THE EFFECT OF ANAESTHESIA AND INTERMITTENT POSITIVE PRESSURE VENTILATION WITH DIFFERENT FREQUENCIES ON THE ANATOMICAL AND ALVEOLAR DEADSPACE

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SUMMARY

Deadspace was measured in nine healthy subjects in the supine position, premedicated but awake and breathing spontaneously at a rate of 12 b.p.m. and subsequently under anaesthesia with artificial ventilation with frequencies of 12 and 24 b.p.m. The minute volume was kept at a relatively constant value. The physiological deadspace was calculated using the Bohr equation and the division into anatomical and alveolar deadspace was made with the aid of capnography. Physiological deadspace was increased by anaesthesia and IPPV, mainly as a consequence of increased rebreathing in the apparatus deadspace. There was no significant change in the anatomical deadspace. Thus, the expected reduction in deadspace brought about by endotracheal intubation was nullified by an increase in the anatomical deadspace distal to the carina. The $V_{anat}/V_T$ ratio remained constant on changing the respiratory frequency. A significant alveolar deadspace was measured during spontaneous breathing. This was unchanged by the induction of anaesthesia and the institution of artificial ventilation. On changing the frequency, the $V_{alv}/V_T$ ratio remained constant. It is concluded that both the anatomical and the alveolar deadspaces increase with increasing tidal volume, but are unaffected by the breathing rate.

During anaesthesia and intermittent positive pressure ventilation, the physiological deadspace, as defined by the sum of the volumes of all spaces in which rebreathing occurs, is greater than it is under spontaneous breathing (Cooper, 1967; Kain, Panday and Nunn, 1969). It is increased further in relation to the tidal volume ($V_d/V_T$ ratio) by increasing the frequency of ventilation, in both healthy subjects and in those undergoing ventilator treatment for respiratory failure (Hedenstierna and Löfström, 1972; Hedenstierna, 1972). However, it is not clear which constituent of the physiological deadspace is increased by these factors. This study has been performed to investigate the effect of anaesthesia and intermittent positive pressure ventilation with different respiratory frequencies on the anatomical and alveolar deadspaces.

PATIENTS

Nine subjects, healthy apart from the condition requiring surgery, were investigated before and during anaesthesia. There were seven women and two men, aged 23–67 years (table I). The scope of the investigation had been described to the patients and their permission obtained.

METHODS

Anaesthesia

After premedication with diazepam 0.15 mg/kg, anaesthesia was induced by the i.v. injection of droperidol 0.05 mg/kg and a sleep-dose of thiopentone. After inflation of the lungs with oxygen, endotracheal intubation was facilitated by the administration of suxamethonium 10 mg/kg, preceded by a very small dose of pancuronium (0.5 mg) to minimize muscular discomfort after operation. After intubation, fentanyl was given (approximately 0.002 mg/kg), and an infusion of thiopentone 1 mg/ml was started at a rate of approximately 1.5 ml/min to eliminate the risk of awareness during the examination. Further increments of fentanyl 0.05–0.1 mg were given as required. With one exception, all the patients were ventilated with air.

Ventilator. The investigations under artificial ventilation were performed using a volume-limited ventilator with accelerating gas flow (Engström 200 or 300, LKB Medical, Stockholm).
Physiological deadspace

The physiological deadspace was determined according to Enghoff's modification of the Bohr equation (Enghoff, 1938).

\[
VD_{phys} = \frac{[P_{acO_2} - P_{eCO_2}]}{(P_{acO_2} - P_{iCO_2})} \times VT
\]

\[\text{VT} = \text{tidal volume} \]

\[P_{acO_2} = \text{arterial carbon dioxide tension} \]

\[P_{eCO_2} = \text{mean } CO_2 \text{ partial pressure in expirate} \]

\[P_{iCO_2} = \text{CO}_2 \text{ partial pressure in inspirate} \]

\[P_{acO_2}\] was determined in blood samples taken from a percutaneous Teflon catheter inserted into the femoral artery. The analyses were performed immediately after the blood samples had been taken, utilizing a Severinghaus electrode (E 5036, Radiometer, Copenhagen). The electrode was calibrated with water-saturated gas at the correct temperature. The mean of two analyses was taken. VT and \[P_{eCO_2}\] were obtained by the collection and analysis of the expirate. During spontaneous breathing the expirate was collected from the mouth-piece through a valve into a Douglas bag, and during artificial ventilation by means of a pneumatic valve (LKB Medical, Stockholm), to prevent contamination by compressed gas in the ventilator tubes. Gas analysis was performed according to the method of Scholander (1947).

Apparatus deadspace

The volume of the apparatus deadspace (\(VD_{app}\)) was obtained by filling that part of the apparatus in which rebreathing took place with water.

Anatomical deadspace

Three methods were used to determine the anatomical deadspace: the equal-area method according to Fowler (1948) and two methods based on modifications of the Bohr equation.

The carbon dioxide content of the expirate was continuously recorded at the mouth-piece by means of an infra-red meter (E. Jaeger, Wurzburg) in order to determine the anatomical deadspace according to the equal-area method. The volume of the expirate was registered simultaneously using a pneumotachograph (flow head, Fleisch, Godart, Bilthoven, Holland; differential pressure transducer EMT 35 and amplifier EMT 31, Elema-Schöndander, Stockholm; integrator AN-1, SRA, Stockholm). Both signals were recorded using an ink-jet writer (Minografi 81, Elema-Schöndander, Stockholm).

The anatomical deadspace was also determined according to the following two formulae, which are modifications of the Bohr equation (Nunn, 1969):

\[
VD_{anat} = \frac{([P_{eCO_2} - P_{iCO_2}]/[P_{eCO_2} - P_{iCO_2}]) \times VT}{(i)}
\]

\[
VD_{anat} = \frac{[P_{acO_2} - P_{eCO_2}]/[P_{acO_2} - P_{iCO_2}]) \times VT}{(ii)}
\]

\[P_{eCO_2} = \text{end tidal } CO_2 \text{ partial pressure.} \]

\[P_{iCO_2} = \text{mean } CO_2 \text{ partial pressure in inspirate.} \]

Statistical analysis.

Statistical analysis of the results was made using Student's \(t\) test for paired values.

PROCEDURE

All investigations were performed with the subjects premedicated and in the supine position. They were investigated both awake, breathing spontaneously at a rate of 12 b.p.m. and under anaesthesia with artificial ventilation at frequencies of 12 and 24 b.p.m., the respiratory minute volume being kept relatively constant (table II). When the conscious subjects had
been breathing for 15 min at the rate determined by a metronome, the expire was collected over a period of 5 min, while two arterial samples were taken and 15–20 breaths were recorded by the carbon dioxide analyser. The anatomical deadspace was determined on three consecutive breaths according to the equal-area method. The patient was then anaesthetized and ventilated mechanically.

After 30 min of artificial ventilation with air at lower frequency, while the alveolar deadspace was approximately 70 ml. after changing on changing the frequency of ventilation. The alveolar-arterial P\(_{\text{CO}_2}\) difference was significantly greater with artificial ventilation, but was not changed with anaesthesia and artificial ventilation it was under anaesthesia. The result of bypassing the upper airways by the endotracheal tube was offset by an increase in the anatomical deadspace distal to the carina. The ratio between the deadspace volume and the tidal volume (V\(_{\text{alv}}\)/VT) was the same with spontaneous ventilation as it was with artificial ventilation with different frequencies.

The anatomical deadspace was determined after a 5-sec end-inspiratory pause in two patients. It was reduced by approximately 20\% irrespective of the frequency of the preceding ventilation (fig. 3).

With the patient awake and breathing spontaneously, the alveolar deadspace was approximately 70 ml. There was no significant change with anaesthesia and artificial ventilation at the lower frequency. With the higher frequency, while the alveolar deadspace was approximately halved, its relationship to the tidal volume V\(_{\text{alv}}\)/VT was unchanged.

### RESULTS

**Alveolar, end-expiratory and arterial P\(_{\text{O}_2}\) and P\(_{\text{CO}_2}\) (table II)**

The alveolar-arterial P\(_{\text{O}_2}\) difference was significantly greater with artificial ventilation, but was not changed on changing the frequency of ventilation.

The P\(_{\text{CO}_2}\) was less during artificial ventilation than during spontaneous breathing and greater with the higher frequency of ventilation. The end-expiratory P\(_{\text{CO}_2}\) was approximately 4 mm Hg less than the arterial value under all conditions.

**Deadspace (tables III and IV, figs. 1-3)**

The physiological deadspace was greater under anaesthesia and artificial ventilation than under spontaneous breathing with the same frequency, both in absolute terms and in relation to the volume breathed (V\(_{\text{DPHYS}}\)/VT). While the physiological deadspace was less with the higher frequency of artificial ventilation the V\(_{\text{DPHYS}}\)/VT ratio was greater.

The functional apparatus deadspace with spontaneous breathing was approximately 30 ml, and under anaesthesia and artificial ventilation it was 80 ml.

The results derived from the different methods of calculating the anatomical deadspace are shown in table IV. They do not differ to any significant extent. After the induction of anaesthesia, endotracheal intubation and the institution of intermittent positive pressure ventilation, the expected reduction in the deadspace brought about by intubation (Kain, Panday and Nunn, 1969) did not occur. Thus the anatomical deadspace was approximately the same with the patient awake and breathing spontaneously as it was under anaesthesia. The result of bypassing the upper airways by the endotracheal tube was offset by an increase in the anatomical deadspace distal to the carina. The ratio between the deadspace volume and the tidal volume (V\(_{\text{alv}}\)/VT) was the same with spontaneous ventilation as it was with artificial ventilation with different frequencies.

### Table II. Mean values and SD for tidal volume (VT), alveolar (A), end-tidal (T) and arterial (a) gases.

<table>
<thead>
<tr>
<th></th>
<th>VT (ml)</th>
<th>Partial pressures (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>PA(_{\text{O}_2})</td>
</tr>
<tr>
<td>Spontaneous, f:12</td>
<td>657±101</td>
<td>113±8</td>
</tr>
<tr>
<td>Artificial, f:12</td>
<td>740±68</td>
<td>121±2</td>
</tr>
<tr>
<td>Artificial, f:24</td>
<td>372±32</td>
<td>116±5</td>
</tr>
</tbody>
</table>

**Significance of differences**

- Spontaneous-artificial P = <0.01<br>- Artificial f:12-f:24 P = <0.001
TABLE IV. **Anatomical deadspace during anaesthesia and artificial ventilation with 12 b.p.m. as determined by three different methods: n=9.**

<table>
<thead>
<tr>
<th>Method</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1: equal area method (Fowler, 1948)</td>
<td>115.0</td>
<td>33.5</td>
</tr>
<tr>
<td>2: derivation from the Bohr equation (i) (see methods)</td>
<td>112.3</td>
<td>37.5</td>
</tr>
<tr>
<td>3: derivation from the Bohr equation (ii) (see methods)</td>
<td>96.7</td>
<td>37.4</td>
</tr>
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There were no significant differences between the methods.

**FIG. 1.** Physiological deadspace with subdivisions (apparatus, anatomical and alveolar deadspace) during spontaneous breathing (S.B.) awake, and during artificial ventilation (Art. Vent.) when anaesthetized.

**FIG. 2.** Physiological deadspace/tidal volume ratio with subdivisions during spontaneous breathing and artificial ventilation.

**DISCUSSION**

The measurement of deadspace using carbon dioxide as a tracer gas postulates a synchronous emptying of the alveoli during expiration. Previous studies of gas distribution have shown that in healthy subjects it is the same during anaesthesia with artificial ventilation as with spontaneous breathing when the subject is awake (Rehder et al., 1971; Hedenstierna and McCarthy, 1975). Furthermore, three different methods of calculating the anatomical deadspace were employed in order to reduce to a minimum sources of error in the graphical analyses.

Fowler's (1948) method of calculating the anatomical deadspace is dependent on the end-inspiratory pause; the shorter the pause, the greater is the calculated deadspace (Bartels et al., 1954; Shepard et al., 1957; see also fig. 3 in this study). Engel and his coworkers (1973) have shown convincingly that this is a result of the mixing of alveolar gas with that in the airways, brought about by the heartbeat and by diffusion. In order to reduce this source of error, we have used a very short end-inspiratory pause, less than 1 sec.

A moderate decrease in arterial systolic pressure
from a mean of 120 mm Hg (range 130–105) to a mean of 110 mm Hg (range 130–85), accompanied the induction of anaesthesia and the institution of artificial ventilation. While this may have been a result of a reduction in cardiac output, it can hardly have affected our results. Such a decrease may be expected to decrease the uniformity of blood-flow distribution to the lungs, and thus increase the alveolar deadspace during artificial ventilation. We found no such increase.

The effect of anaesthesia and artificial ventilation

While the physiological deadspace was significantly greater during anaesthesia with artificial ventilation than it was with the patient awake and breathing spontaneously, this increase was less, both absolutely and in relation to the tidal volume, than that reported in earlier investigations in subjects with healthy lungs (Campbell, Nunn and Peckett, 1958; Nunn, Bergman and Coleman, 1965; Cooper, 1967; Hedenstierna and Löfström, 1972). That our results differ from those of earlier workers may be explained by differences in apparatus deadspace, and that our subjects were, with one exception, ventilated with air. Thus, Sykes and Finlay (1971) have shown that the physiological deadspace increases on enriching the inspirate with oxygen during anaesthesia with artificial ventilation. They suggest that this increase is a result of an increase in alveolar deadspace caused by dilatation of pulmonary vessels by hyperoxygenation, or by a reduction in the cardiac output.

The anatomical deadspace distal to the carina was increased during intermittent positive pressure ventilation by an amount approximately equal to the volume bypassed in the upper airways. This increase in deadspace may be caused by dilatation of the airways because of the increased intrathoracic pressure and by a reduction in tone in the bronchial muscle. Another possible explanation may be a reduced mixing of gas in the airways with that in the alveoli with artificial ventilation. Thus Rehder and colleagues (1971) have drawn attention to diminished cardiac oscillations on the tracer gas recordings during artificial ventilation. However, any such possible variation in gas mixing must be of small significance when the end-inspiratory pause is very short (Engel et al., 1973).

The existence of an alveolar deadspace in healthy subjects breathing spontaneously can seldom be demonstrated (Nunn and Hill, 1960). However, we found that it could be demonstrated in our subjects when lying down and premedicated. This alveolar deadspace was not significantly increased by the subsequent administration of anaesthesia and the institution of artificial ventilation. Nunn and Hill demonstrated that the alveolar deadspace was increased above the value expected in patients premedicated and anaesthetized, both when breathing spontaneously and when ventilated. It may be that the increase which they found was merely a result of premedication and the horizontal position.

The effect of the frequency of ventilation on the anatomical and alveolar deadspaces

As in earlier studies, the physiological deadspace ratio (VDphys/VT) proved to be dependent on the frequency of artificial ventilation: the higher the frequency, the higher the deadspace ratio. This was a result of the apparatus deadspace accounting for a greater part of the tidal volume, since the anatomical and alveolar deadspaces ratios were unaltered. Since alveolar deadspace may be taken as an approximate index of the uniformity of blood-flow distribution to the lung (Severinghaus and Stupfel, 1957) these findings point to a reasonably constant pattern of ventilation and perfusion when the frequency of ventilation is altered.

ACKNOWLEDGEMENTS

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REFERENCES


**EFFETS DE L'ANESTHESIE ET VENTILATION AU MOYEN DE RESPIRATEURS A PRESSION POSITIVE INTERMITTENTE A DIFFERENTES FREQUENCES SUR L'ESPACE MORT ANATOMIQUE ET SUR L'ESPACE MORT ALVEOLAIRE**

**RESUME**

L'espace mort a été mesuré sur neuf sujets en bonne santé en décubitus, prémédiqués mais éveillés et respirant spontanément au rythme de 12 respirations par minute que l'on a par la suite mis sous anesthésie avec ventilation artificielle à des fréquences de 12 et de 24 respirations par minute. Le volume par minute a été maintenu à une valeur relativement constante. L'espace mort physiologique a été calculé à l'aide de l'équation de Bohr et la séparation en espace mort anatomique et espace mort alvéolaire a été faite par capnographie. L'espace mort physiologique a été augmenté par l'anesthésie et la ventilation au moyen de respirateurs à pression positive intermittente, surtout en tant que conséquence de l'augmentation de la re-ventilation dans l'espace mort de l'appareil. Il n'y a eu aucun changement important en l'espace mort alvéolaire. Ainsi, la réduction de l'espace mort alvéolaire a été causée par le tubage endotrachéal a été annulée par l'augmentation de l'espace mort anatomique distal par rapport à la carène. Le rapport \( \frac{V_{p\text{anat}}}{V_T} \) est resté constant lors des changements de la fréquence respiratoire. On a mesuré un espace mort alvéolaire significatif pendant la respiration spontanée. Ceci n'a pas été modifié par l'induction de l'anesthésie et l'adoption de la ventilation artificielle. Lorsqu'on a modifié la fréquence, le rapport \( \frac{V_{p\text{alv}}}{V_T} \) est resté constant. On en a conclu que les espaces morts anatomique et alvéolaire augmentent avec l'accroissement du volume de l'air de respiration mais qu'ils ne sont pas affectés par le rythme de la respiration.

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**DIE AUSWIRKUNG VON NARKOSE UND VON INTERMITTIERENDER POSITIV-DRUCKBELUFTUNG (IPPV) MIT VERSCHIEDENEN FREQUENZEN AUF DEN ANATOMISCHEN UND ALVEOLAREN TOTRAUM**

**ZUSAMMENFASSUNG**

Der Totraum wurde bei neun gesunden, liegenden Versuchspersonen gemessen, die einer Behandlung unterzogen waren, aber bei Bewusstsein waren und spontan mit 12 Atemzügen pro Minute atmeten; in der darauffolgenden Narkose unter künstlicher Beatmung atmeten sie mit 12-14 Atemzügen pro Minute. Das Minutenvolumen wurde auf einem relativ konstanten Wert gehalten. Der physiologische Totraum wurde unter Verwendung der Bohr-Gleichung errechnet, und die Teilung in anatomischen und alveolaren Totraum wurde mittels Capnographie durchgeführt. Der physiologische Totraum wurde durch Narkose und durch IPPV vergrößert, vor allem als Folge von Wiedereinatmung im Totraumapparat, während es beim anatomischen Totraum zu keiner wesentlichen Veränderung kam. So wurde die erwartete Verringerung von Totraum durch endotrachale Röhrenbeihilfe aufgrund einer Vergrößerung des anatomischen Totraums distal vom Kamm aufgehoben. Das Verhältnis \( \frac{V_{p\text{alv}}}{V_T} \) blieb bei Veränderung der Atemfrequenz konstant. Ein signifikanter alveolarer Totraum wurde bei spontaner Atmung gemessen. Er wurde durch die Herbeiführung der Narkose und einer künstlichen Beatmung nicht verändert. Bei Veränderung der Frequenz blieb das Verhältnis \( \frac{V_{p\text{alv}}}{V_T} \) konstant. Zusammenfassend wird festgestellt, daß sowohl der anatomische wie auch der alveolare Totraum sich mit steigendem Atemvolumen vergrößern, von der Atmungsgeschwindigkeit aber unbeeinflußt sind.

**EL EFECTO DE LA ANESTESIA Y LA VENTILACION POR PRESION POSITIVA INTERMITENTE CON DIFERENTES FRECUENCIAS EN LOS ESPACIOS VACIOS ANATOMICOS Y ALVEOLARES**

**SUMARIO**

Se midió el espacio vacío en nueve personas sanas en posición supina, a los que se había administrado medicamentos, pero estaban despiertos y respiraban espontáneamente a una velocidad de 12 exhalaciones por minuto y posteriormente bajo anestesia con ventilación artificial a frecuencias de 12 y 24 respiraciones por minuto. El volumen por minuto se mantuvo a un valor relativamente constante. El espacio vacío fisiológico se calculó utilizando la ecuación de Bohr y la división entre espacio vacío anatómico y alveolar se realizó por capnografía. El espacio vacío fisiológico se aumentó por la anestesia y la ventilación por presión positiva intermitente, principalmente como consecuencia de creciente respiración en el espacio vacío del aparato. No se registró cambio importante alguno en el espacio vacío anatómico. Así pues, la reducción de espacio vacío provocada por la entubación endotráquea quedó anulada al aumentar el espacio vacío anatómico distal de la carina. La relación \( \frac{V_{p\text{alv}}}{V_T} \) permaneció constante al cambiar la frecuencia respiratoria. Durante la respiración espontánea se midió un espacio vacío alveolar importante. Este espacio permaneció inalterado por la inducción de anestesia y la utilización de ventilación artificial. Al cambiar la frecuencia, la relación \( \frac{V_{p\text{alv}}}{V_T} \) permaneció constante. Se concluye, por lo tanto, que los espacios vacíos anatómicos y alveolares aumentan con el creciente volumen de impulso, pero no se ven afectados por el ritmo de respiración.