THE ADEQUACY OF THE RADFORD NOMOGRAM DURING ANAESTHESIA

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THE SUBJECT OF VENTILATION during anaesthesia has undoubtedly been the main topic of discussion and research by anaesthetists for many years, and as a result a great deal of valuable knowledge has been produced which by now should enable us to ventilate our patients correctly, maintaining, normal or near normal arterial gas tensions. Unfortunately like so many things in medicine, our behaviour is like a pendulum swinging from one extreme to the other, only striking a happy balance after many years. We seem to be approaching this balance slowly.

In the past the anaesthetist's only concern with ventilation was that the patient should be breathing and that he should be pink. In the period of completely closed system absorption anaesthesia this was easy as the near 100 per cent oxygen concentrations used made it easy to have a pink patient, and this seemed to signifiy adequate ventilation. When it was discovered in the forties that many of our arrhythmias and asystoles were the result of an excess of carbon dioxide, and also when we learned of the effects of hypercapnia on the activity of the vagus and on catecholamine production, the trend to hyperventilation then commenced. With this trend came high gas flows, non-rebreathing valves and jumbo absorbers, all designed to remove every possible particle of CO_2 from the blood and, if possible, from the tissues. With the impetus of the gas-relaxant technique and the realization that respiratory alkalosis was essential if the patient was to be unconscious and not awake and paralysed, the hyperventilation became more intense, reaching the ultimate in the present Liverpool teaching of rapid shallow respiration in order that physiological dead space be at a minimum and alveolar ventilation at a maximum. This concept of ventilation arose from the discovery that hyperventilation increases the physiological dead space in direct proportion to the tidal volume. The pendulum was now far to the left and, as one would expect, the battle was joined between those claiming nothing but good from hyperventilation[1] and those who were not so sure, and who proceeded to investigate the possible reasons for the apparent additive effect of respiratory alkalosis to the anaesthetic state, and others who are trying to discover what physiological disturbances are being produced.[2,3,4] It would appear that the weight of evidence at present is in favour of those who believe that a marked respiratory alkalosis is not entirely beneficial and may well have harmful effects and that a little more anaesthetic or analgesic supplement might be a preferable method of guaranteeing unconsciousness.

But all of us have said "let us err on the side of "hyperventilation"" as surely from our sad experiences of the past we know that the patient is better off with hyperventilation and respiratory alkalosis than he or she is with hypoventilation and its consequences of hypercarbia and possibly hypoxia. Few anaesthetists

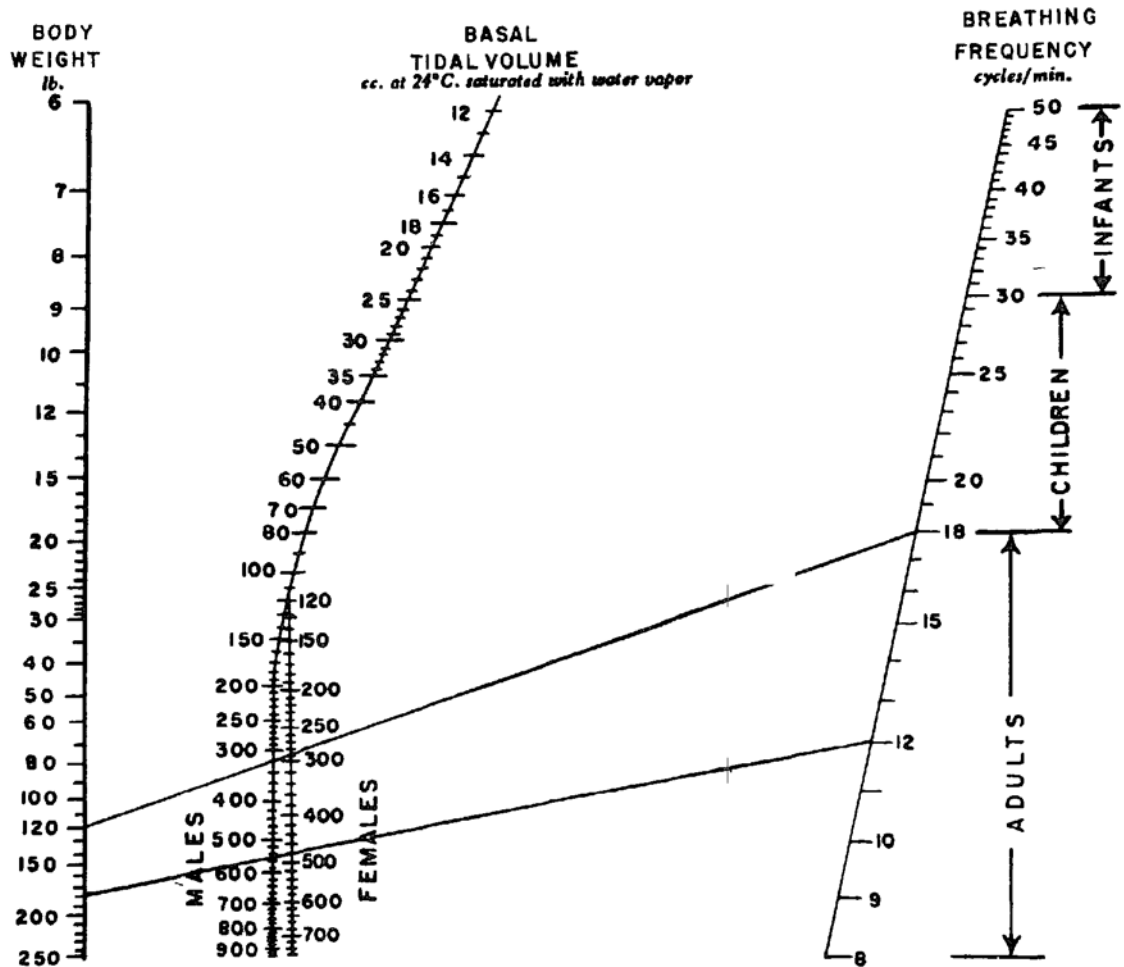*The Department of Anaesthesia, University of Alberta Hospital, Edmonton, Alberta.

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FIGURE 1. Corrections to be applied to basal tidal volume as required:
  Daily activity: Add 10%
  Fever: Add 5% for each degree F. above 99° (rectal)
  Altitude: Add 5% for each 2000 feet above sea level
  Metabolic acidosis during anaesthesia: Add 20%
  Tracheotomy and endotracheal intubations: Subtract a volume equal to one-half the body weight
  Added dead space with anaesthesia apparatus. Add volume of apparatus and mask dead space

would argue with this. As a result most anaesthetists have followed the teaching that every patient under general anaesthesia requires assistance to ventilation especially with our more popular agents and techniques; for this we have relied on the "educated hand" which has so often been said to be very uneducated and grossly inaccurate.[5] Fortunately this hand usually errs on the side of hyperventilation but not always, especially with an open pleura. What we needed therefore was some simple method of determining what constituted adequate ventilation and a simple means of measuring it.

In 1954 and 1955 Radford[6,7] published a nomogram which was designed to tell us the tidal volume required to maintain the arterial pCO_2 at 40 mm. Hg. This nomogram was based on the fact that basal carbon dioxide production and respiratory dead space are related to body size and may be estimated from body

weight. It was determined that respiratory dead space in millilitres is roughly equal to the body weight in pounds. From these factors a nomogram was constructed which would give the tidal volume necessary to meet basal conditions and maintain an alveolar pCO_2 of 40 mm. Hg. These workers also included many factors which might require correction such as daily activity, fever, altitude, metabolic acidosis during anaesthesia, tracheotomy and endotracheal intubation, and added dead space from anaesthetic apparatus. They found that the nomogram predicted with sufficient accuracy for clinical use in poliomyelitis patients, in other patients, and infants. Radford stated that there were some conditions under which the nomogram could not be applied, the most important of these being the presence of muscle activity.[6] It obviously cannot be applied without corrections in hyper- and hypo-metabolic states, or in conditions where the physiological or anatomical dead space is increased, as in certain pulmonary conditions. Finally it cannot be applied when there is severe metabolic alkalosis or acidosis, especially in the presence of kidney disease. Except for muscle activity all of the above conditions are relatively rare and therefore should not interfere seriously with the routine usefulness of the nomogram. The ventilation standards are designed to provide for the removal of carbon dioxide with the implication that if carbon dioxide is removed oxygen supply is also adequate. The routine use of high inspired oxygen mixtures makes this assumption almost invariably true.[7]

This nomogram has come to be accepted by anaesthetists and clinical investigators as a reliable means of determining the total minute ventilation which a patient requires to maintain normal pCO_2 levels in the arterial blood. The recommended use of this nomogram by the anaesthetist is frequently referred to the anaesthesia journals. Such a useful nomogram is of little use to us unless we have means of measuring either the tidal volume or the arterial pCO_2. Few clinical anaesthetists have the means for doing blood gas studies or the money to acquire the equipment and staff necessary, but all can easily acquire one of the many simple ventilation meters which are as easy to use as is counting the pulse. One then knows whether the patient is receiving the required tidal volume indicated by the nomogram. If one uses the non-rebreathing technique, then it is only necessary to set the flow meters at the predetermined minute volume and one then knows if the patient is receiving the indicated minute volume of gas.

It was therefore surprising that it took so long for those with the facilities to study the use of this nomogram in anaesthesia to determine whether it was accurate or not when applied to the anaesthetized patient under clinical conditions. Nunn[8] reported such a study in 1960 and stated that he was of the opinion that under anaesthesia the nomogram overestimated the carbon dioxide output and underestimated the dead space, but as these errors are opposite in effect they may be expected to cancel one another at normal tidal volumes. He found that the nomogram was an accurate guide with artificial ventilation with the chest closed but during spontaneous respiration or with the chest open there was poor correlation between pCO_2 and ventilation. Why should the nomogram fail during spontaneous respiration? The most likely explanation is that

the muscle activity required by spontaneous respiration is very great and the resulting energy used increases carbon dioxide output invalidating the nomogram as stated by Radford,[7] who lists muscle activity as the most important condition under which the nomogram cannot be used.

When we come to look at the problem of the failure of the nomogram with the open chest it becomes a much more complex situation. Campbell[9] found that when patients were ventilated artificially there was a highly significant increase in dead space but little increase in the alveolar-arterial oxygen gradient which implied overventilation of parts of the lung which had a small pulmonary blood flow. They found that even in normal cases with intact abdominal and chest walls various abnormal patterns of ventilation and blood flow distribution may occur during artificial ventilation. These changes were too small to affect respiratory exchange or arterialization of the blood provided total ventilation was adequate. The use of an endotracheal tube compensated almost exactly for this increase in dead space. Therefore, even with an intact chest or abdomen during artificial respiration the distribution of ventilation and blood flow are less than ideal. With an open abdomen and especially an open chest a much less ideal situation may prevail which is not compensated for by an endotracheal tube; this undoubtedly explains in part why the Radford nomogram is not applicable during thoracotomy.

Theye and Fowler,[10] working with dogs, found that the ventilatory requirements are not influenced specifically by position or by an open pneumothorax but rather they are determined to a large degree by the metabolic production of CO_2 and the degree to which ventilation is distributed to perfused alveoli. Thornton[11] found during artificial respiration that changes in the physiological dead space may be brought about by distribution of gases, changes in the anatomic dead space, or altered perfusion. The findings of an increase in the physiological dead space during artificial positive-pressure ventilation suggests the need for a change in the pattern or volume of ventilation. This is the basis for the Liverpool technique of rapid shallow ventilation, to increase the minute volume and reduce the physiological dead space. The increase in dead space which Thornton[11] found appeared to be for the most part due to alterations in the distribution of blood perfusion. Nunn and Hill[12] reported studies on the anatomical and physiological dead space and none of their findings appeared to depend on whether the respiration was spontaneous or artificial, that is the type of respiration did not affect anatomical dead space. The physiological dead space, however, did show a clear relationship to tidal volume and this ratio was reasonably constant and independent of the manner of ventilation. The alveolar dead space appeared to be related to the tidal volume with no relation to the manner of ventilation. These workers found that the alveolar ventilation may be expressed as a fraction of the minute volume, that is 0.7 of the minute volume as they found the physiological dead space to be 0.3 of the tidal volume. The changes in the physiological dead space with tidal volume tend to protect the patient from the extremes of under- and over-ventilation and this can be of importance during hypoventilation associated with deep anaesthesia, which unfortunately is still practised by some. This increase in

physiological dead space is apparently due largely to an increase in the alveolar component and this is probably due to: (a) certain alveoli may be deprived of blood supply; (b) certain inactive alveoli may become ventilated but not perfused; (c) an appreciable number of alveoli are relatively overventilated. Therefore there are many factors to be considered. We cannot separate circulation and respiration; what we do to one effects the other. The effect of drugs on cardiac output, the effect of positive pressure on venous return, the effect on ventilation-perfusion ratios are all factors to be considered. It is not surprising then that the Radford nomogram has been found to be inadequate during anaesthesia with the open chest when there are such marked disturbances in ventilation patterns, in ventilation perfusion ratios, in venous return to the heart, and the mechanical surgical disturbances to ventilation and circulation.

It therefore becomes important that some means be at hand to determine the adequacy of ventilation during open-chest surgery. Nomograms are obviously inaccurate; end-tidal samples and gas analysis are still subject to a large error because, during artificial ventilation, some alveoli are ventilated but not perfused and the unperfused alveoli contain little carbon dioxide and so will dilute the gas from the perfused alveoli. Thus the expired alveolar carbon dioxide tension will be less than the carbon dioxide tension in perfused alveoli or the arterial blood. This is of importance, as the measurement of carbon dioxide tension in alveolar air is probably easier and more frequently used than are arterial blood measurements.

For those equipped with the newer pieces of apparatus[13] it is easy to obtain an arterial sample and in 5 minutes to know exactly how efficient ventilation is. For those without such equipment the use of a Wright ventilation meter in conjunction with the Radford nomogram would appear to be a simple method for routine clinical use, at least when dealing with a closed chest and controlled or artificial respiration. As mentioned previously this method is frequently reported as the procedure of choice for determining the adequacy of ventilation during clinical anaesthesia.

The Wright respirometer is a simple easy-to-use ventilation meter which indicates both tidal and minute volume. Nunn[14] has recently studied the accuracy of this meter and found it to be adequate for clinical monitoring and that only during gross underventilation and moderate overventilation will the error exceed 10 per cent. The error exaggerates the departure from normality, a desirable type of error. The accuracy is greatest at a ventilation which is likely to keep the arterial pCO_2 close to normal. It was decided to use the Radford nomogram as originally described by Radford,[6,7] for since this time Radford has recommended a change in the correction factor for tracheotomy and endotracheal intubation. It is now recommended that a volume equal to one-quarter of the body weight rather than one-half of the body weight be used.[15] We do not believe that this change in the nomogram is widely known, and therefore decided against its adoption for this study. This change in the correction factor for tracheotomy and endotracheal intubation could in many cases provide another half to one litre per minute of ventilation, which would undoubtedly have improved our results with the nomogram.

Therefore, before recommending the use of this combination of Radford nomogram and Wright ventilation meter to our staff we decided to carry out a study of the adequacy of our ventilation under everyday conditions, using spot checks of arterial pCO_2 on unselected patients being ventilated with the "educated hand" by many different anaesthetists, including residents. Carbon dioxide arterial tensions were determined using the Astrup apparatus.[13] Table I shows the results of a few of these unselected cases and, as expected, most patients were overventilated but none were grossly underventilated.

TABLE I

ARTERIAL pCO_2—VENTILATION CONTROLLED, AND MINUTE VOLUME DETERMINED BY "EDUCATED HAND"

	Arterial pCO_2	Operation
1.	46 5	Gastrectomy
2	29 5	Discectomy (prone)
3.	34 5	Hysterectomy
4.	43 0	Nailing femur
5.	31 0	Cholecystectomy
6.	23 5	Cholecystectomy
7.	32 0	Herniorrhaphy
8.	32 5	Radical mastectomy
9.	29 0	Gastrectomy
10.	41 0	Excision olecranon bursa
11.	32 0	Hysterectomy
12.	28 0	Colon resection
13	31 0	Rhinoplasty
14.	29 4	Mandibulectomy
15.	36 0	Arthrotomy—Knee
16.	24 5	Thoracotomy—Lobectomy
17.	31 0	Mitral valvotomy
18.	28 0	Mitral valvotomy
19.	27 0	Mitral valvotomy
20	16 8	Radical mastectomy

Spot checks were made of unselected patients being ventilated by a mechanical ventilator and the adequacy assessed by the anaesthetist's clinical judgment. These are shown in Table II. We had expected much more hyperventilation than was found in these few cases. The few cases with high pCO_2 levels all had respiratory function disturbances. All of the others were hyperventilated.

The use of the Radford nomogram and Wright ventilation meter was similarly checked in selected cases, that is those patients without lesions, which Radford excludes.[6,7] Ventilation was provided with a Bennett or Bird ventilator, and was monitored constantly with the Wright ventilation meter on the expiratory side of the absorber circuit. All patients were intubated. Roswell-Park type absorbers were used in each instance and all pCO_2 determinations were made after 30-minute periods. The first column in Table III shows a few of these results.

The ventilation was increased, when possible, by 25 per cent and/or 50 per cent over the requirements. of the nomogram and the results are shown in columns 2 and 3 in Table III. If longer periods had been allowed the arterial pCO_2 levels would undoubtedly have been lower in some and higher in others.

Next to be studied was a group of thoracotomies, and great difficulty was experienced in finding adequate numbers of suitable cases where disturbances

TABLE II

ARTERIAL pCO_2—VENTILATION WITH AUTOMATIC VENTILATOR,
MINUTE VOLUME DETERMINED BY
"ANAESTHETISTS' ASSESSMENT"

	Arterial pCO_2	Operation
1.	61.4	Pneumonectomy*
2.	43	Colectomy
3.	36 8	Mediastinotomy
4.	37.5	Aortic resection
5.	35 0	Aortic resection
6.	36 8	Exploratory laparotomy
7.	32.6	Mitral valve replacement*
8.	23 2	Exploratory laparotomy
9.	25 8	Hysterectomy
10.	29.0	Abdomino-perineal resection
11.	36 5	Cholecystectomy
12	33 6	Cholecystectomy
13.	27 8	Colectomy
14.	28 8	Mediastinotomy
15.	26 0	Gastrectomy
16	33 0	Oesophageal replacement
17.	61 5	Mitral valve replacement*
18.	54 0	Aortic valve replacement*
19.	31 0	Gastrectomy
20	27 0	Anterior resection—colon

*Known pulmonary function disturbance.

TABLE III

ARTERIAL pCO_2—VENTILATION CONTROLLED, MINUTE VOLUME DETERMINED
BY RADFORD NOMOGRAM

	Radford	Radford +25%	Radford +50%	Operation
1.	48	52.0		Exploratory laparotomy
2.	44	36 2		Gastro-enterostomy
3	40	37 0		Abdomino-perineal resection
4.	42	36 5		Hitatus hernia repair
5.	46 0	38 5		Cholecystectomy
6.	45 0	37 0		Cholecystectomy
7.	46 8	45 0		Inguinla hernia repair
8.	38 0	31.0		Colectomy
9.	47 0	36 0	32 7	Colostomy
10.	44 5	39 8	34 5	Gastectomy
11.	46 0	40 0	38 5	Abdominal hysterectomy
12.	45 0	41 0	39 0	Cholecystectomy
13.	48 0	46 5	40 0	Abdomino-perineal resection
14.	40.5	38 0	35 5	Oophorectomy
15.	45 0	41 0	37 5	Vaginal hysterectomy
16.	44 5	41 2	32 5	Open reduction radius
17.	45 0	40 0	39 0	Arthroplasty—hip
18.	55.0	46 0	44 5	Cholecystectomy
19.	49.5	43 2	40 0	S.P. nailing
20.	50.0	44 4	37 0	Inguinal hernia repair
21.	52 0	42 0	37 5	Renal artery repair
22.	43.5	41.0	38 5	Inguinal hernia repair
23.	44.0	40 0	36 0	Skin graft—foot
24.	44.5	36.4	29 5	Strabismus correction
25.	52.0	44 2	37 5	Open reduction humerus
26.	47 0	43 0	39 5	Oophorectomy

TABLE IV

ARTERIAL pCO_2—VENTILATION CONTROLLED AND MINUTE VOLUME
DETERMINED BY RADFORD NOMOGRAM

	Radford, pleura closed	Radford, pleura open	Radford +100%, pleura open	Operation
1.	48 5	65 0		Mediastinal cyst
2.	42 4	53 4	40 4	Patent ductus
3.	51.0	54 0	42 0	Lobectomy
4.	46 0	50 0	38 4	Lobectomy
5.	48 0	56 0	44 5	Mitral valvotomy
6.	55 0	61 0	40 0	A.S.D.
7.	48 0	52 0	28 4	Oesophagectomy
8.	46 4	51 0	41 4	A.S.D.
9.	42 0	44 8	35 0	Pulmonary stenosis valvotomy
10.	49 5		40 0	Lung biopsy
11.	50 0		37 0	Exploratory thoracotomy
12	43 5		38 5	Pneumonectomy
13.	44 0		40 0	Mitral valvotomy
14.	52 0		37 5	A.S.D.
15.	44 0	51 0	38 0	Oesophagectomy
16.	43 4	48 0	36 4	Decortication
17.	42 0	44 8	35 5	Transthoracic vagotomy
18.	43 5	49 0	37 0	Coarctation
19.	39 5	43 5	30 0	Excision lung cyst
20.	42 5	46 0	24 5	Heller procedure
21.	48 0	58 0	33 0	Coarctation

in ventilation or ventilation perfusion ratios or diffusion defects were not present. It will be seen that many of the thoracotomies were cardiac cases and undoubtedly some diffusion and/or perfusion defects existed, but we tried to obtain patients who showed no clinical signs or in whom pulmonary function tests had eliminated the possibility of diffusion and other defects. Table IV shows the results in a few cases with open and closed pleura when ventilated according to the Radford nomogram. The results agree with Nunn's.[8] The ventilation was then doubled when the pleura was opened. It is noted in these cases that there is a satisfactory fall of arterial pCO_2 to normal levels.

Many factors other than those previously discussed come into play during clinical anaesthesia which will influence the results and are in part responsible for the wide variation in arterial CO_2 tensions. Undoubtedly the patient's compliance and the compliance of the anaesthetic equipment are important factors. Mushin[15] states that 10 per cent should be added to the Radford nomogram for the compliance of the rebreathing tubes. This will vary greatly depending on the construction and age of the tubes. The patient's rate of carbon dioxide production and level of anaesthesia, the resulting depression of metabolism and reflex activity, and the degree of muscle relaxation will all influence the results. Although not carefully studied it was certainly our impression that the greater the relaxation and the less the positive pressure required to attain the indicated tidal volume the closer the carbon dioxide tensions approached the ideal of 40 mm. of mercury. This is in keeping with Radford's views on the effects of muscle activity on the accuracy of the nomogram. Also, if the patient's compliance is decreased, more of the tidal volume will appear in the distensible rebreathing tubes.

CONCLUSION

It would appear from this small study that the "educated hand" and the anaesthetist's clinical judgment are really not too uneducated and that they more consistently produce safe arterial pCO_2 levels than does the use of the Radford nomogram as originally described, and the Wright ventilation meter when used under standard everyday conditions of practice. The nomogram can be used to provide adequate ventilation if the sources of error are appreciated and allowances are made for these by increasing the values by at least 25 per cent in extrathoracic cases and by 100 per cent in those cases with open pneumothorax. It is unlikely that significant hypoventilation or hyperventilation will be produced in the average case if such additions to the nomogram are made. If endotracheal anaesthesia is used and Radford's modified correction factor for this is applied, then it is likely, in extrathoracic cases at least, that the Radford nomogram will provide adequate ventilation under clinical conditions using everyday standard anaesthetic equipment. Because of the frequency of the sudden changes which can occur in the surgical procedure or in the anaesthetic state, both of which can rapidly change compliance, it is essential that the tidal volume be monitored frequently if adequate exchange is to be maintained throughout the operative procedure when using the nomogram with a mechanical ventilator. If the range of normal arterial pCO_2 tensions is taken to be 35–45 mm of mercury, then the modified nomogram will usually provide these levels in most extrathoracic cases anaesthetized and ventilated under everyday conditions of anaesthetic practice.

SUMMARY

The Radford nomogram and the Wright ventilation meter have been often recommended as a useful, reliable method of providing satisfactory ventilation during anaesthesia, with certain reservations. Previous studies have been reviewed. The use of the nomogram and meter under usual clinical conditions has been carried out and the adequacy of ventilation has been determined by measurement of arterial carbon dioxide tension. It was found that the much-maligned "educated hand" was a more reliable means of maintaining arterial carbon dioxide tensions at safe levels than was the nomogram under these conditions. It is believed that in extrathoracic cases increasing the nomogram value by at least 25 per cent, and with an open pleura by 100 per cent, the nomogram will provide safe levels of arterial carbon dioxide during anaesthesia with controlled or artificial respiration.

RÉSUMÉ

On a souvent recommandé le nomogramme de Radford et l'usage du ventimètre de Wright comme méthode utile et fiable d'assurer une ventilation satisfaisante au cours de l'anesthésie, mais sous certaines réserves. Nous avons fouillé la littérature. Dans les conditions cliniques ordinaires, on a employé le nomogramme et le ventimètre et l'on a vérifié si la ventilation était adéquate en mesurant la pression du CO_2 artériel.

Les résultats révèlent que la main la plus malhabile demeure un moyen beaucoup plus fiable pour maintenir des tensions de CO_2 artériel à des taux normaux que le nomogramme dans les mêmes conditions. Nous sommes d'opinion que, dans les cas extrathoraciques, il faudrait augmenter le nomogramme d'au moins 25 pour cent et, quand la plèvre est ouverte, de 100 pour cent, si l'on veut conserver des taux de CO_2 artériel de tout repos au cours de l'anesthésie sous respiration contrôlée ou artificielle.

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