{CO_2} in patients breathing spontaneously if anaesthetic respiratory depression is avoided. We are not convinced that the pattern of ventilation has a significant influence on CO_2 elimination from the circuit.

Our modified circuit is now used exclusively in all hospitals attended by members of the Department of Anaesthesia of the University of Western Ontario (who conduct over 40,000 anaesthetic procedures annually) for all operations under general anaesthesia and in all age groups with controlled and spontaneous ventilation. Frequent monitoring of blood gases has confirmed the adequacy of CO_2 elimination with the fresh gas inflows we recommended and in clinical practice it works well. "How it works" (to quote from a letter by Dr. Philip Ayre—Anaesthesia 22: 359, 1967), "or what exactly goes on inside its lumen is still a mystery even after 30 years." Perhaps Dr. Conway might explain it mathematically; since he finds our reported data inadequate for his analytical appraisal, we can only suggest that he make his own observations.

W.E. SPOEREL, Professor and Chairman
J.A. BAIN, Clinical Associate Professor
Department of Anaesthesia
University of Western Ontario, London, Canada.

SIR,

It is hoped that the report by Paterson and Vanhooydonk¹ on "Improper Connection of the Bain Circuit" is the last we shall see on this topic. The proper functioning of every Bain circuit should be checked by Pethick's test² before use. This quick and simple test, demonstrating collapse of the reservoir bag when the oxygen flush button is pressed, proves that the circuit is correctly assembled. In the situation described in this case and also the one reported by Hannallah and Rosales,³ the reservoir bag is seen to inflate.

If Pethick's test had been applied in these cases, a fault in the circuit would have been discovered. This could then have been identified and corrected before the anaesthetic was started and the near disasters thereby avoided.

J.R. MALTBY, M.B., B.CH., F.F.A.R.C.S., F.R.C.P.(C),
Foothills Hospital, Calgary, Alberta.

REFERENCES

1. PATERSON, J.G. & VANHOODYDONK, V. A hazard associated with improper connection of the Bain breathing circuit. *C.A.S.J.* 22: 373 (1975).
2. PETHICK, S.L. Correspondence. *C.A.S.J.* 22: 115 (1975).
3. HANNALLAH, R. & ROSALES, J.K. A hazard connected with re-use of the Bain circuit: a case report. *C.A.S.J.* 21: 5 (1975).

DEAR DR. GORDON:

We recently discovered a cause of obstruction to the inflow of fresh gas into a "Montreal" pediatric circuit. This set had been in use for some time. The addition of a plastic right-angle connector between the "T" piece and a straight endotracheal tube connector, resulted in obstruction to inflow. It was discovered that the plastic elbow male 15 mm. fits very deeply into the "T" piece to the full depth of the taper, to occlude the fresh gas inlet. (Figure 1). Other connectors do not fit in so deeply, and so do not block the side entry.

We recommend that the "T" piece collar be extended to prevent recurrence (Figure 2).

Yours very truly,
F.C.HALEY, F.R.C.P.(C),
Department of Anaesthesia,
University of Alberta Hospital,
Edmonton, Alberta.