CARBON DIOXIDE OUTPUT IN ANAESTHESIA*

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FOR THE PAST FOUR YEARS we have used a modified Mapleson D type breathing circuit[1] (Figure 1) routinely to the virtual exclusion of the circle breathing system as well as other breathing systems recommended for paediatric anaesthesia. Our recommendation of a fresh gas inflow of 70 ml/kg body weight[2] was found to be satisfactory in adult patients; we also recommended not to reduce the fresh gas inflow below 3.5 l/min, partly because of higher rates of CO_2 output in children, and partly because commonly used vaporizers do not provide the indicated concentrations at lower flows.

It could be shown that the patient's arterial CO_2 level could be manipulated with this breathing system independent of pulmonary ventilation as long as the

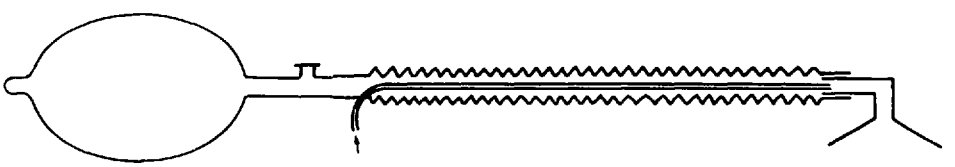

FIGURE 1. Modified Mapleson D circuit[1] ↑ fresh gas inflow.

minute volume on the ventilator was well in excess of the volume of fresh gas entering the system.[3–5] A curve was constructed showing that the arterial P_{CO_2} will change predictably with an increase or decrease in fresh gas flow (Figure 2). This curve was based on patients whose CO_2 output was assumed to be within a normal range; if the patient's CO_2 production was increased, as it would be in the case of elevated body temperature, a higher fresh gas inflow would be required for the elimination of CO_2 to maintain a normal arterial P_{CO_2}.

In order to elucidate the mode of CO_2 elimination in the circuit, CO_2 was measured at various intervals along the breathing tube, using a mass spectrometer (Elmer-Perkins) in patients breathing spontaneously and on controlled ventilation. In the tracheal tube, the CO_2 concentration fluctuated between the alveolar CO_2 concentration at the end of expiration and a low value at the end of inspiration, depending on fresh gas inflow and the amount of rebreathing (Figure 3). However, proximal to the point of fresh gas inflow, these fluctuations between inspiratory and expiratory CO_2 concentration were markedly diminished and a nearly constant CO_2 level was observed at the end of the breathing tube near the reservoir bag throughout the respiratory phases.

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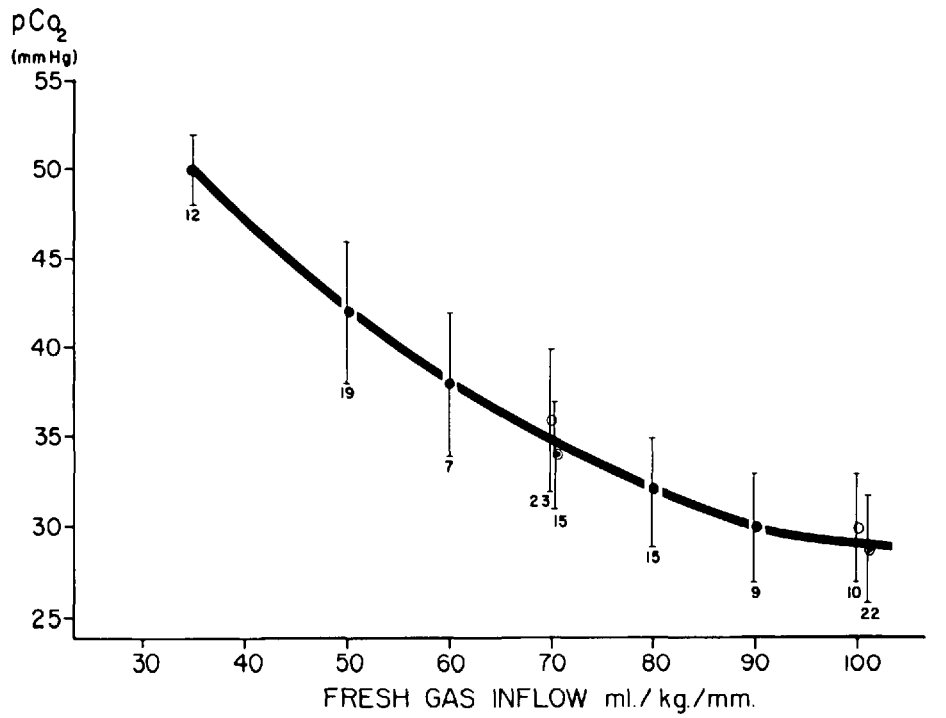

FIGURE 2. Relationship between P_{aCO_2} and fresh gas inflow during controlled ventilation.[3] ⌶ = standard deviation.

In anaesthesia with nitrous oxide and oxygen supplemented with potent anaesthetic vapours, a significant volume of gas is taken up only during the first 15 minutes of anaesthesia.[6] Thereafter the uptake of N_2O is only minimal and it can be assumed that the volume of fresh gas inflow is nearly equal to the volume of gas outflow from the expiratory valve throughout the maintenance period of anaesthesia, providing that the same fresh gas composition is maintained and the fresh gas inflow is constant and sufficient to maintain a near normal arterial CO_2 tension. Since the CO_2 concentration in the gas emerging from the expiratory valve is nearly constant throughout the respiratory cycle, and the fresh gas inflow is set on the gas machine, the patient's CO_2 output at any given moment can be estimated by determining the CO_2 concentration in volume per cent with a CO_2 analyzer and multiplying this value by the volume of fresh gas inflow per minute. We have used instruments by Beckman and by Hartmann and Braun (Germany) to obtain the data to be reported here, but instruments or methods with much slower response time would be adequate.

If the gas flow is kept constant throughout the course of anaesthesia, and the patient is on controlled ventilation with a constant rate and volume, then changes in the CO_2 output measured by this technique should be a reflection of the patient's CO_2 production and the technique would allow a continuous monitoring of the patient's metabolic activity. In spontaneously breathing patients, changes in the patient's respiratory volume would induce transient fluctuation; however, obser-

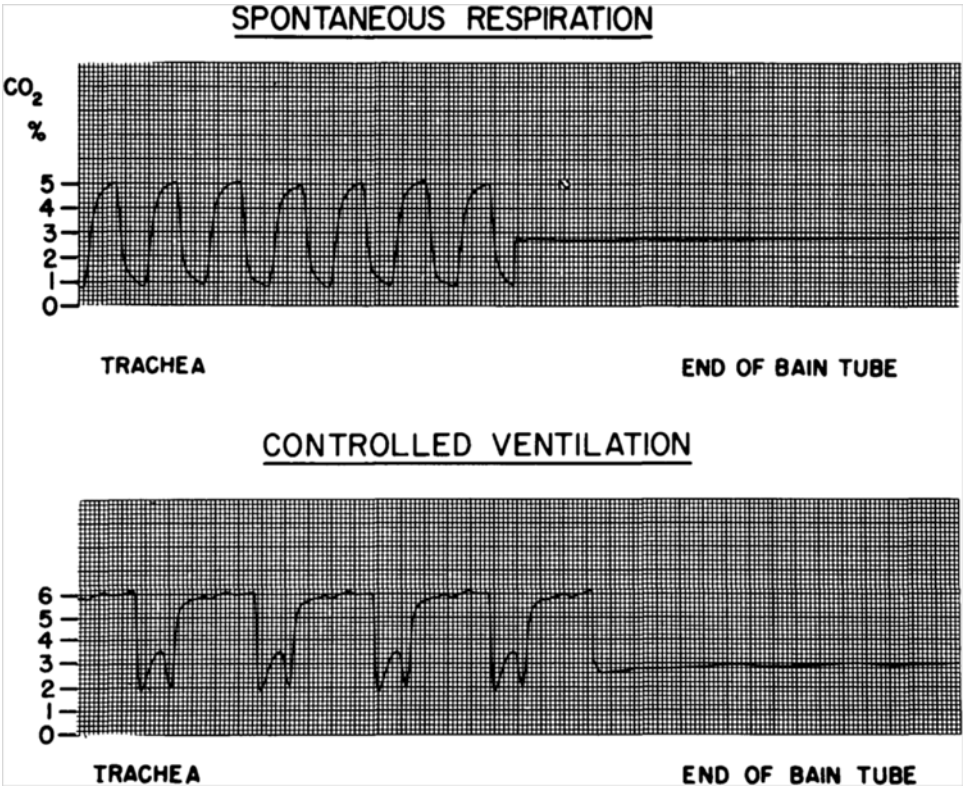

FIGURE 3. CO_2 levels (vol %) during spontaneous and controlled ventilation measured inside the trachea and at the end of the breathing tube (Bain tube) near the reservoir bag or the bellows of the ventilator.

vation over prolonged periods would still make it possible to employ this measurement as a useful indicator. Modern flowmeters on anaesthetic gas machines provide a nearly constant flow and the CO_2 analyzers employed can be read accurately to 0.1 volume per cent. Thus changes in the CO_2 output of the magnitude of 5 ml/min should be readily detectable.

METHOD OF STUDY AND RESULTS

(a) *Spontaneous Breathing*

In order to test this hypothesis, we compared two anaesthetic techniques in a uniform group of out-patients undergoing anaesthesia for the removal of impacted molars. All were healthy patients between 19 and 30 years of age and their weight was normal. No pre-medication was given and all were induced with a sleeping dose of thiopentone, intubated nasotracheally after a single dose of succinylcholine and allowed to breathe spontaneously a mixture of N_2O and O_2 at a ratio of 5:2 at a flow rate of about 70 ml/kg. The flow rate was accurately set and not changed during the procedure. One group (24 patients – 11 male, 13 female) was given alphaprodine (Nisentil) in increments of 6 mg as required and the dosage was

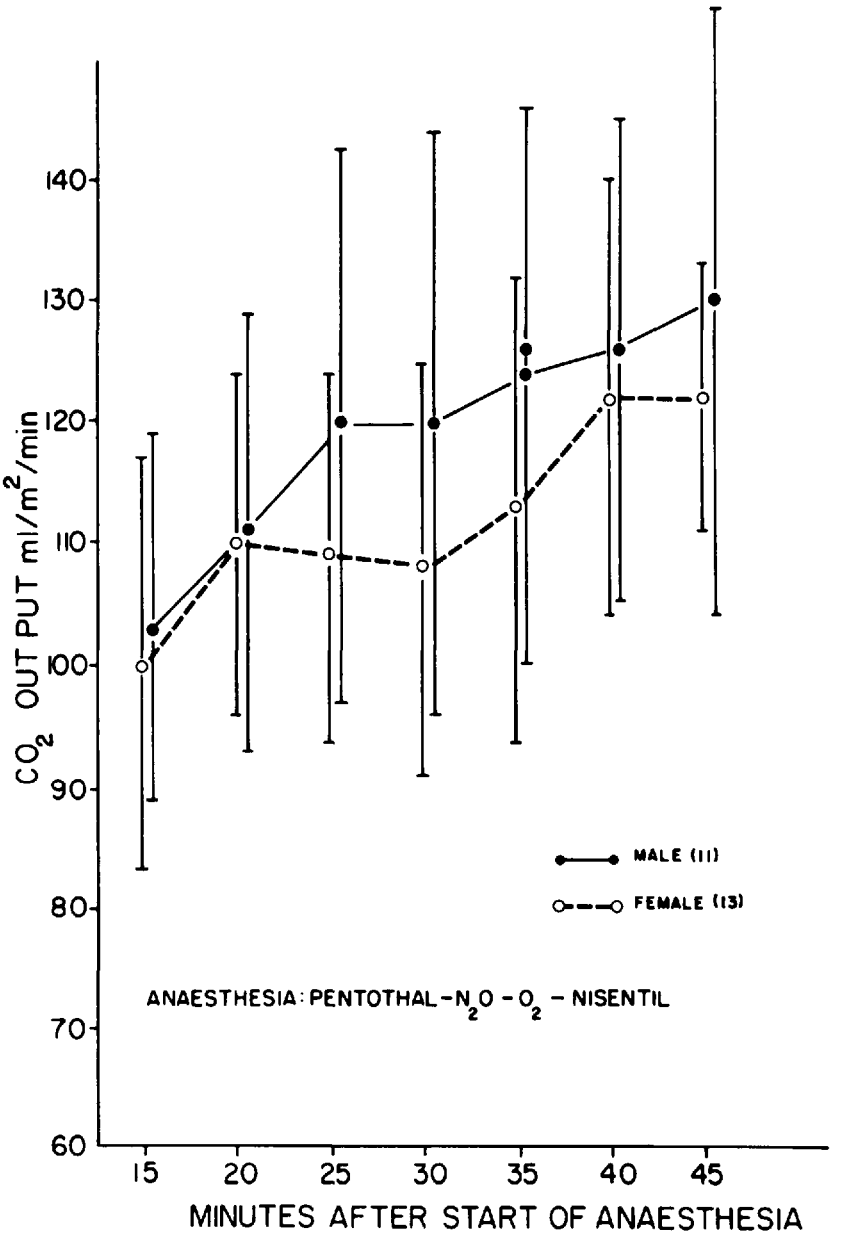

FIGURE 4. Dental anaesthesia: CO_2 output in 24 patients breathing spontaneously through nasotracheal tube. Maintenance with N_2O–O_2 (70 ml/kg) and alphaprodine.
I = standard deviation.

gauged to maintain a respiratory rate of about 12/minute. The other group (25 patients – 8 male, 17 female) received 1 per cent halothane from a Fluotec Mark III vaporizer throughout the course of the anaesthetic. Carbon dioxide was measured with a Uras CO_2 analyzer (Hartmann and Braun), sampling near the expiratory valve. The CO_2 output was expressed in ml/m^2/min, the surface area being

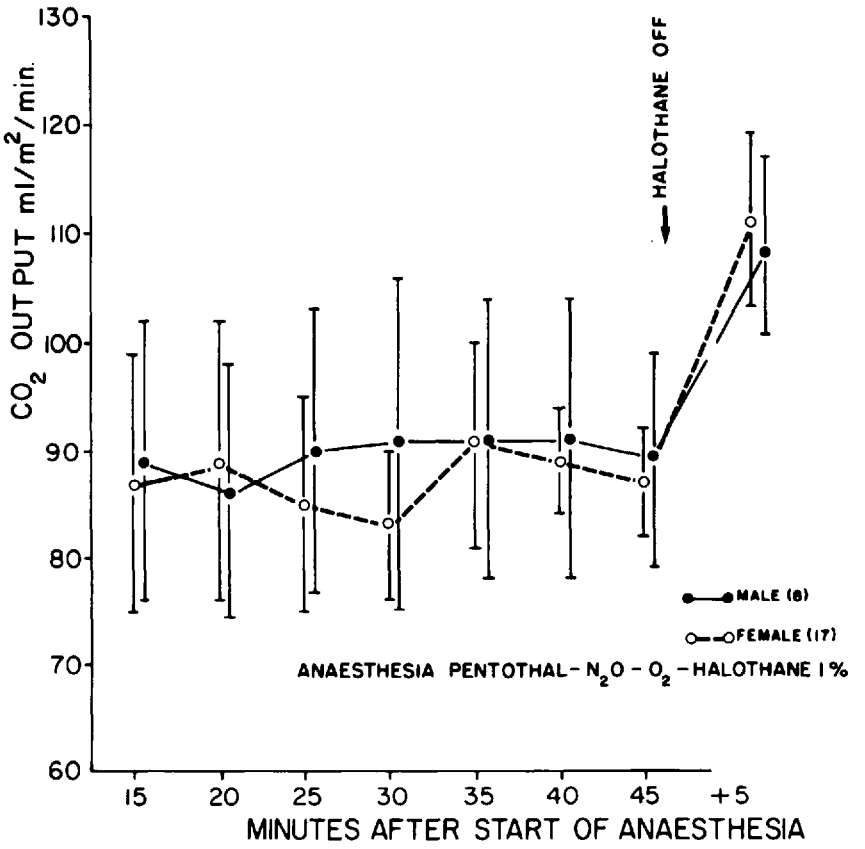

FIGURE 5. Dental anaesthesia: CO_2 output in 25 patients breathing spontaneously through nasotracheal tube. Maintenance with N_2O–O_2 (70 ml/kg inflow) and halothane 1 per cent.
I = standard deviation.

determined from weight and height tables. In addition, the Pa_{CO_2} was determined about 40 minutes after the induction of anaesthesia, prior to the end of surgery.

The results show a marked difference between the two groups, (Figure 4): in light anaesthesia with N_2O and alphaprodine, the CO_2 output increased from an initial low level of about 100 ml/m²/min at 15 minutes after induction to a mean high of about 130 ml/m²/min. The incremental doses of the narcotic tended to produce a transient plateau in most patients. There were considerable differences between individual patients, as indicated by the large standard deviations. In spite of the light level of anaesthesia and a low total dose of alphaprodine (mean dosage 24 mg in females and 30 mg in males) there was respiratory depression indicated by a mean Pa_{CO_2} of 44 ± 4 torr in this group.

The halothane cases (Figure 5) showed a fairly constant CO_2 output throughout the procedure at a lower level of about 90 ml/m²/min and the individual differences were less. When halothane was turned off without any other changes and without disturbing the patient, a significant rise in CO_2 output was noted within five minutes in all cases. The Pa_{CO_2} in this group was 37 ± 2 torr.

TABLE I

CO_2 OUTPUT—CONTROLLED VENTILATION

Main agent	No. of pat.	Age (mean)	Range	Surface area (m^2) (mean)	Body temp. (°C) (mean ± S.D.)	CO_2 output ($ml/m^2/min$) (mean ± S.D.)
			Values one hour after induction			
Halothane	11	55	(40–74)	1.92	35.7±0.7	87±11
Ethrane	14	53	(21–74)	1.87	36.2±0.7	98±19
Nisentil	17	53	(22–75)	1.72	35.8±0.5	93±13

(b) *Controlled Ventilation*

In patients on controlled ventilation, the CO_2 output was determined by the same technique. Forty-two patients of normal body build and in category 1 or 2 of the ASA Classification, undergoing major surgical procedures were induced with thiopentone and ventilated with an Air Shields ventilator-ventimeter with a tidal volume of 10 ml/kg body weight. The fresh gas inflow of N_2O and O_2, at a ratio of 5:2, was about 70 ml/kg/min. Anaesthesia was maintained with halothane 0.7 per cent to 1 per cent, ethrane 1 per cent to 1.5 per cent or intermittent doses of alphaprodine (Table I). Tubarine or pancuronium were used as muscle relaxants. The rectal or oesophageal temperature was monitored. Carbon dioxide was sampled from the breathing tube near the ventilator and measured with a Beckman CO_2 analyzer; only the values obtained one hour after induction are reported here. As in the spontaneously breathing patients there were considerable individual variations, but the mean values were of the same order. The CO_2 output values for halothane (mean 87 ± 11 $ml/m^2/min$) were lower than for ethrane (98 ± 19), although this difference was not statistically significant. We had expected higher values in the narcotic group (mean 92 ± 13) in accordance with the observations in spontaneously breathing patients. However, the dosage of alphaprodine was higher in this group and all patients were pre-medicated with narcotics and paralyzed with muscle relaxants.

Since the CO_2 output was not significantly different between the three groups, is seemed reasonable to calculate a mean CO_2 output of 93 ± 14 $ml/m^2/min$ for the 42 patients.

This was in contrast to six obese patients (three males, three females) with a mean weight of 110 kg and a mean surface area of 2.15 m^2. Their mean CO_2 output of 114 ± 17 $ml/m^2/kg$ (Table II) was significantly higher than that of patients in a normal weight range. One might expect a lower value considering the lower metabolic rate of fat tissue and further studies are required to verify this observation. However, when the CO_2 output was related to body weight there was no difference.

After observing unexpectedly high values in a patient who underwent operation while on hyperalimentation, we have measured the CO_2 output in eight patients on hyperalimentation under anaesthesia and all showed a marked elevation (Table II). Their mean CO_2 output was 160 ± 25 $ml/m^2/min$, significantly higher than the value of 93 ± 14 $ml/m^2/min$ in our group of normal patients, representing

TABLE II

CO_2 OUTPUT—CONTROLLED VENTILATION

	Values one hour after induction						
	No.	Age (mean)	Range	Surface (m^2) (mean)	Body temp. (°C) (mean)	CO_2 output ($ml/m^2/min$) (mean ± S.D.)	CO_2 output (ml/kg/min) (mean ± S.D.)
Normal (Table I)	42	54	(21–75)	1.83	35.9	93±14	2.25±0.26
Obesity	6	50	(23–62)	2.15	36.4	114±17	2.23±0.20
Hyperalimentation	8	52	(28–81)	1.64	36.3	160±25	4.38±0.63

possibly a 72 per cent increase in metabolism. Related to body weight, the CO_2 output was almost double (92 per cent when compared to normal patients). The higher metabolic rate in these patients has been attributed to the free amino-acids contained in the hyperalimentation solution.[7]

Discussion

Carbon dioxide elimination in a partial rebreathing system depends on CO_2 production and fresh gas inflow. The application of standards for the prediction of a required fresh gas inflow is only valid in patients in a normal metabolic state. For the safe use of partial rebreathing circuits an estimate of the patient's CO_2 production is helpful to avoid CO_2 accumulation and respiratory acidosis in unrecognized states of increased metabolic activity.

The values for CO_2 output obtained by this technique are of the same order as those reported by other investigators.[8,9] The technique measures only the actual CO_2 output and this would reflect CO_2 production if the patient is in a steady state. Changes in ventilation and in fresh gas inflow will alter the steady state. Our anaesthetic technique with flow rates near the alveolar ventilation represents an almost isocapnic ventilation and marked fluctuations in the patient's CO_2 levels are avoided. We think the technique of estimating CO_2 output is valid 15 minutes after the change of the patient's atmosphere from room air to a N_2O–O_2 mixture at induction of anaesthesia; at that time the alveolar N_2O concentration should be above 90 per cent of the inspired concentration and the period of a significant uptake of the N_2O has passed.[6]

Our findings indicate that the degree of anaesthetic depression represents a factor in the relationship between fresh gas inflow and the Pa_{CO_2}. A significantly higher CO_2 output was seen in spontaneously breathing patients under light nitrous oxide–narcotic anaesthesia when compared to patients under halothane anaesthesia. In our small series of patients on hyperalimentation a markedly elevated CO_2 output was found. This must be taken into account when such patients are anaesthetized using a partial rebreathing system and fresh gas inflows of 120 ml/kg/min are recommended or actual monitoring of the CO_2 output as described. For obese patients the CO_2 output per square meter is higher than in patients within a normal weight range; however, their CO_2 output was of the same order as in normal patients when related to body mass. It would seem advisable to calculate the flow rates required for obese patients on the basis of their body weight.

The breathing circuit[1] now in general use in our hospitals makes it possible to determine the patient's CO_2 output instantaneously and continuously by the technique described. It allows the continuous monitoring of the CO_2 output with sufficient accuracy to be clinically useful and to detect otherwise unrecognizable states of increased metabolic activity.

Summary

In a Mapleson D circuit the carbon dioxide content of gases, sampled at the breathing bag or near the bellows of the ventilator, is virtually constant throughout

the phases of respiration. Assuming that after induction of anaesthesia the fresh gas inflow, if kept constant, is essentially equal in volume to the gas vented at the expiratory valve, CO_2 output can be calculated by multiplying the fresh gas inflow by the CO_2 content of the vented gas measured with a suitable CO_2 analyzer.

Anaesthesia with nitrous oxide-oxygen, supplemented with low doses of alphaprodine or halothane was compared in two groups of young patients who underwent dental surgery and who were breathing spontaneously. While the CO_2 output in the group supplemented with alphaprodine increased from about 100 to 130 ml/m²/min, the halothane group showed a constant CO_2 output of about 90 ml/m²/min followed by a significant rise within 5 minutes after halothane was discontinued.

In 42 patients on controlled ventilation, no significant difference was found in the CO_2 output estimated one hour after induction of anaesthesia in nitrous oxide-oxygen anaesthesia supplemented by halothane, ethrane or alphaprodine.

The values obtained were 87 ± 11 ml/m²/min for halothane (11 patients), 98 ± 19 ml/m²/min for ethrane (14) and 93 ± 13 ml/m²/min for the narcotic supplemented anaesthesia (17). The mean CO_2 output for all 42 patients was 93 ± 14 ml/m²/min.

Six markedly obese patients under the same anaesthetic technique had a CO_2 output of 114 ± 17 ml/m²/min; however, their CO_2 output was similar to normal patients when calculated on the basis of body weight. A marked increase in CO_2 output to a mean of 160 ± 25 ml/m²/min was found in eight patients undergoing operation while on hyperalimentation.

The technique described appears suitable to monitor CO_2 output under anaesthesia. In order to avoid hypercarbia when using a partial rebreathing system, the fresh gas inflow must be increased above recommended values in cases with increased metabolic activity (e.g. patients receiving hyperalimentation). In obese patients the fresh gas inflow should be calculated on the basis of body weight.

Résumé

Avec le circuit de Mapleson D, le contenu en gaz carbonique mesuré dans le ballon ou à la sortie des soufflets du respirateur est constant en pratique pendant toutes les phases de la ventilation. En admettant, qu'après l'induction de l'anesthésie le débit de gaz frais, s'il est maintenu constant, est nécessairement égal au volume de gaz s'échappant par la valve expiratoire, la production de CO_2 peut être calculée en multipliant le débit de gaz frais par le contenu en CO_2 de gaz s'échappant par la valve à l'aide d'un analyseur de CO_2.

En ventilation spontanée, l'anesthésie par le mélange oxygène-protoxyde d'azote complété par de faibles doses d'alphaprodine ou d'halothane fut comparée chez deux groupes de patients jeunes au cours d'une chirurgie dentaire. Tandis que la production de CO_2 du groupe endormi avec un complément d'alphaprodine passait de 100 à 130 ml/m²/min, celle du groupe sous fluothane restait constante aux alentours de 90 ml/m²/min et montrait ensuite une augmentation significative durant less cinq minutes suivant l'arrêt du fluothane.

Les valeurs obtenues furent 87 ± 11 ml/m²/min pour l'halothane (11 patients),

98 ± 19 ml/m²/min pour l'éthrane (14 patients) et 93 ± 13 ml/m²/min pour le narcotique (17). La production moyenne de CO_2 pour les 42 patients fut 93 ± 14 ml/m²/min.

Six patients nettement obèses eurent une production de CO_2 de 114 ± 13 ml/m²/min avec la même technique anesthésique. Cependant, leur production de CO_2 fut comparable à celle des patients normaux quand elle fut rapportée au poids. Une augmentation marquée de la production de CO_2 à une moyenne de 160 ± 25 ml/m²/min fut trouvée chez huit patients qui étaient opérés pendant une hyperalimentation.

La technique décrite apparaît utilisable pour le monitoring de la production du CO_2 pendant l'anesthésie. Afin d'éviter une hypercapnie quand on utilise un système avec rebreathing partiel, le débit de gaz frais doit être augmenté au-dessus des valeurs recommandées pour les patients ayant une élévation du métabolisme (patients recevant une hyperalimentation). Chez les patients obèses le débit des gaz frais doit être calculé en se référant au poids.

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