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THE RE-BREATHING TECHNIQUE

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The principle of deliberate re-breathing of anaesthetic atmospheres was introduced by McKesson in 1910 with the intention of conserving the gas in the end-inspiratory dead space which normally is of the same composition as the ambient atmosphere inhaled by the patient. Although the reason for the adoption of this method was primarily economic, McKesson pointed out that in addition 'as much gas containing CO₂ may be administered as desired',⁷ claiming, on the basis of work by Yandall Henderson, that this prevented the development of shock from acapnia which the rapid respirations of the anaesthetic state produced.

McKesson's unique anaesthetic machine was constructed with 'an adjustable graduated re-breathing bag by which the volume of gas breathed at each respiration may be measured, and by which any measured portion of this gas may be re-breathed at the following respiration'.⁷ This precise control of the volume of gas which is re-breathed is immensely important, for if the volume is too great, not only is there an increase in the CO₂ content but there is also a decrease in the O₂ content of the gas inhaled by the patient. Both these factors are critical in the method of anaesthesia which McKesson popularized, for precise control of the O₂ content of the inhaled mixtures of N₂O and O₂ was the point upon which the success or the failure of an administration pivoted. There is little doubt that the untoward effects of N₂O discussed so ably by Courville³ are due to anoxic anoxia, and that frequently the accidents and fatalities in patients held deliberately on gas mixture containing 10% or less of O₂ by volume were due to decreases in the O₂ tensions of the gas mixtures produced by excessive re-breathing.¹⁰ The hyperpnoea induced by hypoxia and hypercarbia would naturally increase the physiological dead space, further contributing to the unfavourable conditions.

Portability, the need for economy in anaesthetic gases, and provision for peak inspiratory flow rates, governed the design of the Magill attachment⁸ with its reservoir bag. In principle (see Fig. 1) there should only be re-breathing

of conserved gas in the end-inspiratory dead space when this attachment is correctly used. But while the McKesson apparatus provided for precise manual adjustment of the volume re-breathed, the Magill attachment (and all similar apparatus provided with a reservoir and using a constant rate of flow of fresh gas) relies, for control of the volume re-breathed, on alterations of respiratory volumes and of the rate of flow of fresh gas. An increase in the flow of fresh gas or a decrease in the respiratory ventilating volume will reduce the volume re-breathed and *vice versa*.

Measurement of the flow of fresh gas is obligatory in very many types of anaesthetic machine with which the Magill attachment may be used, in order to determine the proportion of oxygen that is being administered from the machine. The flow of fresh gas thus constitutes a variable which is directly under the control of the anaesthetist. An arbitrary rate of total flow is chosen for the majority of patients.

But the respiratory ventilating volumes are not under the control of the anaesthetist unless he is using artificial respiration. They vary from minute to minute in the same patient and, as a rule, they are never measured. In setting his arbitrary total flow of fresh gas for an individual patient the anaesthetist hopes that the patient's respiratory minute volume will not exceed it. Apparently basing their estimates upon normals of 11-14 breaths per minute and 500-600 ml. per breath for average adults at rest,² anaesthetic manuals recommend rates of total fresh gas flow between 5 and 8 litres per minute.

Molyneux and Pask⁹ have pointed out that ventilating volumes of 14 litres per minute and more are not uncommon during N₂O-O₂-ether anaesthesia. Indeed, the addition of the 'mechanical dead space' of the face-piece, connections and expiratory valve stock (often as great as 100 ml.) to the existing anatomical dead space of the patient, by reducing the effective alveolar ventilating volume must lead to hypercarbia. A decrease in alveolar ventilating volumes will reduce the efficiency of CO₂ wash-out from the alveoli,

and the resulting hypercapnia will lead to a compensatory increase in tidal volume or in rate or both. If the flow of fresh gas is not increased *pari passu*, the degree of re-breathing will in turn be further increased.

The depression of the respiratory centre by pre-medicating drugs and by anaesthetic agents may produce the extreme case where tidal volume is equal to dead-space volume. Under such conditions (see Fig. 2) no oxygen reaches the blood and no CO_2 can be excreted from the body except by the inefficient mechanism of 'diffusion respiration' described by Draper and Whitehead.⁴ The anaesthetized patient lies always between normal states and this extreme, and hardly ever (unless vigorous artificial respiration is practised) on the opposite or respiratory alkalotic side of normal. Little wonder, then, that anaesthetic drugs have so long been accused of producing an acidosis.

We have already reported that the failure to adjust for the widespread individual variations in respiratory minute volumes when the Magill attachment is used led to excessive re-breathing and accumulation of CO_2 .⁵ At the proximal end of the Magill attachment a CO_2 content of 0.53 vols. % was found when the flows of fresh gas were 8 litres per minute. In 2 experiments hyperventilation produced values of the order of 4 vols. % CO_2 at this same sampling point. These experiments were conducted on unpremedicated conscious volunteers, breathing air; for the determination of CO_2 tensions in anaesthetic gas mixtures is very difficult, the more so since the molecular weights of N_2O and CO_2 are the same.

As the complex and expensive equipment for such estimations is not available to us, an indirect attempt to deduce an increase or a decrease in the CO_2 content of anaesthetic atmospheres has been undertaken. This study is based upon the assumption that, as alterations in CO_2 tension in the blood form the most delicate and efficient regulator of respiratory ventilating volumes, the measurement of such volumes under varying conditions might provide a clue to the CO_2 tensions. Planned as a comparative study only, the work is not intended to provide a qualitative or a quantitative measurement of CO_2 tensions in anaesthetic atmospheres.

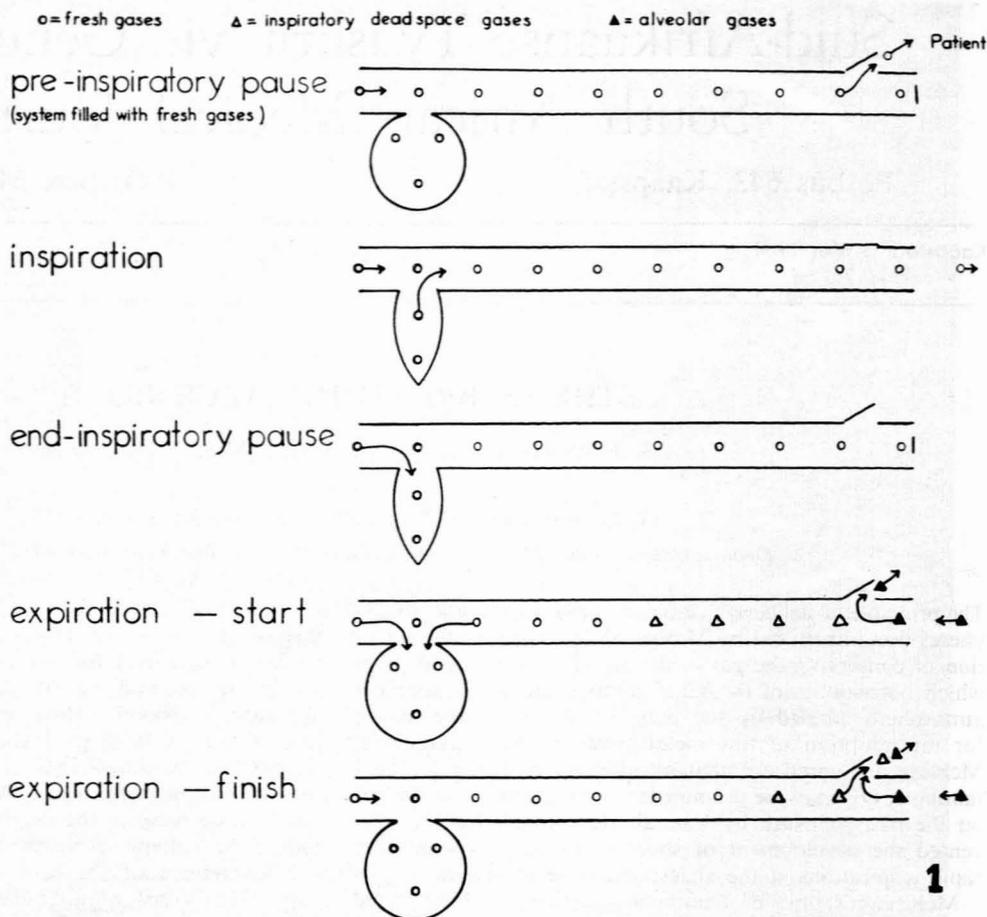


Fig. 1. Principle of the Magill attachment. If the total flow of the fresh gases is exactly equal to the respiratory minute volume, then, starting with the system filled with fresh gas, the patient will re-breathe only the conserved end-inspiratory dead-space gas, which is virtually fresh.

METHOD

Respiratory minute volumes were measured in 2 groups of patients receiving thiopentone- N_2O - O_2 -ether anaesthesia for intra-abdominal operations, extra-abdominal operations and operations upon the head, neck and extremities. No selection was exercised beyond excluding any patient for whom the use of a muscle relaxant became necessary. Usual forms of pre-medication were employed.

In the first group the patients received the anaesthetic in a routine fashion from a Boyle's machine and Magill attachment. The rates of flow of fresh gas were held constant from start to finish at 2 litres per minute for O_2 and 6 litres per minute for N_2O . Thiopentone (2.5% solution) was used in divided intravenous doses to assist induction (see below).

In the second group a Gillies mark III machine was used. Unidirectional valves at the face-piece prevented any re-breathing. The Magill attachment was used to conduct gases to the patient, but the reservoir bag now could only accumulate fresh gases flowing from the machine during the expiratory phase. The gas flows were varied so that the reservoir bag was never more than one-third filled.

This was done to prevent the flow of gas through the measuring system during the expiratory phase. The O₂ content of the fresh gas was always held at 25% or more. Peak respiratory demands were met by permitting draw-over of air through the machine. The whole volume of the expiratory phase was exhausted to room air.

Gas volumes were measured with a Siebe-Gorman dry-flow gas meter calibrated in one-tenth litres and litres and multiples thereof. This gas meter was inserted on the inspiratory limb of the circuit at all times.

As the schematic representations of the circuit demonstrate in Fig. 3, it was easy to insert the gas meter on the inspiratory side in the 'non-rebreathing group' but as the meter measures flow in one direction only, some adaptation was required to measure inspiratory volumes in the group on the Magill attachment. Here the unidirectional valves at the face-piece were also used, but the exhalations were led back to the Magill attachment at a point just distal to the expiratory valves. They were thus able to pass back towards the reservoir bag and escape from the valve in the usual way. As the unidirectional valves and flow meter were interposed between the expiratory valve and the face-piece but did not permit any re-breathing within their own circuit, their added volumes did not lead to any increase in the mechanical dead-space or in the volumes re-breathed. They did add fractionally to the resistance and they introduced a time lag, for the gas re-breathed was that which had been exhaled by the patient several breaths earlier.

In general the respiratory ventilating volumes were measured for the whole period of the anaesthetic and were subsequently divided by elapsed time in order to determine the mean respiratory minute volume. Since the metabolic rate, and hence the respiratory functions, is related more closely to surface area than to height alone or weight alone, the mean respiratory minute volume was in turn divided by the patient's surface area (derived from height and weight

$$\text{Alveolar ventilating volume} = \text{Tidal volume} - \text{Dead space volume}$$

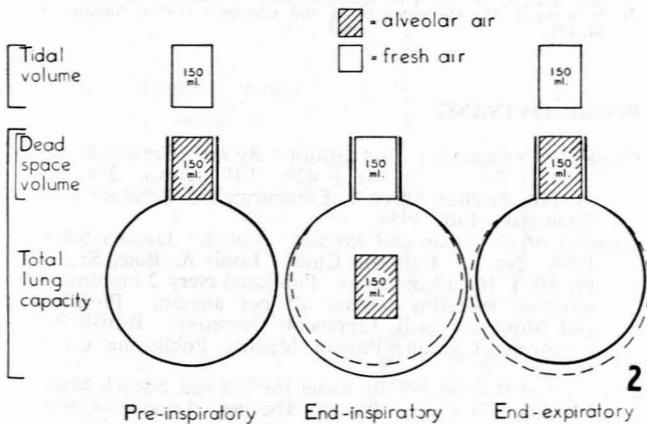
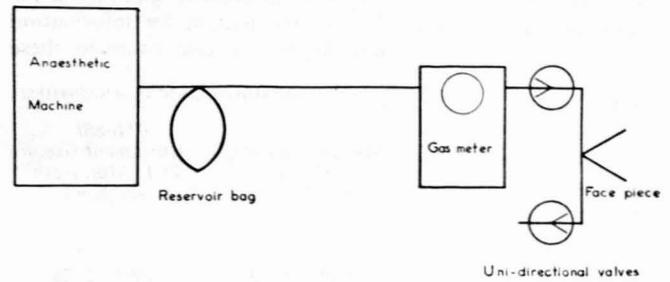


Fig. 2. Effective pulmonary ventilation. End-inspiratory dead-space air is fresh air and is exhaled as such. End-expiratory dead-space air is alveolar air and is re-inhaled unchanged in composition. Thus the dead-space air does not affect the composition of alveolar air. To determine the volume of gas which adds oxygen (or anaesthetic) to, and receives CO₂ from, alveolar air, the dead-space volume must be subtracted from the tidal volume. Multiply by rate to determine the effective ventilation per minute. In this example tidal volume is equal to dead-space volume and alveolar ventilation is nil.

NON REBREATHING



MAGILL ATTACHMENT

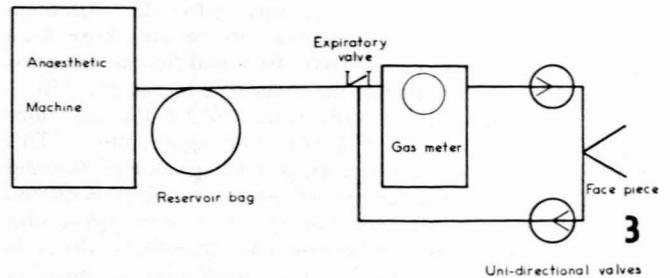


Fig. 3. Gas Flow Diagrams. Upper: Non-rebreathing circuit. Lower: Circuit for the Magill attachment.

by reference to an appropriate table¹) and the resultant was expressed in terms of litres of gas per square metre of body surface area per minute (l./sq.m./min.).

It was originally intended to use a N₂O-O₂-ether technique only, but the first attempt to achieve this with the non-re-breathing circuit underlined very graphically the reason for the popularity of the McKesson 'fractional re-breathing technique' and the Magill attachment. It served also to strengthen the validity of the assumption on which this study was based and it emphasized the work of Haggard⁵ on the importance of the volume of breathing during the induction of ether anaesthesia. Without the respiratory stimulation which the re-breathing of exhaled CO₂ provided, it proved almost impossible to induce anaesthesia with N₂O-O₂-ether within a reasonable period of time. The use of thiopentone to assist the induction of anaesthesia with the non-re-breathing technique became obligatory and, to balance the groups, the use was extended to all patients. The total dose of thiopentone seldom exceeded 500 mg. and the maximum used in any single patient was 700 mg. over a period of 2½ hours. With the assistance of thiopentone the non-re-breathing technique provided very satisfactory anaesthesia and excellent muscular relaxation in light planes of anaesthesia. A gastric resection was one of the operations performed under this form of anaesthesia.

RESULTS

The results are set out in Table I. It must again be emphasized that this work was undertaken as a comparative

study. The flow-meters on the Boyle and Gillies anaesthetic machines were not re-calibrated to determine their accuracy, nor was the gas meter. Because in clinical anaesthesia the anaesthetist must depend upon the patient for information concerning his height and weight or else estimate these

TABLE I. PULMONARY VENTILATION DURING CLINICAL ANAESTHESIA

	<i>Non-Re-breathing Group</i>	<i>Magill Attachment Group (8 L/Min. fresh gas flow)</i>
Mean Ventilating Volumes (in litres per square metre of body surface per minute) ..	4.18±1.80	5.62±2.24
Number of subjects ..	23	22
Age range (years) ..	13-86	15-75
Height range (inches) ..	58-72	58-74
Weight range (lb.) ..	95-300	95-195

All subjects received routine pre-anaesthetic medication and were scheduled for elective operations.

parameters, the latter practice was followed. Although checks usually proved the guesses to be not more than 1 inch or 2-3 lb. below or above the actual figures, the error in these data is probably of the order of 2 inches and 5 lb.

The mean respiratory ventilating volume for the 'non-re-breathing' group was 4.18 ± 1.80 l./sq.m./min. This corresponds closely to the average resting value of between 3 and 4 l./sq.m./min. for normal adult men which is quoted by Comroe.² It suggests that the respiratory depression produced by the pre-medicating and anaesthetic drugs is approximately cancelled by the respiratory stimulation which increase of dead-space (by the addition of the mechanical dead-space of the face-piece) normally produces.

The mean respiratory ventilating volume for the 'Magill attachment' group was 5.62 ± 2.24 l./sq.m./min. This represents an increase of 34% over the mean for the 'non-re-breathing group', and suggests that, after the effects of the added mechanical dead-space and of drug-induced alterations to respiration have been allowed for, there is a

large increase in ventilating volumes. We feel that this increase is almost certainly due to an increase in the CO₂ content of the anaesthetic atmosphere arising from accumulation of exhaled gas other than the end-inspiratory dead-space gas.

While the increase in CO₂ is of technical value in the induction of anaesthesia with N₂O-O₂-ether it must be remembered that it is a product of 2 variables, the flow of fresh gas and the respiratory minute volume. Unless the former, which is under the control of the anaesthetist, is held approximately equal to the latter, CO₂ may accumulate to toxic levels.

SUMMARY

Unless precisely controlled, re-breathing of anaesthetic atmospheres may lead to hypoxia and to accumulation of CO₂.

In an attempt to deduce the presence or absence of CO₂ in anaesthetic atmospheres without recourse to elaborate and costly instruments, comparative studies were undertaken on 2 groups of patients during clinical anaesthesia.

Respiratory minute volumes measured in the group anaesthetized by means of the Magill attachment with re-breathing were 34% greater than those for the group anaesthetized by means of a 'non-re-breathing' technique.

It is felt that this indicates a considerable degree of CO₂ accumulation during the use of the Magill attachment when an arbitrary total flow of 8 litres of fresh gas per minute is administered to the patient.

REFERENCES

- Best, C. H. and Taylor, N. B. (1955): *The Physiological Basis of Medical Practice*, 6th ed. Baltimore: Williams and Wilkins Co.
- Comroe, J. H. and Forster, R. E. (1955): *The Lung: Clinical Physiology and Pulmonary Function Tests*. Chicago: The Year Book Publishers.
- Courville, C. B. (1939): *Untoward Effects of Nitrous Oxide Anesthesia*. California: Pacific Press Publishing Association.
- Draper, W. B. and Whitehead, R. W. (1944): *Anesthesiology*, 5, 262.
- Haggard, H. W. (1924): *J. Biol. Chem.*, 59, 795.
- Harrison, G. G. and Jones, C. S. (1955): *Brit. J. Anaesth.*, 27, 162.
- McKesson, E. I. (1911): *Surg. Gynec. Obstet.*, 13, 456.
- Magill, I. W. (1930): *Brit. Med. J.*, 2, 817.
- Molyneux, L. and Pask, E. A. (1951): *Brit. J. Anaesth.*, 23, 81.
- Schwartz, C. H., Alexander, M. H. and Adriani, J. (1953): *Anesthesiology*, 14, 437.