AN ATTEMPT TO FIND A PRACTICAL METHOD OF CARBON DIOXIDE MEASUREMENT DURING ANAESTHESIA

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RECENTLY a great deal of attention has been focused on the problem of carbon dioxide accumulation during anaesthesia (Stead et al., 1953; Harbord, and Lucas, 1953). A number of articles have appeared describing investigations on the actual figures for "alveolar air" or arterial carbon dioxide tensions found during anaesthesia. Ringrose et al. (1950) developed a carbon dioxide indicator showing a qualitative measurement of carbon dioxide in anaesthetic atmospheres. Harrison and Jones (1955) were unable to find, however, that this method has a practical value. Engell and Ibsen (1952) used a carbovisor based on a photo-electrical principle to obtain a continuous recording of carbon dioxide estimations during thoracic operations. Eastwood and Harbord (1955) correlated the changes in tidal volume with those of "alveolar air" concentrations.

Most of the methods described are impossible to perform routinely without assistance and the aid of a laboratory to do the analysis. The aim of this article is to show that it is possible to obtain measurements of the "end of expiration air" of sufficient accuracy to be of value to the anaesthetist working alone.

METHOD

A carbon dioxide meter, made in Germany and calibrated up to 10 per cent was used in the experiments. This machine is small and, being made of plastic, weighs very little. The principle is to aspirate a sample of air into a chamber of known volume which is then driven into a set volume of 25 per cent sodium hydroxide which rises into a graduated chamber. As the carbon dioxide is absorbed the level of the hydroxide falls in the measuring chamber and the figure is read off after a minute.

The reading given is apparently accurate up to 0.3 per cent. The indicator should be checked frequently against room air which should give a figure of 0 per cent. The machine should be handled as little as possible to prevent heat transference from the hand affecting the readings. The raising and lowering of the plunger should be done slowly and gently or the readings will be affected.

SAMPLING

The samples were obtained from the upper end of a cuffed endotracheal tube. A short glass tube with a short piece of
rubber tubing attached was fitted into the rubber bung of the endotracheal mount. This rubber tubing was kept clamped when sampling was not in process. Samples were taken at the end of expiration whether respiration was spontaneous or controlled. Henderson and Haggard (1925), Rahn and Otis (1947) and Buckley et al. (1952) have shown that the carbon dioxide content in samples of air taken at the end of expiration approximates to the pCO$_2$ of arterial blood.

**ANAESTHETIC**

All patients were premedicated with papaveretum 20 mg and scopolamine 0.45 mg. Induction was by thiopentone in doses of 200–400 mg. Relaxation was obtained by suxamethonium or gallamine triethiodide. Nitrous oxide and oxygen supplemented with pethidine maintained narcosis and analgesia in 25 cases. Five cases received cyclopropane.

Nitrous oxide is soluble in caustic soda but a number of experiments using samples from the mixture of oxygen 3 litres and nitrous oxide 6 litres per minute showed that an average figure of only 0.3 per cent need be deducted from the carbon dioxide readings to allow for the absorption of nitrous oxide by the caustic. This figure has been deducted in the data given below. Cyclopropane would appear to be insoluble in the caustic.

All measurements were taken by the same person but the anaesthetics were given by a number of anaesthetists. All operations were major, the majority either upper abdominal or thoracic. The blood pressure and pulse rates were recorded at five-minute intervals. The average duration of anaesthesia was two hours, the longest being three hours, the shortest one hour.

**RESULTS**

418 readings were taken during 30 anaesthetics. A normal figure for "alveolar air" has been described as 4.7–6.4 per cent by Eastwood and Harbord (1955). No attempt was made to compress the chest at the end of expiration as it was felt that this could hardly be described as routine procedure during anaesthesia. A practical difficulty was to be certain that the sample analysed was, in fact, an end of expiration sample and not a mixture of incoming gases and "alveolar air". On a number of occasions in the early part of the experiment very low figures were obtained suggesting that this mixing had occurred or that the tidal volume was too low. These low figures were discarded. Some mixing was unavoidable and no doubt the figures recorded are slightly below the true figures. The results are tabulated in table I.

<table>
<thead>
<tr>
<th>Type of respiration</th>
<th>Highest single recording</th>
<th>Lowest single recording</th>
<th>Average for the series</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First 15 cases</td>
<td>8.9</td>
<td>4.2</td>
<td>7.0</td>
</tr>
<tr>
<td>Second 15 cases</td>
<td>7.2</td>
<td>3.7</td>
<td>5.9</td>
</tr>
<tr>
<td>Assisted</td>
<td>8.7</td>
<td>4.3</td>
<td>6.7</td>
</tr>
<tr>
<td>Controlled</td>
<td>7.2</td>
<td>2.4</td>
<td>4.4</td>
</tr>
</tbody>
</table>

(1) *Spontaneous respiration.*

In 50 per cent of the cases the readings were within the normal limits. The average reading for the remaining 50 per cent was above normal, i.e. 7 per cent indicating some degree of underventilation. It must be stated that most of these
high figures were obtained in the early part of the experiment as, later on, respiration was controlled when the carbon dioxide tension began to rise. No apparent ill effects were observed in these cases though the figures were not alarmingly high and the longest period during which hypercapnoea was permitted was only 25 minutes except in one case.

(2) Assisted respiration.
This technique was only used in the early part as it soon became obvious that the readings were just as high as when respiration was spontaneous.

(3) Controlled respiration.
Respiration was defined as being controlled when no respiratory movements were observed for 30 seconds on releasing the bag. It was difficult to be certain at what point control of respiration had been affected by carbon dioxide elimination as the factors affecting control are variable. On a number of occasions control had been gained when additional surgical stimuli produced respiratory movement. In one case the readings were 3.7 per cent when extra pressure on a retractor in the abdomen caused wrinkling of the forehead and respiration became spontaneous.

At the beginning of the series control of respiration was maintained at considerably varying levels, i.e. the lowest average was 3.3 per cent and the highest average 6.1 per cent, showing that the artificial ventilation had been inconsistent and that in some cases the patient had been over-ventilated, while in others either central depression had been excessive or the degree of muscular paralysis was high. In the latter part of the series control was maintained at levels of carbon dioxide in the region of "low normal", i.e. 4.4 per cent.

In one case control was maintained for the first hour at a level of 3.8 per cent. Spontaneous respiration was then permitted for 25 minutes, with the absorber off, on a mixture of nitrous oxide 4 litres and oxygen 2 litres per minute. The carbon dioxide readings (nine in all) averaged 7.1 per cent, the highest being 8 per cent and the lowest 6.2 per cent. During the next hour respiration was again controlled but it was found difficult to get the readings down to 4.6 per cent during this second period of control.

Extremely high figures were obtained in a case of lobectomy. These readings are not included in table I. On three occasions figures of well over 10 per cent were found. The actual figures could not be determined as the meter is not calibrated to above 10 per cent. On these isolated occasions the respirations were controlled. Engell and Ibsen (1952) found figures of up to 16 per cent from time to time in chest operations and attributed these rises in some cases to obstruction of the lower respiratory tract by secretions.

CARBON DIOXIDE READINGS AND THE BLOOD PRESSURE
It was hoped that some correlation between the blood pressure, pulse rate and the carbon dioxide levels would be found. No obvious relationship was noticed, however, probably because the hypercapnoea was not excessive. Some hypertension, i.e. a systolic pressure persistently raised 25 mm Hg above the bed-resting level was encountered in three cases (all fit young men). In all three cases the carbon
dioxide readings were well within the normal range. Another case was allowed to maintain a carbon dioxide percentage of 6.3–7.5 per cent for 30 minutes but the blood pressure remained at the patient's normal level of 110/70 mm Hg. On the other hand low blood pressure readings were recorded in two other cases where the carbon dioxide readings were approximately 7 per cent during the same period. The pulse rate appeared to be affected far more by factors such as the depth of anaesthesia in relation to the degree of surgical trauma and the cardiac accelerator action of gallamine than by these degrees of change in the carbon dioxide concentration.

DISCUSSION AND CONCLUSIONS

The clinical assessment as to the adequacy of respiration, either spontaneous or controlled, is not always satisfactory.

Spontaneous respiration.

It is common anaesthetic practice to give inhaled mixtures containing more than 20 per cent oxygen. This obviously presupposes that ventilation will be inadequate or the extra oxygen would not be needed. Experience of positive pressure respiration in a poliomyelitis unit shows that even with total respiratory paralysis air alone is satisfactory provided that ventilation is adequate (Smith et al., 1954; Lassen, 1953; Harries and Lawes, 1955). In fact extra oxygen is only needed in these cases during emergencies or where there is hyperpyrexia. Pask (1955) has pointed out the dangers of accepting spontaneous respiration as being adequate when depressant drugs or relaxants have been used. Cyanosis only indicates a high degree of suboxygenation. Lassen (1955) has stated that cyanosis occurs when the arterial oxygen saturation is reduced to approximately 82 per cent and it seems reasonable to assume that if the colour of the patient is used as the only guide to the presence or absence of suboxygenation then some degree of hypercapnia may have occurred long before cyanosis is evident. The degree of respiratory ventilation as assessed by the movements of the bag may be far from accurate. In one case intubation was performed under suxamethonium and the only drugs given after induction were pethidine 50 mg in two doses with nitrous oxide 6 litres and oxygen 2 litres. The carbon dioxide estimations were between 6.5 per cent and 7.2 per cent during the entire operation of one hour. In this case the movements of the bag appeared to be definitely satisfactory but, as the figures show, some degree of hypercapnoea had occurred.

Assisted respiration.

This method was only used in the early part of the series as the figures obtained were just as great as those found during spontaneous respiration. Pask (1955) points out that true assisted respiration is very difficult if not impossible to perform, and that properly assisted respiration soon becomes controlled respiration. If it is accepted that it is desirable to keep the carbon dioxide tension within normal limits there seems little reason for using this technique as a means of eliminating hypercapnoea.

Controlled respiration.

The control of respiration depends upon a number of factors which must vary
greatly in each individual case. These factors are—the sensitivity of the respiratory centre; the degree of muscular paralysis; the carbon dioxide tension of the arterial blood; the extent of the surgical stimuli and probably the state of activity of the stretch receptors. In a clinical condition which depends upon at least five factors it is extremely difficult to assess the role played by any single one of them.

As most anaesthetic agents depress the sensitivity of the respiratory centre to carbon dioxide the ideal level of carbon dioxide tension in controlled respiration would be within the normal range but unless some means is available to measure the pCO₂ or the percentage in "alveolar air" it is difficult to see how this ideal can be obtained. If relaxants or central depressants are the main factor in control then high "normal" carbon dioxide levels may occur. If carbon dioxide elimination predominates the patient may be overventilated, as respiratory movement can occur in overventilated patients in a light plane of anaesthesia if the surgical stimuli are proportionately excessive.

**Carbon dioxide narcosis.**

There are many illustrations in the literature of the narcotic action of carbon dioxide (Dripps and Comroe, 1947; Meduna, 1950). It has been used as an anaesthetic by Waters (1937) when one of the three cases described developed convulsions on the table. It must be emphasized, however, that in these cases where carbon dioxide was inhaled to produce unconsciousness the concentrations were extremely high. In the series described by Westlake et al. (1955), of carbon dioxide narcosis in emphysematous patients the majority had greatly raised carbon dioxide tensions in their arterial blood, i.e. over 100 mm Hg, though a number of patients were drowsy at figures around 80 mm Hg. On the other hand one patient was mentally alert when his arterial tension was 110 mm Hg, showing that the susceptibility of patients to carbon dioxide varies greatly.

*The general effects of carbon dioxide excess.*

The signs and symptoms of hypercapnoea are very protean and are dependent upon the degree of hypercapnoea and the susceptibility of the individual.

Gray and Fenton (1954) describe a case where presumed carbon dioxide excess caused delay in the return of normal respiration. In this case there was marked inadequacy of tidal air exchange compared with the forceful chest movement; tracheal and mandibular tug were much in evidence. Ibsen (1954) describes a rise in blood pressure, cold clammy skin, and gagging and bucking respiration with increased nasal and oral secretions as being due to carbon dioxide excess. Davies et al. (1955) suggest that in some cases of prolonged action of suxamethonium the cause may be due to hypercapnoea and describe lack of intercostal movement, tracheal tug and flaring of the nares. In their experiments with dogs they were able to reproduce the uncertain respiratory movements seen in their patients when the dogs received suxamethonium under conditions of hypercapnoea. Scurr (1954) gives an example, in a six-year-old child, of hypercapnoea simulating cur-
arization, though in this case there was no urgency of respiration or tracheal tug.

Seevers (1944) describes the inhalation of 7–12 per cent carbon dioxide as causing some rise in blood pressure, tachycardia, sweating and hypercapnoea. High concentrations, however, give a depressant effect with a failing respiration (Leake and Waters, 1929). Westlake et al. (1955) describe the general effect of high concentrations of carbon dioxide as being “warm sweating extremities, bounding pulse, low blood pressure, hypothermia and some mental clouding or coma.”

**Neurological effects of carbon dioxide poisoning.**

A common sign of carbon dioxide poisoning would appear to be muscular movement (Leake and Waters, 1929; Davies and Mackinnon, 1949; Hickman et al., 1952). Waters (1937) reports that “twitching of the facial muscles is one of the commonest signs of carbon dioxide excess in anaesthesia”. On the other hand, Pollock (1949) states that the convulsions induced by Metrazol are depressed by carbon dioxide inhalations.

Hypercapnoea may cause cerebral vasodilatation giving a rise of intracranial pressure (Goldensohn et al., 1951). Case and Haldane (1941), working on conscious volunteers breathing 6 per cent carbon dioxide, describe mental distress, photophobia and slight headache while the carbon dioxide was being inhaled and nausea when the subject left the experimental chamber and breathed air.

**Anaesthetic drugs and carbon dioxide excess.**

Many drugs commonly used in anaesthesia are stated to be adrenergic, e.g. morphine (Bodo et al., 1937), chloroform (Bathia et al., 1933), ether (Elliott, 1912), trichlorethylene (Johnstone, 1951), and cyclopropane (Allen et al., 1940). Johnstone (1950) has shown that carbon dioxide retention may augment the adrenergic effect of certain anaesthetic gases with the production of ventricular extrasystoles.

Beecher and Todd (1954) surveyed more than half a million anaesthetics and described a six-fold increase in anaesthetic deaths since the introduction of relaxants, and attribute this to some inherent toxicity of these agents which affects the cardiovascular system. Most British anaesthetists would probably not agree with their conclusions but can hardly argue with their facts. As the main untoward effect of relaxant drugs is underventilation, hypercapnoea and suboxygénation may have played a part in this increased mortality as suggested by Pask (1955). Johnstone (1955) has postulated that the use of adrenergic drugs in the presence of carbon dioxide excess would partially explain the raised mortality rate as the increase was more striking in the group where these drugs were used than in the group where a nitrous oxide-pethidine sequence was employed. Stead et al. (1953) and Harbord and Lucas (1953) have demonstrated that respiratory acidosis, to quite a high degree, may complicate apnoeic techniques with relaxants. Sims et al. (1951), investigating the effect of hypercapnoea and anoxia on the liver, demonstrated that carbon dioxide excess during anaesthesia caused considerable increase in the frequency and severity of hepatic damage with ether and in particular with chloroform.
Hyperventilation.

Excessive artificial ventilation carries its own risks but these are probably not so dangerous as those of hypercapnoea, and hyperventilation is less likely to occur during anaesthesia than hypoventilation, though Dundee (1952) found a rise in pH in manually controlled respiration. Many authors investigating the effects of hyperventilation describe hypotension with, in some cases, loss of consciousness (Seevers et al., 1939; Vincent and Thompson, 1928; Vincent and Cameron, 1915; Dale and Evans, 1922). Kety and Schmidt (1948) describe how mechanical overventilation produced a diminution of cerebral blood flow by 30 per cent which they ascribed as being due to a general vasoconstriction and to a fall in cardiac output; but in mechanical over-ventilation a number of other factors arise, such as the pressure of inspired gases, the inspiration-expiration ratio and the length of inspiration, and the effects they describe may have been due to causes other than alkalosis. Harwood (1938) states that the signs and symptoms of hyperventilation are "dizziness, blurring of vision, numbness and tingling of the extremities" and says that long continued hyperventilation leads to spasms of the muscles of the face, hands and feet, going on to stridor, generalized convulsions and unconsciousness. When this description is compared with that of Waters (1937) it would appear that there is considerable similarity between extreme hypercapnoea and extreme overventilation, though there is not likely to be any confusion during most anaesthetics.

It will be appreciated from the above quotations from the literature that the manifestations of hypercapnoea and respiratory alkalosis are very protean. The clinical signs said to be due to changes of carbon dioxide from the normal are capable of misinterpretation as they can be produced by other causes. Riskin et al. (1954) gave examples of hypertension during anaesthesia which were not due to hypercapnoea. Price et al. (1953) have shown that a rise in the systolic blood pressure can occur without a rise in the carbon dioxide tension though the mean increase is always greater if respiratory acidosis does exist. Westlake et al. (1955) demonstrated that severe hypercapnoea may cause hypotension. It must be borne in mind, however, that many of the effects described are due to extremely high abnormal variations of carbon dioxide tensions and are unlikely to be met with in ordinary anaesthetic practice.

SUMMARY

(1) A simple method of estimating the carbon dioxide content of end-of-expiration air is described.

(2) Figures of the carbon dioxide percentage are given as found during spontaneous, "assisted" and controlled respiration.

(3) The clinical effects of hypercapnoea and hyperventilation are described as gleaned from the literature.

(4) It is suggested that control of respiration during anaesthetic practice may be achieved within a wide range of carbon dioxide tensions unless some means is used to measure the carbon dioxide concentration.
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REFERENCES