CONTROLLED RESPIRATION: STANDARDIZATION OF VENTILATION

BY

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The technique of controlled respiration during anaesthesia was developed primarily to deal with respiratory inadequacy enforced on the patient by an open pneumothorax. This paper is concerned with developing means to adjust the volume of passive ventilation to a physiological level, the basis at present appearing to be entirely empirical.

Originally, in order to institute controlled respiration, active respiration was abolished in two ways or by a combination of the two (Nosworthy, 1941).

(1) By depressing the sensitivity of the respiratory centre, especially to carbon dioxide, by anaesthetics or other drugs.

(2) By reducing the carbon dioxide tension in the arterial blood by hyperventilation. In addition it is possible that artificial inflation abolished the patient's respiratory efforts by affecting the Hering-Breuer mechanism.

At a later date curarizing drugs came into use and, by paralysing the respiratory muscles, made it easier for the anaesthetist to take over the patient's external respiration. The widespread use of these drugs now provides the most frequent and essential indication for the use of controlled respiration.

The first two conditions suggest that assisted respiration may not have much practical application: during anaesthesia the respiratory centre is almost invariably depressed by anaesthetics or other depressant drugs. Recent observations revealed that during anaesthesia by thiopentone, pethidine, nitrous oxide and oxygen the alveolar carbon dioxide tension tended to stabilize at about 50 mm Hg (equivalent to 7 per cent carbon dioxide). If under these conditions the respiration is assisted in any efficient way the carbon dioxide tension will fall, so that the second mechanism will act and apnoea will result with full controlled respiration a necessity. In favour of assisted respiration it has been suggested that retention of some spontaneous respiratory effort is advantageous to the circulation; certainly the manner of artificial ventilation and the mean pressure in the respiratory tract are important if the circulation is not to be impaired (Cournand et al., 1947; Werko, 1947), but the present communication is not concerned with these considerations.

CARBON DIOXIDE RETENTION

The function of respiration is the supplying of oxygen and the elimination of carbon dioxide—these two are not syn-
onymous, nor is the assurance of one a
guarantee that the other is satisfactory.
In experiments on diffusion respiration
(Roth et al., 1947), dogs placed in an
atmosphere of oxygen and saturated with
oxygen were made apnoeic with anaes-
thesics, yet they remained well oxy-
genated indefinitely by virtue of the
haemoglobin oxygen pump and minimal
ventilation by cardiac movements; there
was, however, a progressive rise in carbon
dioxide tension to very high levels (250
mm Hg), death finally resulting from
carbon dioxide poisoning.

Under anaesthesia similar conditions
often prevail: there is an oxygen-rich
atmosphere and an oxygen-rich patient
who will remain a good colour with
minimal respiratory movements or none
for many minutes. It is evident that in
such patients carbon dioxide levels may
mount (Jooste, 1955). Even apart from
such gross respiratory depression a ten-
dency to carbon dioxide retention is ever
present during anaesthesia (Harbord et
al., 1953; Ellison, 1955). An oxygen-rich
atmosphere, by reducing respiratory drive
(Shephard, 1955), and by effects on
carbon dioxide carriage, will of itself tend
to cause carbon dioxide retention. Indeed,
in subjects with chronic respiratory
acidosis, oxygen administration by re-
moval of the anoxic respiratory drive can
precipitate carbon dioxide narcosis
(Donald, 1953) and depression, a danger
insufficiently appreciated (Mushin, 1955).
Ill effects from hypercapnoea may include
cardiac arrhythmias (Johnstone, 1950)
and cardiac arrest, hypertension and
vascular incidents, "cyclopropane shock"
(Dripps, 1947), and self-perpetuating
depression (Scurr, 1954). Effects on the
electroencephalogram have been reported
recently (Clowes, 1953). When the
alveolar carbon dioxide level attains 9 per
cent (in unanaesthetized subjects) res-
piratory stimulant effects are maximal—
further increase leads to progressive
depression. The stimulant effects may be
masked completely by anaesthetic drugs
 especiaIly curarizing agents) but a
tracheal tug on respiration is always a
suggestive sign. Carbon dioxide accu-
ulation with depression in the post-
operative period (Scurr, 1954) may be
a cause of postoperative death with mini-
mal autopsy findings (Pask, 1955).

Supposing instead of an oxygen-rich
mixture during anaesthesia air or 20 per
cent oxygen with 80 per cent nitrous
oxide be used for ventilation: if the
patient remains a good colour (in the
absence of anaemia, etc.), is it a guarantee
of satisfactory carbon dioxide balance?
Calculation shows (Affeldt et al., 1955;
Fenn et al., 1946) that under such con-
ditions hypoventilation such as to double
the alveolar carbon dioxide tension (40
mm Hg -> 80 mm Hg) reduces the alveolar
oxygen tension to 54 mm Hg. The oxy-
haemoglobin dissociation curve reveals
that this represents about 80 per cent
saturation in the arterial blood which is
just about the cyanosis threshold, i.e., the
carbon dioxide tension may be doubled
(equivalent to 11-12 per cent alveolar
carbon dioxide) before cyanosis provides
a warning of hypoventilation.

PHYSIOLOGICAL CONSIDERATIONS
In the resting individual pulmonary
ventilation is adjusted to the rate of
metabolism; the link between metabolism
and breathing is mainly the alteration in
the carbon dioxide tension of the blood, to which the respiratory centre is exquisitely sensitive unless anaesthesia interferes with its sensitivity or blocks the operation of the ventilatory muscles. When the inspired carbon dioxide is negligible and conditions are steady the alveolar ventilation required may be defined in terms of the carbon dioxide production and the alveolar carbon dioxide concentration, as follows:

\[
\text{Alveolar ventilation per minute} = \frac{\text{Volume of CO}_2 \text{ produced per minute}}{\text{Alveolar CO}_2 \text{ fraction}}
\]

E.g., in a patient producing 200 ml CO\(_2\) per minute, to maintain alveolar CO\(_2\) at 5 per cent:

\[
\text{Alveolar ventilation per minute} = \frac{200}{5/100} = 4 \text{ litres per minute.}
\]

The physiological level of the carbon dioxide fraction in alveolar air is 5.6 per cent (representing 40 mm Hg tension) and this is normally the level which artificial ventilation during anaesthesia should be adjusted to maintain.

Carbon dioxide output can be calculated from published data of metabolic rate (Benedict and Talbot, 1921) and thus a graph can be drawn to show carbon dioxide production based on body weight (Radford, 1955), this being the simplest basis for use during anaesthesia, although allowances must be made for the usual sources of error in body weight calculations.

The actual alveolar ventilation per minute is given by the following equation:

\[
\text{Alveolar ventilation per minute} = (\text{Tidal air} - \text{Dead space}) \times \text{Respiratory rate per minute}
\]

The anatomical dead space can also be calculated from the body weight (Radford, 1955) and by happy chance is found to be equal to 1 ml per pound body weight. (Disease, e.g., emphysema, or partial inflation of the lungs by constant pressure in the anaesthetic bag can grossly increase the dead space effect and predispose to carbon dioxide retention (Siebecher et al., 1954; Crafoord, 1938).) Note the important effect of dead space multiplied by respiratory rate on the minute volume. Thus from body weight, (1) carbon dioxide output and (2) dead space can be calculated: the physiological carbon dioxide fraction being known, if a ventilation rate now be chosen (e.g. 20 per minute) the equations can be solved to give the tidal air necessary for a patient of given weight at the chosen respiratory rate. From these data Radford (1955) has constructed a nomogram to provide ventilation standards for use during artificial respiration and anaesthesia. To provide an example from Radford’s nomogram:

Male, 150 pounds: rate = 20 per minute, tidal air needed = 350 ml, i.e. total minute volume = 20 \times 350 ml = 7 litres.

Therefore, if during anaesthesia an approximately constant rate of ventilation (e.g. 20 per minute) be maintained some method of measuring the total minute volume could be employed to implement the calculated standard ventilation.

**Measurement of Ventilation During Anaesthesia**

Various types of apparatus have been tried and found suitable for the purpose of measuring the total minute volume of ventilation:
(1) Dry Gas Meter, Type D.1 (Parkinson and Cowan Industrial Products, London). This type of apparatus is inexpensive but cumbersome, the resistance is very low and it is placed across the inspiratory tube of the circle absorption apparatus.

(2) Bennett Ventilation Meter.

(3) The Jet Anemometer (Wright, 1955; Leader in Brit. med. J., 1955). This apparatus is compact, light and of low resistance. It seems highly suitable for incorporation in anaesthetic circuits to monitor the minute volume of ventilation.

(4) A volumetric rebreathing bag or bellows which shows the tidal volume, e.g. the Blease Pulmoflator or the apparatus of Eastwood and Harbord (1954). A new type of bellows which can be set for a given tidal volume is at present being developed.

These instruments can also be used to check the level of spontaneous ventilation at the end of operation to see if it is adequate and so avoid the perils of respiratory depression in the recovery period.

Application of calculated ventilation volumes during anaesthesia and check estimation of alveolar carbon dioxide levels by the Draeger carbon dioxide analyser has so far demonstrated very satisfactory conformity.

Checks on ventilation levels attained during controlled respiration by various anaesthetists have shown that, apart from the risk of underventilation, in other hands hyperventilation is common.

Minute volumes of 10 to 12 litres per minute were often observed; these would often produce double the required amount of alveolar ventilation. The possible dangers of such excessive ventilation have been pointed out (Mushin, 1955; Campbell, 1953), but are probably less than those of underventilation (Pask, 1948). Changes in blood pH have been noted during anaesthesia as a result of hyperventilation (Dundee, 1952); among other results the skin may be pale cold and moist (Wright, 1945); the significance of the Bohr effect in causing oxygen lack has not been fully assessed. Depletion of the total body store of carbon dioxide assessed at 25 to 30 litres (Spurrell, 1955) may take some time, and presumably would be detected first by a fall in carbon dioxide levels in a sample of mixed venous blood; reports on such changes in body carbon dioxide stores are now available (Rahn and Farlie, 1955).

SUMMARY

The assessment of ventilation during anaesthesia with controlled respiration rests at present on an empirical basis.

The alveolar ventilation required can be estimated from physiological data. Means are suggested for observing ventilation volumes during anaesthesia so that the risks of hypoventilation and hyperventilation can be avoided.

REFERENCES

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**DIRECTOR OF THE RESEARCH DEPARTMENT OF ANAESTHETICS**

of the **FACULTY OF ANAESTHETISTS of the ROYAL COLLEGE OF SURGEONS OF ENGLAND**

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