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NOTE

The views expressed in these lectures represent the views of the authors and do not necessarily reflect the views either of the Editor or of the WFSA.

Erratum

Please note that, in Volume 1985/1, one of the Consulting Editors, Dr S.G. Hershey, was described as 'Chairman, Education Committee, WFSA'. This is incorrect. The Chairman of the Education Committee, WFSA, is Dr Howard Zauder, Syracuse, New York State, USA. The Editor and Publishers apologize for any confusion that may have been caused by this error.

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CONTENTS

PULMONARY PROBLEMS IN ANAESTHESIA*

J. F. Nunn

INTRODUCTION

From the very earliest days of our speciality, there was concern that anaesthetics might have an adverse effect on the respiratory system. An immense and continuing volume of research has confirmed the early suspicions and it is no easy task to summarize the present state of these investigations.

THE RESPIRATORY MUSCLES AND THE FUNCTIONAL RESIDUAL CAPACITY

It is perhaps most convenient to start with consideration of the effect of anaesthesia on the pattern of contraction of the respiratory muscles, because it now appears that this underlies many of the other alterations in respiratory function. Jones *et al* (1979) quantified the progressive loss of contribution of rib cage movement to total respiration as the end-expiratory concentration of halothane was increased. This phenomenon has long been used as one of the signs of the stages of anaesthesia but we shall see that it also has relevance to the respiratory response to carbon dioxide.

A reverse change occurs in the paralysed anaesthetized patient who is ventilated artificially. Vellody *et al* (1978), showed an *increase* in the rib cage contribution to tidal volume under these circumstances. Thus it is clear that the pattern of distribution of inspired gas cannot be the same during anaesthesia with spontaneous breathing as it is during artificial ventilation.

Although the movement of the diaphragm is well preserved during general anaesthesia without paralysis, there is a change in the pattern of its contraction. Muller *et al* (1979) showed that in the conscious state the diaphragm retains some residual tone during expiration, but that this is lost during anaesthesia. The reason for this change is not yet established but nevertheless it has important consequences.

Let us now go back in time to 1974 and the classical study of Froese & Bryan (1974) (Fig. 1). They studied lateral chest

*Lecture delivered at the World Congress of the World Federation of Societies of Anaesthesiologists, Manila, January 1984. Reproduced from the Proceedings of the Congress, Excerpta Medica, with permission of Elsevier Science Publishers, Amsterdam, the Netherlands.

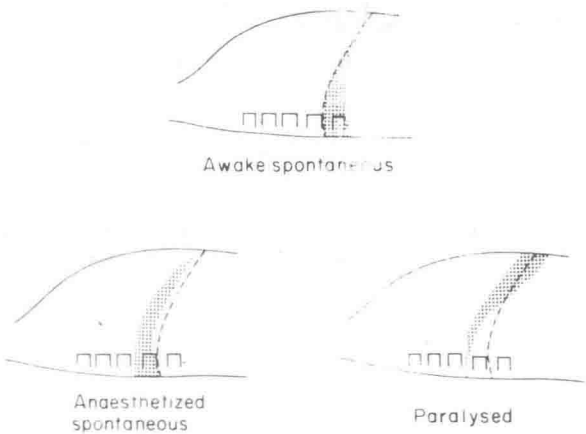

Fig. 1 Lateral chest radiographs from the study of Froese & Bryan (1974), with kind permission of the publishers J.B. Lippincott Co.

X-rays during anaesthesia and the broken line, which is the same in all three figures, represents the end-expiratory position of the diaphragm with the patient awake. The shaded area is the diaphragmatic excursion during breathing. During anaesthesia, the diaphragm rises into the chest by a few centimetres whether breathing is spontaneous or whether the patient is paralysed and ventilated artificially. It now appears that this is mainly due to the loss of end-expiratory tone in the diaphragm as demonstrated by Muller *et al* (1979) and the consequence is a decrease in functional residual capacity, which was in fact known to occur many years before the explanation became apparent. However, it has been independently concluded that redistribution of blood to the abdomen may also contribute to the elevation of the diaphragm during anaesthesia.

Table 1

	Spontaneous respiration	Artificial ventilation
Conscious	0	± 3.1%
Anaesthetized	±16.1%	±15.4%

Table 1 shows our own observations of changes in functional residual capacity during anaesthesia with or without paralysis and artificial ventilation. Changes are related to the conscious state, breathing spontaneously, and it will be seen that artificial ventilation by itself has no effect, but that anaesthesia with or without paralysis results in a reduction of functional residual capacity which is about 16% in each case. These changes accord very well indeed with the lateral chest radiographs shown in Fig. 1.

Many studies by many different workers are in agreement with the observation that functional residual capacity is

reduced during anaesthesia and we may summarize the characteristics of this change.

- It occurs very quickly after induction.
- It occurs with barbiturate anaesthesia as well as inhalational anaesthesia.
- It is uninfluenced by paralysis.
- The change is not progressive.
- It is not related to the presence of absorbable gases (such as oxygen).

We shall return to the important question of the physiological consequences of reduction of functional residual capacity, but before concluding this review of the effect of anaesthesia on the pattern of contraction of the respiratory muscles, it must be mentioned that the induction of anaesthesia without paralysis usually results in phasic contraction of the abdominal expiratory muscles which are normally silent in the supine awake patient. This observation was made by Freud *et al* (1964) and Kaul *et al* (1973). Within a minute of injecting thiopentone, phasic contraction of the external oblique commences. The view of the old-time surgeon that the anaesthetic made the abdomen tighter thus had an element of truth. Of course, this effect is abolished by neuromuscular blockade.

The question may be asked whether this activation of the expiratory muscles contributes to the reduction in functional residual capacity. In fact its contribution is negligible since the reduction in functional residual capacity is still seen when the patient is curarized. It is an interesting phenomenon and the cause of it is quite unknown.

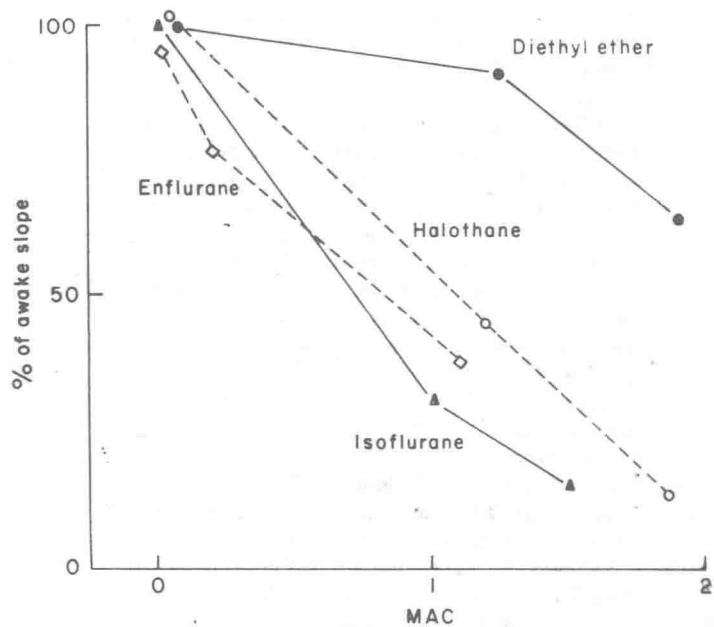

Fig. 2 Effect of different anaesthetics on the P_{CO_2}/ventilation response curve in man (redrawn from the data of Eger).

CONTROL OF BREATHING

It has long been known that anaesthesia causes a decrease in the slope of the P_{CO_2}–ventilation response curve. Figure 2 shows a comparison of the effect of different inhalational anaesthetic agents. Depression of the ventilatory response to CO_2 seems to be a common feature of all inhalational agents, although in the case of ether, an appreciable effect is not seen until the alveolar concentration reaches twice the minimal alveolar concentration required for anaesthesia. This may well be due to catecholamine release at low concentrations and noradrenalin is known to stimulate breathing.

It has generally been assumed that anaesthetics depress the CO_2 drive to breathing either at the central chemoreceptors or at the adjacent medullary neurones which subserve respiration. However, a most important study by Tusiewicz *et al* (1977) showed that conventional wisdom was wrong. They recorded separately the rib cage and abdominal response to increasing CO_2 levels and were thus able to plot the two components of the CO_2 ventilation response curve. They showed that the rib cage response to increasing P_{CO_2} far exceeds that of the abdomen and it must be concluded that hypercapneic ventilatory drive is mediated mainly through the intercostal muscles or, more precisely, those muscle groups which act to increase the cross-sectional area of the rib cage. However, we have already noted that rib cage movement is selectively depressed by anaesthesia and showed that, as the P_{CO_2} was increased during anaesthesia, there was no change in what little was left of rib cage movement while the relatively feeble abdominal response was little altered. The conclusion is that a major part of the effect of anaesthesia on the P_{CO_2}–ventilation response curve seems to be due to depressant activity of anaesthesia on rib cage expansion. That is to say the effect is largely peripheral rather than central as was believed.

Until quite recently, anaesthetists were reassured by the teaching that anaesthetics had little, if any, effect on the hypoxic drive to breathing. Unfortunately, this comfortable doctrine has now been shown to be untrue in the case of inhalational anaesthetics, and Knill & Gelb (1978) showed that the hypoxic drive was not so much depressed as totally abolished by halothane. So far from being a rugged protective reflex, it appears that the hypoxic drive to ventilation is exquisitely sensitive to anaesthesia and it is affected even at sub-anaesthetic concentrations.

The clinical implications are important. Firstly, an anaesthetized patient should not be expected to respond to hypoxia by hyperventilation, and no reliance should be placed on this as a monitor. Secondly, it can be expected that anaesthesia will arrest the breathing of patients who have lost their ventilatory sensitivity to CO_2—particularly patients with chronic bron-

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chitis in the category of the 'blue bloater'. Thirdly, there is an obvious hazard in anaesthetizing a patient in a hypoxic environment; during one of the early expeditions to Everest, there were great difficulties ensuing during attempts to anaesthetize an injured climber with chloroform.

With these powerful effects of anaesthesia on the chemical control of breathing, it would be expected that reflex control might be similarly affected. It is always difficult and often dangerous to anticipate the results of experiments and Nunn & Ezi-Ashi (1961) were surprised to discover that anaesthetized and even partially paralysed patients had a remarkable ability to compensate for added resistance to breathing. Part of the response was delayed and probably attributable to CO_2 retention, but the major part of the response was immediate and remarkably little affected by anaesthesia. For example, patients were able to breathe against a sudden imposition of an inspiratory threshold resistor of 10 cm water. Clearly, this would require the development of tension in the diaphragm far in excess of that attained during the previous unobstructed breath. It is tempting to ascribe this response to a spindle reflex. On this hypothesis, the upper motor neurone would instruct the phrenic diaphragm complex to contract with whatever force was necessary to achieve the required shortening of the diaphragmatic fibres. This would be analogous to the biceps contracting with whatever force was necessary to lift a suitcase of unknown weight.

If it is postulated that the diaphragmatic contraction is based on a spindle reflex, the existence of spindles in the diaphragm must be presupposed because, as has already been seen, the intercostal contribution to breathing is greatly curtailed during anaesthesia. It is known that the intercostal muscles are richly supplied with spindles but until recently it was thought that there were no spindles in the diaphragm. Muller *et al* (1979) have now demonstrated the existence of a small number of spindles and they have also shown the instant response of the diaphragm to increased loading.

The response of the anaesthetized patient to an expiratory load is quite remarkable. Expiration is mainly passive in spite of the fact that there is some contraction of the expiratory muscles in the anaesthetized patient. Therefore, the sudden imposition of a threshold resistor (e.g. a PEEP valve) in the expiratory line will prevent expiration if the threshold pressure is higher than the end-inspiratory recoil pressure of the lungs and chest wall. The Hering-Breuer reflex would lead one to expect inhibition of any further inspiration, but this does not occur and neither is this surprising because the Hering-Breuer reflex is weak in man.

When expiration is checked by the expiratory threshold registor, the next inspiration is augmented and the process continues with the patient inspiring progressively higher into his inspiratory reserve volume until at last the recoil pressure is

sufficient to overcome the expiratory resistance. Thus, the additional work of breathing against the expiratory resistance is transferred to the inspiratory muscles by means of a greater amount of potential energy stored in the elastic structures of lung and chest wall.

This remarkable response was first observed by Campbell *et al* (1957) and was then more fully explored by Nunn & Ezi-Ashi (1961). The physiological implications are interesting. Arrest of respiration at progressively higher lung volumes means that the diaphragmatic fibres are progressively shortened and might be expected to contract less effectively. Therefore, it would appear that the response must depend on a resetting of the diaphragmatic spindles by contraction of the intrafusal fibres, and it is difficult to see how this response can be mediated by any other method.

Whatever the physiological basis of these mechanisms, we can draw comfort from the observation that an anaesthetized patient can respond very well to increased resistance to breathing. Nunn & Ezi-Ashi were surprised to find patients breathing at 80% of control tidal volume against an inspiratory pressure of 10 cm water.

COMPLIANCE

It has long been known that the compliance of the lungs is reduced during anaesthesia when compared to the conscious state.

Figure 3 is based on the work of Westbrook *et al* (1973) and studies at subatmospheric pressure undertaken by Butler & Smith (1957). The alveolar pressure relative to atmosphere is on the X-axis and the lung volume on the Y-axis. The vertical

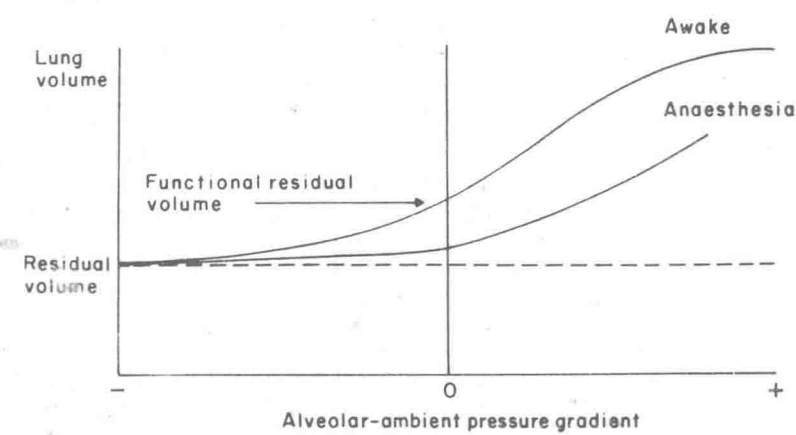

Fig. 3. Pressure/volume relationships of anaesthetized man compared with the conscious state.

After Westbrook *et al* (1973), with kind permission of the American Physiological Society.

line indicates atmospheric pressure and the horizontal line the residual volume. The awake curve intercepts the volume axis at atmospheric pressure at the functional residual capacity. The curve during anaesthesia is below and shows the functional residual capacity reduced to little more than the residual volume. Application of strong sub-atmospheric pressures removes little gas from the lungs.

The causes of these changes are far from clear. In part the lung moves down its compliance curve to a flatter part of the curve as a result of the loss of the expiratory diaphragmatic tone to which I have already referred. However, this is not the whole story. In addition, there is an increased elastic recoil of the lung/chest wall system and the cause of this is not yet clarified. However, it is known that the lungs become stiffened if a subject breathes at low lung volume.

AIRWAY RESISTANCE

Airway resistance may be increased during anaesthesia. A major factor in this change is the decrease in functional residual capacity, which causes a reduction in all components of the distensible parts of the lung, including the airways.

Figure 4, which is redrawn from the work of Lehane *et al* (1980) shows a plot of airway resistance against lung volume at constant bronchomotor tone. The relationship is hyperbolic with a very steep increase in airway resistance as the lung volume is reduced below functional residual capacity. Since this is exactly what happens following the induction of anaesthesia, there is a tendency for airway resistance to increase.

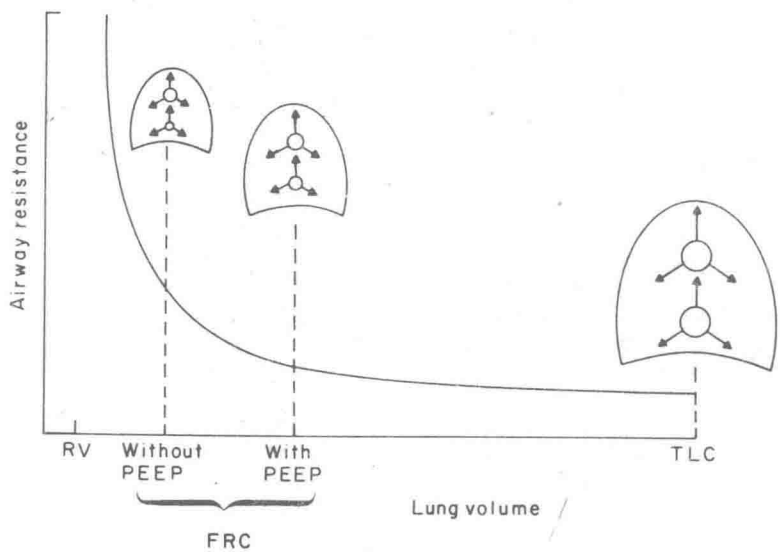

Fig. 4 Airway resistance as a function of lung volume. Redrawn from the work of Lehane *et al* (1980), with kind permission of the publishers, the British Medical Association.

However, this is largely counteracted by the bronchodilator effect which has been demonstrated for diethyl ether, halothane, enflurane and, more recently, for isoflurane as well. To a large extent these effects cancel each other out but small changes may be observed in either direction.

DISTRIBUTION OF INSPIRED GAS

There is ample evidence that the spatial distribution of inspired gas in the anaesthetized patient differs from the pattern of the awake subject. It will follow a different pattern again if the patient is paralysed and ventilated by intermittent positive pressure. It is very difficult to measure the spatial pattern of distribution of inspired gas with sufficient sensitivity to record the effects of the altered patterns of contraction of the respiratory muscles. The usual test based on nitrogen wash-out is not quite the same thing since it is dependent on the sequence of emptying the alveoli. No consistent abnormality has been observed with this test following induction of anaesthesia.

GAS EXCHANGE

This difficult problem has stimulated more research than almost any other aspect of anaesthesia but it should be remembered that the changes, though statistically significant, do not pose a serious problem in the routine surgical patient who had satisfactory lung function before surgery. Defects in the oxygenation of the arterial blood can be compensated by an increase in the concentration of oxygen in the inspired gas and, under normal circumstances, no patient should be subjected to serious hypoxia during or after an anaesthetic.

The Riley 3-compartment model

Historically, the problem of gas exchange during anaesthesia was first investigated in terms of the three compartment model. This approach is still highly relevant to clinical problems of gas exchange and it is directly applicable to the methods of analysis which are available to most of us in the clinical field. Our interest in the approach is therefore not purely historical but is of direct clinical relevance to everyday problems.

The concept makes the assumption that the lung can be divided into three compartments. First of all there are alveoli which are correctly ventilated and perfused, permitting normal gas exchange, the gas in these alveoli being known as 'ideal alveolar gas'. Next, the model postulates the existence of a

group of alveoli which are not perfused—as a result, for example, of a pulmonary embolus. Ventilation of these alveoli is wasted and contributes to the physiological dead space. The third compartment consists of alveoli which are not ventilated and so contributes shunted blood to mingle with the blood from the ideal alveoli.

Dead space

When considering dead space, it is important to distinguish between the anatomical and physiological components. The anatomical dead space is the volume of the conducting air passages, which is seldom significantly increased and its measurement is of little clinical importance. Measurement of physiological dead space by Enghoff's modification of Bohr's equation gives a value which includes apparatus, anatomical and alveolar dead space. This is the value which has physiological relevance.

The physiological dead space was first measured during anaesthesia in 1958 during collaborative studies undertaken by Campbell *et al* (1958). They found that, in the paralysed intubated ventilated patient, the dead space—tidal volume ratio was about 32%—which was the same as in the patients before anaesthesia. However, during anaesthesia the dead space was measured from the carina downwards. Thus, during anaesthesia the dead space below the carina was the same as in the conscious subject in whom trachea, pharynx and mouth were included. It can be concluded, therefore, that there was an increase in the alveolar dead space approximately equal to the volume bypassed by the tracheal tube which is about 75 ml.

Shunt

As is well known, shunts have a very marked effect on the arterial P_{O_2} but comparatively little effect on P_{CO_2}. This is only because of the different slopes and shapes of the dissociation curves for the two gases and the effect on arterial blood *content* is actually similar.

Shortly after the introduction of the polarographic determination of arterial P_{O_2}, many studies showed that the alveolar/arterial P_{O_2} gradient was increased during anaesthesia to a value compatible with a shunt of approximately 10% of the pulmonary blood flow, compared with a normal value in the conscious subject which should not exceed about 3%.

Ventilation : perfusion ratios

So far I have considered gas exchange in the lung as a three

compartment model. This is, of course, a gross over-simplification. The lung contains alveoli with a full spectrum of ventilation : perfusion ratios from zero (that is to say a totally unventilated alveolus) to infinity (that is to say a totally unperfused alveolus). Although this state of affairs had been recognized for many years, its quantification had to wait for the development of sophisticated analytical techniques.

The first major break-through was due to John West, who developed an isotopic method for the separate quantification of ventilation and perfusion in horizontal strata of the lung. In recent years a new and much more powerful technique has become available. This is the 6 inert gas wash-out technique developed by Wagner and West in San Diego. Application of this technique to anaesthesia was a formidable technological challenge. Success has, however, been achieved by three groups: Dueck *et al* (1980) in San Diego; Rehder *et al* (1979) at the Mayo Clinic; and Bindslev *et al* (1981) at the Karolinska Institute, Stockholm.

Rehder's results were obtained in young healthy volunteer subjects and both ventilation and perfusion were found to be distributed to a wider range of ventilation : perfusion ratios after induction of anaesthesia and paralysis. The mean true intrapulmonary shunt was less than 1% during anaesthesia but the alveolar/arterial P_{O_2} gradient was increased and this was attributed to the increased spread of distribution of perfusion. Anatomical dead space was reduced largely because of tracheal intubation, while alveolar dead space was increased and this was partly explained by distribution of ventilation to areas of high ventilation : perfusion ratio.

Bindslev *et al* (1981) studied patients of a considerably older age group (37–64) in four states: awake; anaesthetized and breathing spontaneously; anaesthetized, paralysed and ventilated artificially; and finally with positive end-expiratory pressure. In contrast to Rehder's group they found that the true intrapulmonary shunt was increased in these older patients after the induction of anaesthesia. However, the shunt calculated from the alveolar/arterial P_{O_2} gradient according to the Riley 3-compartment lung model would be larger still and a part of this would be accounted for by the distribution of perfusion to areas of lower ventilation : perfusion ratio. The dead space : tidal volume ratio was increased during anaesthesia in spite of the bypassing of the upper airway with the tracheal tube. Positive end-expiratory pressure reduced the shunt but also reduced the cardiac output and the net effect was virtually no change in the arterial P_{O_2}.

The study of Dueck *et al* (1980) was confined to elderly patients (mean age 60) who all had some deterioration of pulmonary function. Their patients can best be divided into three groups. In the first group there was little increase in true intrapulmonary shunt during anaesthesia but there appeared a shelf of perfusion of regions of very low ventilation : perfusion

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