Fresh Gas Economics of the Magill Circuit

Martin L. Kain, M.D., and*
John F. Nunn, M.B., Ch.B., Ph.D., F.F.A., R.C.S.†

The fresh gas flow rate required to prevent rebreathing in the Magill circuit has been determined in 16 spontaneously-breathing patients anesthetized with nitrous oxide–oxygen–halothane. Rebreathing was defined as an increase in minute volume or tidal volume in the presence of an end-tidal CO_2 concentration which rose or remained unchanged, or *vice versa*. Rebreathing caused substantial increases in ventilation, but observed rises in P_{CO_2} were seldom of clinical importance. The minimum flow of fresh gas adequate to prevent rebreathing varied from 3.1 to 4.6 l./min., with a mean of 3.6 (S.D. 0.63) l./min. Minute volumes of respiration at these levels of fresh gas flow ranged from 3.4 to 8.8 l./min., with a mean of 5.3 (S.D. 1.47) l./min. The mean value for the ratio of minimum adequate fresh gas flow to minute volume was 0.71 (S.D. 0.17). This finding agrees well with the theoretical prediction of Mapleson that rebreathing would be prevented by fresh gas flow rate equal to the alveolar ventilation.

* Research Fellow, supported by U. S. Public Health Service Special Fellowship 1-F3-GM-31, 742-01, National Institute of General Medical Sciences.

Dr. Kain was a resident in anesthesiology at the Presbyterian Hospital, New York, under Dr. E. M. Papper, after which he went to the University of Leeds, England, in 1966 as a Ph.D. student in the Department of Anaesthesia. He was killed together with his wife, his son and his wife's parents in a motor accident in Holland on 9th August 1967.

A preliminary presentation of this study was made to the Royal Society of Medicine (London),[20] and since then a similar study[21] carried out with the Magill circuit on conscious subjects has confirmed our general conclusions about the relationship between fresh gas flow rate and minute volume at the onset of rebreathing. Using a mass spectrometer, these authors found changes in $P_{E'O_2}$ corresponding to those of $P_{E'CO_2}$.

† Professor and Chairman, Department of Anaesthesia, University of Leeds.

Received from the Department of Anaesthesia, University of Leeds, Leeds, England. Accepted for publication March 4, 1968.

THE SEMICLOSED SYSTEM, popularly known as the Magill circuit, was improvised by Sir Ivan Magill about forty years ago, as a practical means of connecting an endotracheal tube to a source of nitrous oxide.[1,2] The circuit consists of a reservoir bag, a length of corrugated tubing, and a relief valve adjacent to the patient (fig. 1). It is extensively used in Britain and other parts of the world to supply gases to anesthetized patients who are breathing spontaneously.

Apart from general theoretical remarks on semiclosed systems by Wynne[3] in 1941, the Magill circuit had to wait until 1951 for the first attempt at analysis of its function by Molyneux and Pask.[4] The assumption by these authors that avoidance of rebreathing requires a fresh gas flow rate high enough to prevent any expired gas from travelling back towards the reservoir bag was refuted by Domaingue[5] in a letter which contained the first explanation of the basis of the efficiency of the circuit. However, there has been no study of the efficiency of the Magill circuit under the conditions in which it is actually used. Therefore, we have examined factors influencing the onset of rebreathing when the Magill circuit–patient system was used with spontaneously-breathing patients under surgical anesthesia.

Methods

Theoretical Basis

Rebreathing may be said to be present when the mixed inspired gas reaching the alveoli contains a concentration of CO_2 greater than could be accounted for by the alveolar gas reinhaled from the patient's anatomical deadspace.[6] Measurement of mean inspired concentration of CO_2 (with respect to volume inspired) is a valid approach employed by Woolmer and Lind[7] in a model system, but the measurement is far more difficult in the anesthetized patient, although it could probably be carried out with a proportional sampler.[8]

Another method of detecting rebreathing follows from examination of the simplified alveolar air equation for CO_2:

$$F_{ACO_2} = F_{ICO_2} + \frac{\dot{V}_{CO_2}}{\dot{V}_A}$$

Initially, rebreathing will cause an increase in the mean inspired CO_2 concentration (F_{ICO_2}). Assuming that CO_2 output ($\dot{V}_{CO_2}$) remains constant, there must be a rise of either alveolar ventilation ($\dot{V}_A$), alveolar CO_2 concentration (F_{ACO_2}) or both of these variables.

Simultaneous rises in F_{ACO_2} and $\dot{V}_A$ can only be caused by the addition of CO_2 to the inspired mixture, or by an increase in total deadspace (of which rebreathing from an anesthetic circuit is one example), or by an increase in CO_2 output. Increases in ventilation due to such factors as surgical stimulus cause an approximately reciprocal reduction in F_{ACO_2}.

These relationships are shown graphically in figure 2. The left-hand curve shows the effect of ventilation on P_{CO_2} (without rebreathing), and is based on experimental data from anesthetized patients breathing spontaneously.[9] The right-hand curve is the predicted relationship between ventilation and CO_2 when inspired CO_2 has been increased to 2 per cent, as by rebreathing. If ventilation is fixed, as with controlled respiration or very deep anesthesia, alveolar CO_2 rises 2 per cent (A to B). However, the usual response is an increase in both variables (line AC), which is, in fact, the P_{CO_2} ventilation response curve.

Criteria of Rebreathing

Our criteria of rebreathing were based on observed changes in ventilation and end-expiratory gas concentrations, and we required one or more of the following conditions to be fulfilled:

1. A rise in minute volume or tidal volume of 10 per cent or more, not accompanied by a corresponding fall in end-expiratory P_{CO_2} ($P_{E'CO_2}$) *or*

2. An increase in $P_{E'CO_2}$ (or a fall in $P_{E'O_2}$) of 5 mm. Hg or more, which could not be accounted for by a decrease in ventilation *or*

3. An increase in ventilation of 5 per cent or more, accompanied by an increase in $P_{E'CO_2}$ of 2 mm. Hg or more.

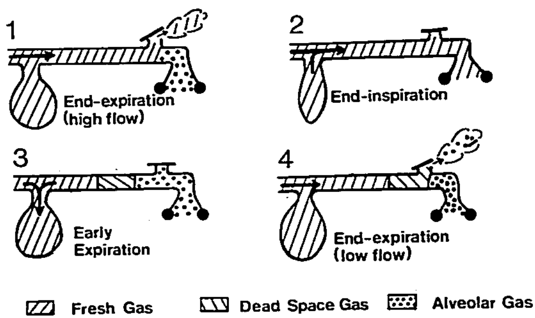

FIG. 1. Sequence of events during a single respiratory cycle with the Magill circuit (spontaneous respiration). In *1*, the circuit is shown purged of expired gas as far forward as the relief valve. In *2*, inspiration has been completed and the circuit is filled with fresh gas. During the early part of expiration (*3*) deadspace gas, followed by alveolar gas, passes into the wide-bore tubing until the reservoir bag is full and the relief valve opens. During the later part of expiration further exhaled alveolar gas passes directly through the relief valve while gas lying in the wide-bore tube is purged by oncoming fresh gas which cannot enter the reservoir bag. Maximal economy is attained when all the alveolar gas is vented but the patient's deadspace gas is retained.

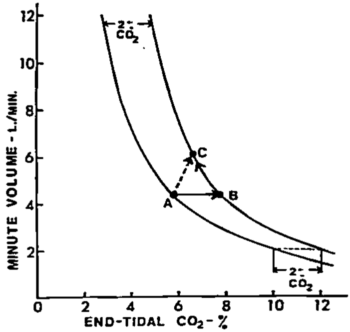

FIG. 2. The left-hand curve shows the effect of changes of ventilation on alveolar F_{CO_2}. Inhalation of 2 per cent carbon dioxide displaces the curve to the right. If ventilation is fixed the change is *A* to *B*. If ventilation is free to respond to the increased F_{CO_2}, the change *B* to *C* occurs. The overall response is then *A* to *C*, which is the carbon dioxide/ventilation response curve.

In fact, the trace of the inspired P_{CO_2} usually failed to return to zero when one of the above criteria was fulfilled but, due to the great difficulty of interpreting such a change in terms of the mean P_{CO_2} of the inspired gas (in respect to volume), these changes have not been presented.

Experimental Arrangement

A pre-mixed cylinder of N_2O/O_2 fed through a calibrated rotameter to a Fluotec vaporizer (fig. 3). A number of cylinders were used; these were prepared manometrically in our department: oxygen concentrations varied from 27 to 34 per cent. Halothane concentration from the Fluotec was monitored with a Hook and Tucker ultraviolet halothane analyzer. The conventional Magill circuit consisted of a two-liter reservoir bag, a one-meter length of corrugated tubing, and a spring-loaded relief valve. The valve opened towards the end of expiration at a pressure just above 1 cm. water, which was the pressure conducted from the reservoir bag to trigger the end-tidal sampler.[10]

Expired minute volume was measured either with a direct-reading Wright respirometer or with an automatically-recording version of a similar instrument[11] placed between the patient and the relief valve. Both instruments were calibrated for cyclical, intermittent gas flows and were found to show the errors introduced by variations in minute volume and breathing pattern previously described by Nunn and Ezi-Ashi.[12] Minute volume measured by these instruments cannot be expressed under standard physiologic conditions (ATPD or BTPS) because the effective temperature at which the gas is measured is between body and ambient, and depends on breathing pattern. No correction for temperature has been applied in our data. The deadspace of each instrument (measured by water displacement) is about 22 ml. Although this is not negligible, it was the same for each measurement.

Gas from the patient's airway was sampled through a polyethylene catheter (length 60 cm., 1.5 mm. in diameter), the tip of which lay at the tracheal end of an endotracheal tube, or at the pharyngeal end of an oropharyngeal airway, in patients who were not intubated. The proximal end of the catheter was connected to a tap, which allowed the gas to pass to a continuously-recording Hartmann and Braun URAS 4 rapid infrared CO_2 analyzer. The analyzer was calibrated with a specially-prepared mixture of nominal 5 per cent CO_2 in an N_2O/O_2 mixture similar to the one administered to the patient. The actual CO_2 concentration of the calibrating gas was determined with a Stow-Severinghaus P_{CO_2} electrode calibrated against CO_2/O_2 gas mixtures analyzed by the Lloyd-Haldane apparatus. The flow rate of the analyzer sampling pump was decreased in each case to the lowest rate which yielded a satisfactory alveolar plateau: this level was 300 ml./min. in most cases. The alveolar plateau was examined in the written record early in each study. In patients in whom the alveolar plateau was abnormal in shape or slope, the study was abandoned. In such patients, interpretation of the exhaled CO_2 record is difficult.

In some cases, gas was also passed by the end-tidal sampler to a Servomex OA-150 paramagnetic oxygen analyzer, for determination of end-expiratory oxygen concentrations. Gas withdrawn from the circuit–patient system for analysis was not returned to the circuit, but the nominal fresh gas flow rate was corrected for these losses in the analysis of the results.

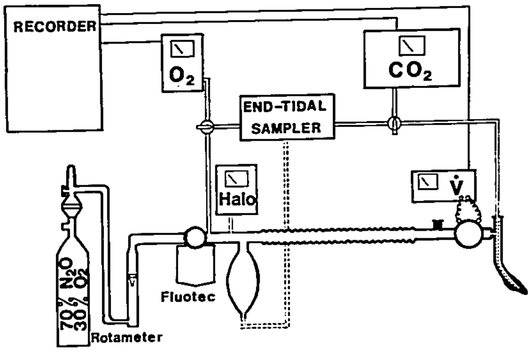

FIG. 3. Arrangements of analytical apparatus in relation to the gas circuit.

Signals from the infrared analyzer, oxygen analyzer and minute volume meter were recorded on a Devices pen oscillograph. Respiratory frequency was obtained from the CO_2 analyzer record. Tidal volume was derived indirectly by dividing minute volume by frequency. Fresh gas flow rate was initially set at a level sufficiently high for small changes to have no influence on ventilation or end-expiratory P_{CO_2}. The flow rate was then decreased stepwise until one or more of the above criteria of rebreathing were fulfilled. Each new level of fresh gas flow was maintained until steady values for ventilation and end-expiratory gas concentrations were obtained, except when gross rebreathing was precipitated by very low fresh gas flow rates and it was evident that ventilation and P_{CO_2} were rising rapidly.

PATIENTS AND ANESTHESIA

The study comprised 16 patients undergoing superficial surgical procedures for which spontaneous respiration was appropriate (table 1). Patients' ages ranged from 18 to 79 years (mean 50 years). No patient had symptoms or signs of cardiac or respiratory disease. All patients were premedicated with a belladonna derivative and a narcotic; nine received the tranquilizer Droperidol (5 mg.) as well. Anesthesia was induced with thiopental (200–300 mg.). The 11 patients who were intubated received succinylcholine chloride (50–100 mg.) as a single dose prior to intubation. In the nine patients in whom anesthesia was maintained by face mask, an oropharyngeal airway was inserted, and the head positioned in extension to secure a clear airway.

Most of the studies were carried out during the surgical operation, after the patient had settled into a steady respiratory pattern breathing a mixture of nitrous oxide, oxygen and halothane (0.5 to 1.5 per cent). Observations were not made during periods of changing or intense surgical stimulation.

Data from patient C. L. were treated separately from the other patients, since he exhibited an unusual degree of ventilatory depression and hypercapnia. He had received morphine, 10 mg., intravenously shortly after induction of anesthesia: this was in addition to 10 mg. given intramuscularly as part of the premedication one hour earlier.

TABLE 1. Patients Studied

Patient	Age	Sex	Weight (kg)	Operation	Tube or Mask
E. F.	60	F	67	Lt. varicose veins	Tube
E. W.	62	F	57	Rt. rad. mastectomy	Tube
C. R.	59	M	77	Lt. inguinal hernia	Tube
M. G.	79	F	73	Lt. simple mastectomy	Tube
D. O.	43	M	84	Rt. varicose veins	Tube
W. R.	52	M	79	Lt. inguinal hernia	Tube
P. C.	36	F	61	Bilat. varicose veins	Tube
I. C.	49	F	59	Rt. partial mastectomy	Tube
W. P.	51	M	69	Lt. inguinal hernia	Tube
T. P.	22	M	63	Rt. inguinal hernia	Tube
P. W.	18	F	57	Rt. varicose veins	Mask
E. P.	64	M	62	Lt. ingunal hernia	Mask
D. W.	47	F	82	Rt. varicose veins	Mask
M. D.	52	F	60	Bilat. varicose veins	Mask
E. P.	51	F	55	Bilat. varicose veins	Mask
*C. L.	46	M	74	Rt. inguinal hernia	Mask

* Patient with respiratory depression due to narcotics. Not included in calculations and graphs. See text.

TABLE 2. Measured and Derived Respiratory Data

Patient	$\dot{V}$ (fresh gas) (l./min.)	$\dot{V}_E$ (l./min.)	Frequency (breaths per min.)	V_T (ml.)	$P_{E'CO_2}$ (mm. Hg)	P_{IO_2}† − $P_{E'O_2}$ (mm. Hg)	V (fresh gas) ÷ $\dot{V}_E$
E. F.	6.6	4.1	16	256	41.4		
	5.3	4.2	16	263	41.4		
	3.6	4.5	16	281	41.4		0.80
	2.8	5.4	16	338	42.8		0.52
	2.3	6.4	15	427	45.7		
E. W.	6.7	4.9	32	153	44.9		
	5.3	4.9	30	163	44.9		
	3.9	3.9	26	150	44.9		
	3.2	3.4	23	148	49.1		0.94
	2.2	3.6	23	157	51.9		0.61
	1.5	5.8	21	276	54.8		
C. R.	6.6	5.8	17	341	35.4		
	3.1	5.8	19	305	38.2		
	2.6	5.4	18	300	41.1		0.49
	2.3	10.3	18	572	42.3		0.21
M. G.	6.6	3.6	20	180	49.1		
	4.9	4.2	19	221	49.1		
	3.8	3.8	17	224	51.9		
	3.1	3.8	16	238	51.2		0.82
	2.1	4.7	15	313	49.8		0.45
	1.7	8.4	14	600	51.2		
D. O.	6.6	4.6	26	177	38.8	26	
	4.6	5.0	25	200	38.5	30	0.92
	3.6	12.8	23	557	50.6	40	0.27
W. R.	4.6	8.8	30	293	49.2	34	
	3.6	8.8	28	314	52.1	35	0.41
	3.1	10.1	27	374	55.6	41	0.31
P. C.	6.6	6.1	28	218	47.2	27	
	4.6	6.0	28	214	49.2	29	0.77
	3.6	8.1	28	289	53.5	36	0.44
I. C.	6.6	5.9	24	246	50.3		
	3.6	6.0	25	240	51.0		0.60
	3.1	6.8	24	283	53.2		0.46
W. P.	6.9	3.9	22	177	70.0		
	4.9	4.2	22	191	70.0		
	3.9	4.9	21	233	68.8		0.80
	3.1	5.4	21	257	72.1		0.57

† P_{IO_2} is taken as "ideal inspired," equal to P_{O_2} of fresh gas.
* Patient with respiratory depression due to narcotics. Not included in calculations and graphs.

TABLE 2. Measured and Derived Respiratory Data—(*Continued*)

Patient	V (fresh gas) (l./min.)	$\dot{V}_E$ (l./min.)	Frequency (breaths per min.)	V_T (ml.)	$P_{E'CO_2}$ (mm. Hg)	P_{IO_2}† − $P_{E'O_2}$ (mm. Hg)	V (fresh gas) ÷ $\dot{V}_E$
T. P.	6.9	3.7	19	195	64.9		
	4.9	4.3	18	239	64.2		
	3.9	4.6	19	242	64.1		
	3.4	4.7	19	247	61.7		0.72
	2.7	5.8	19	305	63.4		0.47
P. W.	6.6	4.0	14	296	64.8		
	4.9	3.9	13	300	63.1		
	3.1	3.9	16	244	65.2		0.80
	2.1	6.3	16	394	68.3		0.34
E. P.	6.6	7.5	22	341	54.8		
	3.9	6.8	24	283	55.5		
	3.1	7.0	22	318	54.8		0.44
	2.1	8.4	21	400	56.9		0.25
D. W.	5.6	7.1	24	296	50.3	47	
	3.9	6.3	26	242	51.7	50	0.62
	3.0	6.9	26	265	52.4	60	0.43
	1.9	14.3	24	596	55.2	64	
M. D.	6.6	4.7	14	343	57.6		
	3.1	3.9	13	300	59.0		0.79
	2.6	4.6	13	354	61.1		0.57
	1.7	7.9	13	608	65.3		
E. Pc.	6.5	6.4	22	291	46.5		
	4.5	5.7	21	271	50.1		0.79
	3.5	7.2	19	379	51.6		0.49
C. L.*	5.6	5.1	14	364	79.7	65	
	3.9	4.4	14	314	79.7	72	
	3.0	4.5	12	375	81.1	76	
	2.2	4.6	12	383	84.0	79	
	1.8	4.4	12	367	86.9	86	0.41
	1.3	5.9	12	492	91.2	93	0.22

Results

Observations were obtained with fresh gas flow rates down to values within the range 1.3 to 3.9 l./min., and evidence of rebreathing was found in all patients. In the tabulation of respiratory variables (table 2), the dotted lines demarcate for each patient the flow rate below which one or more of the criteria of rebreathing were fulfilled. In 13 of 16 patients, increase in ventilation was the first evidence of rebreathing.

Typical results are shown in figure 4. Satisfactory records of end-expiratory P_{O_2} were obtained in only five patients. Since inspired oxygen concentrations varied from one premixed cylinder to another, values of end-expiratory P_{O_2} are not directly comparable from one patient to another. Therefore, the

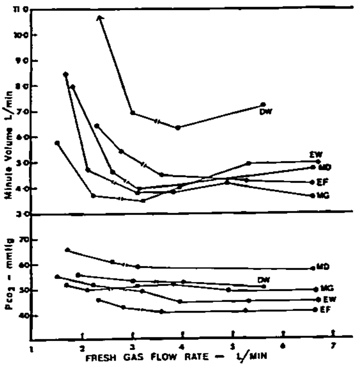

FIG. 4. Changes of ventilation and P_{CO_2} in response to reduction of fresh gas flow rate in five patients on whom observations were made at two levels of rebreathing. The broken sections of each curve indicate those sections in which rebreathing was considered to have taken place.

differences between inspired and end-expiratory P_{O_2} have been presented in table 2.

There were no statistically significant differences of any values between intubated patients and those breathing from masks. Taking the group of 15 patients as a whole (excluding patient C. L.) the mean minute volume before the onset of rebreathing was 5.28 l./min. However, rebreathing was not apparent at a mean fresh gas flow rate of 3.59 l./min. According to our criteria there was evidence of rebreathing when the fresh gas flow was reduced to a mean value of 2.83 l./min., and we may assume that rebreathing actually commenced somewhere between these limits. The mean ratio of fresh gas flow to minute volume was 0.714 before and 0.426 after rebreathing was apparent. The ratios showed considerable scatter, although this was not entirely random and fresh gas requirement could be related to respiratory frequency (fig. 5).

The onset of rebreathing caused a large increase in minute volume and a small rise in P_{CO_2}, both changes for both groups of patients being significant ($P < 0.05$) (table 3). These changes accord with P_{CO_2}/ventilation response curves, with a mean slope of 0.66 l./min./mm. Hg for intubated patients and 0.70 l./min./mm. Hg for patients breathing from a mask (fig. 6). In spite of the very low fresh gas flow rates obtained, in no patient (other than C. L.) did the P_{CO_2} exceed 72.1 mm. Hg, and increases attributable to rebreathing only exceeded 5 mm. Hg in one patient (D. O.). Changes in P_{O_2} tended to be larger (table 2) but falls of end-expiratory P_{O_2} attributable to rebreathing did not exceed 10 mm. Hg, equivalent to a reduction in inspired oxygen concentration of 1.5 per cent.

Patient C. L. differed from the other patients in the failure of ventilatory response to the elevation of P_{CO_2} before the onset of rebreathing. This was probably due to generous narcotic dosage. No appreciable increase in ventilation was seen until the fresh gas flow rate was reduced to 1.3 l./min., but the steady rise of P_{CO_2} following previous decrements of fresh gas flow suggests that rebreathing was already present at a higher gas flow rate. This cannot be defined on the basis of the criteria of rebreathing which we have adopted.

Discussion

Predicted Performance of the Circuit

The crucial feature in explaining the efficiency of the Magill circuit is the fact that expiration may be divided into two portions.[5] The first part of the gas to be exhaled from the patient comes from his anatomical deadspace, and has the same composition as fresh gas. The remainder of the expirate consists of alve-

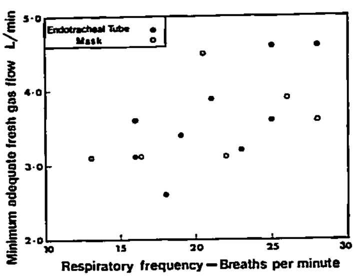

FIG. 5. Minimal adequate fresh gas flow rate plotted against respiratory frequency. The correlation is significant (r = +0.59; t = 2.61; 0.025 > P > 0.020).

TABLE 3. Conditions Immediately before and after the Onset of Rebreathing

	Endotracheal Tube			Mask and Pharyngeal Airway		
	Mean	S.D.	S.E.M.	Mean	S.D.	S.E.M.
At minimal fresh gas flow *without* rebreathing						
Minute volume (l./min.)	5.25	1.50	0.475	5.34	1.41	0.631
Tidal volume (ml.)	241	49.0	15.5	275	33.7	15.1
End-expiratory P_{CO_2} (mm. Hg)	50.4	9.32	2.95	56.2	6.09	2.72
Fresh gas flow (l./min.)	3.62	0.627	0.198	3.54	0.639	0.286
Fresh gas flow/min. vol.	0.727	0.175	0.0554	0.688	0.158	0.0705
At maximal fresh gas flow *with* evidence of rebreathing						
Minute volume (l./min.)	7.30	2.94	0.930	6.68	1.39	0.622
Tidal volume (ml.)	345	129	40.8	358	55.1	24.7
End-expiratory P_{CO_2} (mm. Hg)	53.5	8.90	2.81	58.1	6.88	3.08
Fresh gas flow (l./min.)	2.86	0.540	0.171	2.66	0.602	0.269
Fresh gas flow/min. vol.	0.431	0.130	0.0410	0.416	0.115	0.0056

olar gas, which has been in contact with the gas-exchange tissues of the lung. It is only this latter portion of an expiration which must raise the alveolar P_{CO_2} if rebreathing occurs. Since it is unlikely that there is plug flow, there is probably a transitional zone of mixed gas between the two parts.

Figure 1 shows the sequence of gas flow relationships during a single respiratory cycle. In the context of this simplified model, fresh gas flow rate should be sufficient to ensure that alveolar gas is purged from the circuit during the latter part of expiration, which begins when the bag has filled with fresh gas and the pressure in the circuit has suddenly risen enough to open the relief valve. If the fresh gas flow rate is higher than necessary, fresh gas from the patient's deadspace and also from the anesthetic machine will be lost through the relief valve. If fresh gas flow is too low, alveolar gas will remain in the circuit at the end of expiration, and be rebreathed. If fresh gas flow is just right, all alveolar gas, but no deadspace gas or fresh gas, will be eliminated from the circuit. The Magill circuit is thus able to attain maximal economy of fresh gas flow (without using soda lime) without rebreathing, but only during spontaneous respiration, when the relief valve opens during the latter part of expiration.[13, 14]

To take full advantage of the circuit's efficiency, it is necessary to know what fresh gas flow is needed in a particular case to ensure the optimal behavior shown in figure 1. Woolmer and Lind[7] studied this problem by means of a pneumatic model system. They concluded that "for the average adult patient with normal metabolic rate and corresponding ventilation, a flow of fresh gas of 7 liters per minute into a Magill's attachment will limit the percentage of CO_2 rebreathed to a satisfactory level."

Mapleson's classical mathematical analysis of five semiclosed anesthetic systems included the Magill circuit as Type A.[15] Mapleson made a series of simplifying assumptions, and went on to show that of these the only one likely to introduce serious error was the assumption of "plug flow" or absence of longitudinal mixing of gases. If this assumption is accepted, Mapleson showed that the volume of expired air remaining in the anesthetic circuit at the beginning of the next inspiration is equal to

$$V_T - \frac{\dot{V}\text{ (fresh gas)}}{f}$$

The volume of expired gas not containing CO_2 is taken to be equal to the patient's anatomical deadspace, and this is the volume which may safely be rebreathed. Thus, under ideal conditions of efficiency of the circuit,

$$V_T - \frac{\dot{V}\text{ (fresh gas)}}{f} = V_D\text{ (anat)}$$

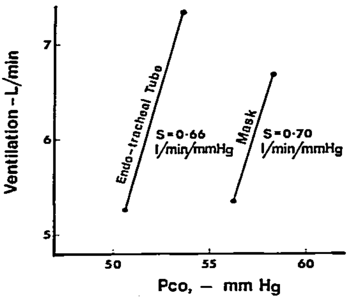

FIG. 6. Relationship of mean changes of P_{CO_2} and ventilation occurring with minimal rebreathing.

Solving for the fresh gas flow,

$$\dot{V}\text{ (fresh gas)} = (V_T - V_D\text{ (anat)})f = \dot{V}_A{}^*$$

that is, fresh gas flow must be equal to alveolar ventilation or, as Mapleson concluded, "The volume flow rate of anaesthetic mixture must be not less than the patient's effective minute volume." Because of the likelihood that longitudinal mixing of gases will lead to less-than-ideal behavior, Mapleson thought that the actual performance might be less satisfactory and, for the sake of safety, recommended employing a fresh gas flow rate equal to the patient's minute volume of respiration ($\dot{V}_E$).

Comparison of Results with Theory

In deriving the relationships discussed above, Mapleson used the calssical formula for alveolar ventilation, $\dot{V}_A = [V_T - V_D\ (\text{anat})]\ f$. This formula is likely to be inapplicable to the anesthetized patient, because of the increased alveolar deadspace, presumably due to alterations in the distribution of ventilation to perfusion.[16] Expired gas coming from underperfused or unperfused alveoli will have a lower CO_2 concentration than the remainder of alveolar gas. Thus, alveolar deadspace gas can be reinhaled with less effect on end-tidal P_{CO_2} than gas from normally-perfused alveoli. The result is that it is not the anatomical, but rather the physiologic, deadspace which should affect the performance of the Magill circuit. The central factor is still the effective alveolar ventilation, but it is now determined from the formula, $\dot{V}_A = [V_T - V_D\ (\text{physiol})]f$.

There are few studies of physiologic deadspace in anesthetized patients breathing spontaneously under halothane anesthesia. In a series studied by Nunn[17] the mean value of the ratio V_D (physiol)/V_T was 0.32 for intubated patients, the same figure being obtained in a later study by Marshall.[18] The mean effective alveolar ventilation would thus be 0.68 $\dot{V}_E$. The addition of a face mask in place of an endotracheal tube results in an increase in both anatomical and apparatus deadspace, so the effective alveolar ventilation would be a still-smaller fraction of minute volume. These considerations accord well with our observation that rebreathing commenced when the mean value of the ratio $\dot{V}$ (fresh gas)/$\dot{V}_E$ was less than 0.714 but more than 0.426.

A further prediction arrived at by both Mapleson[19] and Nunn and Newman[6] was that, under conditions of rebreathing, P_{CO_2} would be controlled by fresh gas flow rather than by alveolar ventilation. That is, the equation

$$P_{A_{CO_2}} = (P_B - 47)\,\frac{\dot{V}_{CO_2}}{\dot{V}_A}$$

would give way to

$$P_{A_{CO_2}} = (P_B - 47)\,\frac{\dot{V}_{CO_2}}{\dot{V}\text{ (fresh gas)}}$$

and the patient would be unable to influence his P_{CO_2} by hypertentilation.

This hypothesis is difficult to test, because CO_2 output is likely to change with the onset of rebreathing, and thus three variables rather than two would need to be measured. The present study, however, gives some support to the prediction. In figure 4 values of $P_{E'_{CO_2}}$ have been plotted against fresh gas flow for all the patients in whom readings were made at more than one level of rebreathing. The similarity in the slopes of the lines for the various

* In this context, $\dot{V}_A$ is defined on the assumption that alveolar deadspace is zero. The following symbols are used: F = fractional concentration; P = partial pressure or tension; V = gas volume; $\dot{V}$ = volume flow rate of gas, or output of specific gas; f = respiratory frequency or rate. A = alveolar; D(anat) = anatomical deadspace; D(physiol) = physiologic deadspace; E = expired; E′ = end-expired or end-tidal; I = mixed inspired; T = tidal; B = barometric.

cases suggests similar effects of changing fresh gas flow in all cases.

Adequacy of the Criterion of Rebreathing

The strict test of rebreathing applied to the results was chosen to give the fairest comparison between theoretical predictions and experimental findings. In practical terms, it was observed during the study that those patients in whom "first rebreathing" involved only small changes in minute volume and $P_{E'CO_2}$ appeared in clinically good condition, and the rebreathing could be determined only by measurement. In a preliminary communication[20] concentrating on the practical performance of the Magill circuit, the fresh gas flow at which a notable increase in minute volume occurred was described as an "end point" and it was noted that this flow rate lay well below minute volume in all cases. As seen in figure 4 and table 2, the increases in P_{CO_2} caused by rebreathing were relatively small, although some patients exhibited moderate degrees of hypercapnia typical of halothane anesthesia with unassisted spontaneous respiration.

End–tidal Oxygen

The measurement of end-tidal oxygen proved technically difficult, largely because the combination of end-tidal sampler and paramagnetic analyzer had a 90 per cent response time of the order of two minutes, particularly when the respiratory rate was low. In the five cases in whom satisfactory measurements were completed, the difference between inspired and end-tidal oxygen concentrations rose progressively, even before rebreathing began. The two patients breathing from masks (C. L. and D. W.) had notably higher values for inspired end-tidal oxygen difference than the three intubated patients, though in no case was end-tidal oxygen low enough to be of clinical concern. Further work is needed to account for these findings. Norman, Adams and Sykes[21] measured oxygen concentrations with a mass spectrometer in conscious subjects breathing via a Magill circuit. No fall in end-tidal oxygen was noted until fresh gas flow reached a level at which there was other evidence of rebreathing.

Clinical Implications

It is clear that the Magill circuit will indeed prevent rebreathing with a fresh gas flow considerably less than the patient's minute volume. The mean minute volume of respiration was 5.28 l./min. in the present series and, in another series of intubated patients breathing a similar gas mixture, the mean was 4.95 l./min.[17] In no patient was rebreathing evident at a fresh gas flow rate of more than 3.6 l./min. and, in a number of patients, the fresh gas flow rate was reduced below 3 l./min. before rebreathing was evident. These flow rates contrast with the widespread use of a total fresh gas flow rate of 7 l./min.

Even when our sensitive criteria of rebreathing were fulfilled, there was usually no obvious sign of rebreathing. When the fresh gas flow rate was further reduced, the increase in minute volume was frequently apparent to the unaided senses of the observer, but the increases in P_{CO_2} were almost never of clinical significance and were within the range of natural scatter of P_{CO_2} values during halothane anesthesia with unassisted spontaneous respiration. For example, the respiratory depression in patient C. L. produced P_{CO_2} values (in the absence of rebreathing) which were in excess of any levels attributable to rebreathing in the other patients.

It appears that the customary fresh gas flow rates used with the Magill circuit are in excess of requirements and that the actual minimal adequate fresh gas flow rate is of the same order as the flow rate used by many anesthetists for circle-absorbtion circuits. The Magill system has, furthermore, the advantages of being cheap, simple and easy to clean. Unfortunately, it is unsatisfactory during artificial ventilation,[13, 22] and its use is confined to situations in which it is anticipated that adequate spontaneous respiration will be maintained throughout the operation.

The upper airway and apparatus deadspace is larger in patients breathing from a mask than in those who are intubated. It might be expected, therefore, that the former would have higher values for ventilation and P_{CO_2} before the onset of rebreathing. This has not been found to the extent that might be anticipated, but the observations were made on two relatively small groups of patients who were not intentionally matched. A further study, not completed, has been designed to measure the magnitude of this effect with paired observations in the same patients.

The authors are grateful to the anesthetic staff of the Leeds General Infirmary for their co-operation during these studies. Particular thanks are due to Professor J. C. Goligher and Mr. C. G. Clark and their assistants, of the Department of Surgery, University of Leeds, for permission to study patients under their care.

References

1. Magill, I.: Endotracheal anaesthesia, Proc. Roy. Soc. Med. 22: 83, 1928.
2. Magill, I.: Discussion *in* Kain, M. L., and Nunn, J. F.: Fresh gas flow and rebreathing in the Magill circuit with spontaneous respiration, Proc. Roy. Soc. Med. 60: 749, 1967.
3. Wynne, R. L.: Mechanism of partial rebreathing in anaesthesia, Brit. Med. J. 1: 155, 1941.
4. Molyneux, L., and Pask, E. A.: The flow of gases in a semi-closed anaesthetic system, Brit. J. Anaesth. 23: 81, 1951.
5. Domaingue, F. G.: Rebreathing in a semi-closed system (correspondence), Brit. J. Anaesth. 23: 249, 1951.
6. Nunn, J. F., and Newman, H. C.: Inspired gas, rebreathing and apparatus deadspace, Brit. J. Anaesth. 36: 5, 1964.
7. Woolmer, R., and Lind, B.: Rebreathing with a semi-closed system, Brit. J. Anaesth. 26: 316, 1954.
8. Bookallil, M., and Smith, W. D. A.: A proportional respiratory sampling apparatus, Proceedings of the Anaesthetic Research Group, Brit. J. Anaesth. 36: 527, 1964.
9. Nunn, J. F.: Ventilation and end-tidal carbon dioxide tension, Anaesthesia, 13: 124, 1958.
10. Nunn, J. F., and Pincock, A. C.: A time-phased end-tidal sampler suitable for use during anaesthesia, Brit. J. Anaesth. 29: 28, 1957.
11. Webb, S. J. S.: A continuously recording respiratory flowmeter, Proceedings of the Anaesthetic Research Group, Brit. J. Anaesth. 37: 292, 1965.
12. Nunn, J. F., and Ezi-Ashi, T. I.: The accuracy of the respirometer and the ventigrator, Brit. J. Anaesth. 34: 422, 1962.
13. Sykes, M. K.: Rebreathing during controlled respiration with the Magill attachment, Brit. J. Anaesth. 31: 247, 1959.
14. Brown, E. S., Seniff, A. M., and Elam, J. O.: Carbon dioxide elimination in semiclosed systems, ANESTHESIOLOGY 25: 31, 1964.
15. Mapleson, W. W.: The elimination of rebreathing in various semi-closed anaesthetic systems, Brit. J. Anaesth. 26: 323, 1954.
16. Nunn, J. F., and Hill, D. W.: Respiratory deadspace and arterial to end-tidal CO_2 tension difference in anaesthetized man, J. Appl. Physiol. 15: 383, 1960.
17. Nunn, J. F.: Factors influencing the arterial oxygen tension during halothane anaesthesia with spontaneous respiration, Brit. J. Anaesth. 36: 327, 1964.
18. Marshall, B. E.: Physiological shunting and dead space during spontaneous respiration with halothane–oxygen anaesthesia and the influence of intubation on the physiological dead space, Brit. J. Anaesth. 38: 912, 1966.
19. Mapleson, W. W.: Theoretical considerations of the effects of rebreathing in two semi-closed anaesthetic systems, Brit. Med. Bull. 14: 64, 1958.
20. Kain, M. L., and Nunn, J. F.: Fresh gas flow and rebreathing in the Magill circuit with spontaneous respiration, Proc. Roy. Soc. Med. 60: 749, 1967.
21. Norman, J., Adams, A. P., and Sykes, M. K.: Rebreathing with the Magill attachment, Anaesthesia 23: 75, 1968.
22. Waters, D. J., and Mapleson, W. W.: Rebreathing during controlled respiration with various semiclosed anaesthetic systems, Brit. J. Anaesth. 33: 374, 1961.