PHYSIOLOGICAL SHUNTING AND DEADSPACE DURING SPONTANEOUS RESPIRATION WITH HALOTHANE-OXYGEN ANAESTHESIA AND THE INFLUENCE OF INTUBATION ON THE PHYSIOLOGICAL DEADSPACE

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SUMMARY

In thirteen patients the physiological deadspace:tidal volume ratio and the alveolar to arterial oxygen tension difference have been measured immediately before and during anaesthesia with halothane in oxygen after 30–70 minutes spontaneous respiration. The mean values were unchanged and it was concluded that the physiological shunt was also unaltered except in two instances which are discussed. In a further series of ten patients an average reduction of 27 ml in the physiological deadspace was measured as a result of endotracheal intubation.

Several investigators have found an increase in the physiological deadspace (Askrog et al., 1964) and physiological shunt (Nunn, 1964) during halothane anaesthesia.

However, these measurements vary quantitatively between individuals, both physiologically and in disease, and in the same individual according to the volume history of the lungs (Laver et al. 1964; Mead and Collier, 1959), to body posture (Riley et al., 1959) or to the administration of drugs (Dripps and Severinghaus, 1955). It is apparent that a conclusive assessment of the effect of general anaesthesia on individual patients is not possible in the absence of preanaesthetic control measurements.

In the studies reported here, measurements of the physiological deadspace:tidal volume ratio and of the physiological shunt were made immediately before induction of anaesthesia and subsequently during spontaneous respiration of halothane in oxygen. The patients were intubated and studies of the effect of intubation on the physiological deadspace are also reported.

METHODS

Study I.

In thirteen patients the Vdp/Vt ratio and the alveolar to arterial oxygen tension difference (A-a Po₂ diff.) were measured before and during anaesthesia. Details of the patients, operations, premedication, medical condition and oral or nasopharyngeal temperature are given in table I. Also shown is the time interval between induction of anaesthesia and the subsequent measurement, which averaged 51 minutes.

Premedication was given 40–60 minutes before the pre-operative measurements were made. Anaesthesia was induced with a sleep dose of sodium thiopentone (200–350 mg) and intubation with a cuffed endotracheal tube was assisted with suxamethonium (50–100 mg); manual ventilation with halothane in oxygen was maintained until spontaneous respiration was re-established, at which time the endotracheal catheter mount was connected to a non-return valve (Hook and Tucker Ltd.) and a circle absorber system into which flowed fresh oxygen and halothane.

For the pre-operative measurements the patients breathed 100 per cent oxygen via a mouthpiece...
and noseclip, through the same valve and circle system.

The circle system consisted of a 6-litre Palmer multipurpose spirometer combined with a non-return valve, mixing chamber and carbon dioxide absorber (fig. 1); with this arrangement the patients could breathe for an indefinite period with fresh gas supplied and the escape valve open, denitrogenation could be achieved, and a steady state was approached.

During periods of measurement the fresh gas flow was discontinued, the escape valve closed and the spirometer recorder switched on; the patients now breathed in a closed circle, and samples of blood and gas were obtained as the recording proceeded.

The gas samples were withdrawn slowly into siliconized 20-ml syringes; duplicate samples of inspired gas were taken from tap I (fig. 1), and triplicate samples of mixed expired gas from tap M: the syringes were then capped. During the recording period blood was withdrawn slowly into a 5-ml syringe, from an indwelling Riley needle in the radial artery; the syringe contained a mixing disc and the deadspace was filled with heparin (5000 U/ml); the blood samples were stored in ice/water until analyzed.

At the completion of anaesthesia the lungs were manually over-inflated before extubation.

Study II.

The physiological deadspace was measured in ten anaesthetized adults, first when intubated and then after the endotracheal tube had been removed and substituted with a Dräger mouthpiece and noseclip. The depth of anaesthesia was controlled to maintain the tidal volume constant; the difference between the two measurements was therefore the volume by which the physiological deadspace had been reduced by endotracheal intubation.

The first five patients, data from whom are

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**TABLE I**

*Study I. Details of the thirteen patients and surgical procedures performed.*

Temperature °C refers to oral or nasopharynx sites.

A=Atropine 0.6 mg. M=Morphine 15 mg. P=Pethidine 100 mg.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Weight (lb.)</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Pre-medication</th>
<th>Operation</th>
<th>Induction to study interval (min)</th>
<th>Temperature °C</th>
<th>Medical condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>160</td>
<td>80</td>
<td>F</td>
<td>Atropine 0.6</td>
<td>Fractured hip</td>
<td>40</td>
<td>37.6</td>
<td>37.1</td>
</tr>
<tr>
<td>2</td>
<td>163</td>
<td>48</td>
<td>M</td>
<td>P &amp; A</td>
<td>Meniscectomy</td>
<td>70</td>
<td>36.8</td>
<td>36.7</td>
</tr>
<tr>
<td>3</td>
<td>149</td>
<td>44</td>
<td>M</td>
<td>M &amp; A</td>
<td>Fusion, wrist</td>
<td>30</td>
<td>36.8</td>
<td>Fit</td>
</tr>
<tr>
<td>4</td>
<td>161</td>
<td>20</td>
<td>M</td>
<td>M &amp; A</td>
<td>Meniscectomy</td>
<td>65</td>
<td>36.4</td>
<td>36.9</td>
</tr>
<tr>
<td>5</td>
<td>146</td>
<td>37</td>
<td>F</td>
<td>P &amp; A</td>
<td>Skin graft, hand</td>
<td>50</td>
<td>36.5</td>
<td>36.2</td>
</tr>
<tr>
<td>6</td>
<td>181</td>
<td>35</td>
<td>M</td>
<td>M &amp; A</td>
<td>Meniscectomy</td>
<td>60</td>
<td>37.1</td>
<td>Fit</td>
</tr>
<tr>
<td>7</td>
<td>142</td>
<td>72</td>
<td>M</td>
<td>Phenergan 50</td>
<td>Fractured hip</td>
<td>40</td>
<td>36.7</td>
<td>36.6</td>
</tr>
<tr>
<td>8</td>
<td>155</td>
<td>54</td>
<td>F</td>
<td>P &amp; A</td>
<td>Keller's bilateral</td>
<td>55</td>
<td>36.7</td>
<td>Fit</td>
</tr>
<tr>
<td>9</td>
<td>140</td>
<td>23</td>
<td>M</td>
<td>M &amp; A</td>
<td>Meniscectomy</td>
<td>35</td>
<td>37.2</td>
<td>37.2</td>
</tr>
<tr>
<td>10</td>
<td>151</td>
<td>60</td>
<td>F</td>
<td>M &amp; A</td>
<td>Fractured hip</td>
<td>30</td>
<td>35.4</td>
<td>35.2</td>
</tr>
<tr>
<td>11</td>
<td>143</td>
<td>17</td>
<td>M</td>
<td>M &amp; A</td>
<td>Dislocated shoulder</td>
<td>75</td>
<td>36.9</td>
<td>37.1</td>
</tr>
<tr>
<td>12</td>
<td>130</td>
<td>60</td>
<td>F</td>
<td>P &amp; A</td>
<td>Fractured tibia</td>
<td>65</td>
<td>36.4</td>
<td>36.3</td>
</tr>
<tr>
<td>13</td>
<td>161</td>
<td>66</td>
<td>M</td>
<td>P &amp; A</td>
<td>Fractured hip</td>
<td>50</td>
<td>36.6</td>
<td>36.5</td>
</tr>
</tbody>
</table>
shown in table IV, were connected either by endotracheal tube or mouthpiece to the circle system described above, and were anaesthetized with halothane in oxygen. The remaining five patients were anaesthetized with ether in air from an EMO Inhaler, and the gas emerging from the expiratory limb of the Hook and Tucker valve was collected in a Douglas bag. The volume of the bag was measured by expelling its contents through a Parkinson-Cowan meter, after taking samples of mixed expired gas for analysis.

The inspired gas was sampled from the inspiratory side of the non-return valve and a sample of blood was withdrawn at the start and at the finish of each gas collection period; measurements from this pair of blood samples were averaged for use in the calculations.

**Blood analysis.**

The arterial oxygen tension (PaO₂) was measured polarographically with a modified Clark Cell (Silver, 1963); the method of calibration has been described elsewhere and the overall random error of measurement has been shown to have a coefficient of variation of 3 per cent (Marshall, in preparation, 1966). Factors were used to correct the measured blood oxygen tensions at the electrode temperature (36.5–37°C) to the actual body temperature of the patient. The factors described by Bradley, Stupfel and Severinghaus (1956) were calculated to correct for the shift in position of the haemoglobin dissociation curve. As has been pointed out by Hedley-Whyte and Laver (1964), these are inapplicable when the haemoglobin is fully saturated at an oxygen tension above about

![Diagram of the circle system](https://academic.oup.com/bja/article-abstract/38/12/912/277195)
250 mm Hg. The changes in oxygen tension then become dependent on changes in the solubility coefficients of oxygen in blood. The correction factors suggested by the latter authors have therefore been used when the arterial oxygen tension exceeded 250 mm Hg.

The arterial carbon dioxide tension (\(P_{a\text{CO}_2}\)), pH, and standard bicarbonate (\(\text{HCO}_3^-\)) were measured using the micro-Astrup electrode for whole blood and the Siggaard-Andersen nomogram (Siggaard-Andersen and Engel, 1960). The factors described by Rosenthal (1948) were used to correct the values for any temperature difference between the electrode and the patient.

**Gas analysis.**

The carbon dioxide tension of the mixed expired gas (\(P_{e\text{CO}_2}\)) was measured with a carbon dioxide electrode (Radiometer Ltd.).

The oxygen tension of the inspired gas (\(P_{i\text{O}_2}\)) was measured polarographically; the random error of gas analysis showed a coefficient of variation of 1.6 per cent.

The gas mixtures used for calibration of the electrodes, with the exception of an assumed concentration of 99.5 per cent oxygen, were analyzed with the Scholander micro-analyzer (Scholander, 1947); the gas tensions were calculated from the equation:

\[
P_{\text{gas}} = F_{\text{gas}} \times (B - W),
\]

where \(B\) was the barometric pressure and \(W\) the saturated water vapour pressure at the temperature of the electrode.

**Calculations.**

Respiratory gas volumes were measured by summation of the expired tidal volumes from the spirometer records (fig. 2) and all gas volumes were corrected from spirometer or ambient temperature to BTPS.

Physiological deadspace was calculated from the following form of the Bohr equation:

\[
V_D = V_T \frac{(P_{a\text{CO}_2} - P_{e\text{CO}_2})}{P_{a\text{CO}_2}} - V_{Dapp}.
\]

Apparatus deadspace (\(V_{Dapp}\)) was determined by water displacement and excludes the endotracheal tube volume and in Study II the mouthpiece volume.

Alveolar oxygen tension was calculated from the equation:

\[
P_{a\text{O}_2} = P_{i\text{O}_2} - P_{a\text{CO}_2} \text{ (assuming } P_{a\text{CO}_2} = P_{e\text{CO}_2})
\]

This equation may be used since the effect of changes in respiratory exchange ratio become minimized at high oxygen concentrations. The effect of the uptake of halothane was not allowed for, but concentrations below 2 per cent were used and even if all of this was taken up in the lungs the alveolar oxygen tension would increase by only some 14 mm Hg; the effect on the measured physiological shunt would therefore be negligible.

Alveolar to arterial oxygen tension difference (\(A-a P_{\text{O}_2}\) Diff.) was calculated by subtraction.

Physiological shunt may be calculated from the following equation (Finley et al., 1960):

\[
\frac{Q_s}{Q} \times 100 = \frac{(P_{a\text{O}_2} - P_{a\text{O}_2}) 0.0031}{(P_{a\text{O}_2} - P_{a\text{O}_2}) 0.0031 + a-v O_2 \text{ diff.}}
\]

However, the mixed venous blood oxygen content (\(C_{vo2}\)) was not measured in the present study and the physiological shunt was calculated at two levels of arteriovenous oxygen content difference (3.5 and 5 vols. per cent). The calculation of the percentage shunt was then simplified to:

\[
\frac{Q_s}{Q} \times 100 = \frac{(P_{a\text{O}_2} - P_{a\text{O}_2}) 0.0031}{(P_{a\text{O}_2} - P_{a\text{O}_2}) 0.0031 + a-v O_2 \text{ diff.}}
\]

where 0.0031 is the solubility coefficient of oxygen in whole blood.

**RESULTS**

**Study I.**

The detailed results from the thirteen patients studied pre-operatively and again during surgery are shown in table II. The interval between the measurements ranged from 30 to 70 min (mean = 51 min).
The means and standard deviations of these results are shown in Table III, together with the P value for the significance of the differences between the two groups by Student's "t" test.

Table III shows that there was a highly significant fall in alveolar ventilation (VA) from 5.37 l./min pre-operatively, to 3.19 l./min during halothane anaesthesia, and this was accompanied by a rise in PaO₂ from 40 to 51 mm Hg indicating that the metabolic rate was not depressed proportionately. The decrease in standard bicarbonate (from 22.9 to 22.4 m.equiv/l.) was not statistically significant, and the ratio of physiological deadspace to tidal volume was not significantly changed (from 0.31 to 0.32). The most interesting finding was that no significant overall change in A-a Po₂ diff. occurred and therefore the calculated physiological shunt remained constant.

However, for patients Nos. 1 and 7 in Table II
TABLE III  
Study I. Means and standard deviation of the derived values from the data shown in Table II together with the P value for the significance of the differences between the pre-anaesthetic values and those during anaesthesia, obtained from paired Student's "t" tests.

<table>
<thead>
<tr>
<th></th>
<th>Pre-anesthetic</th>
<th>Anesthetized</th>
<th>P value for difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Paw, HCO₃⁻</td>
<td>40</td>
<td>3.6</td>
<td>51.3</td>
</tr>
<tr>
<td></td>
<td>(mm Hg)</td>
<td>(m.equiv/l.)</td>
<td>(mm Hg)</td>
</tr>
<tr>
<td>VA</td>
<td>22.9</td>
<td>1.0</td>
<td>22.4</td>
</tr>
<tr>
<td></td>
<td>(L/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VT</td>
<td>0.31</td>
<td>0.14</td>
<td>0.32</td>
</tr>
<tr>
<td></td>
<td>(b.p.m.)</td>
<td></td>
<td>(b.p.m.)</td>
</tr>
<tr>
<td>A-a Po₂</td>
<td>142</td>
<td>100</td>
<td>154</td>
</tr>
<tr>
<td></td>
<td>(mm Hg)</td>
<td></td>
<td>(mm Hg)</td>
</tr>
<tr>
<td>% Shunt a-v O₂</td>
<td>3.5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>content diff.</td>
<td>11</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>7</td>
<td>9</td>
</tr>
</tbody>
</table>

TABLE IV  
Study II. Details of blood and gas analysis and physiological deadspace in patients intubated (I) and with the orotracheal mouthpiece (M). The last column shows the difference between the values; below is shown the mean and standard deviations. The significance of the difference between the two measurements of physiological deadspace was obtained from Student's "t" test and the P value is shown.

<table>
<thead>
<tr>
<th>Patient, age</th>
<th>VD App (ml)</th>
<th>f (per min)</th>
<th>VT (BTPS, ml)</th>
<th>P₈₅₀₂ (mm Hg)</th>
<th>P₈₅₀₂ (mm Hg)</th>
<th>VD-VD App (ml)</th>
<th>VD difference (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (60)</td>
<td>I 33</td>
<td>25.5</td>
<td>323</td>
<td>21.3</td>
<td>52</td>
<td>158</td>
<td>45</td>
</tr>
<tr>
<td>2 (21)</td>
<td>I 33</td>
<td>18</td>
<td>370</td>
<td>32.8</td>
<td>49</td>
<td>89</td>
<td>44</td>
</tr>
<tr>
<td>3 (60)</td>
<td>I 33</td>
<td>12</td>
<td>254</td>
<td>28.1</td>
<td>60.5</td>
<td>103</td>
<td>23</td>
</tr>
<tr>
<td>4 (20)</td>
<td>I 33</td>
<td>20</td>
<td>228</td>
<td>34.3</td>
<td>57</td>
<td>59</td>
<td>23</td>
</tr>
<tr>
<td>5 (48)</td>
<td>I 33</td>
<td>20</td>
<td>274</td>
<td>34.3</td>
<td>57</td>
<td>59</td>
<td>23</td>
</tr>
<tr>
<td>6 (49)</td>
<td>I 33</td>
<td>33</td>
<td>349</td>
<td>15.1</td>
<td>33</td>
<td>140</td>
<td>10</td>
</tr>
<tr>
<td>7 (24)</td>
<td>I 33</td>
<td>22</td>
<td>314</td>
<td>19.4</td>
<td>32</td>
<td>88</td>
<td>20</td>
</tr>
<tr>
<td>8 (48)</td>
<td>I 33</td>
<td>24</td>
<td>305</td>
<td>18.6</td>
<td>32</td>
<td>108</td>
<td>31</td>
</tr>
<tr>
<td>9 (44)</td>
<td>I 33</td>
<td>14</td>
<td>331</td>
<td>24.2</td>
<td>40</td>
<td>99</td>
<td>31</td>
</tr>
<tr>
<td>10 (21)</td>
<td>I 33</td>
<td>26</td>
<td>250</td>
<td>24.2</td>
<td>40</td>
<td>99</td>
<td>31</td>
</tr>
<tr>
<td>Mean</td>
<td>I 22.3</td>
<td>±6.2</td>
<td>298</td>
<td>±65</td>
<td>±37</td>
<td>±14</td>
<td>27</td>
</tr>
<tr>
<td>SD</td>
<td>M 21.1</td>
<td>±6.9</td>
<td>312</td>
<td>±75</td>
<td>126</td>
<td>±34</td>
<td>14</td>
</tr>
</tbody>
</table>

\[ t=6; \ P=0.001 \]
a considerable increase in A-a Po\textsubscript{2} diff. occurred during anaesthesia; furthermore, in these patients and also in Nos. 12 and 13 large A-a Po\textsubscript{2} differences were present pre-operatively. These four patients all showed clinical evidence of respiratory disease.

For patients Nos. 1, 13 and 7 further measurements were performed postoperatively on the first day. The physiological shunt (assuming an a-v O\textsubscript{2} diff. to be 3.5 ml/100 ml blood) was found to be 21 per cent in No. 1 and 20 per cent in No. 7, a return below and to the pre-operative levels respectively.

However, in patient No. 13, who showed no change during surgery, the shunt was found to be 30 per cent on the first postoperative day. This coincided with a right lower lobe collapse (fig. 3); on the third day the shunt was 22 per cent and despite physiotherapy the collapse was evident. On the 13th postoperative day the physiological shunt was 14 per cent and the chest X-ray was normal.

**Study II.**

The detailed values for the ten patients studied are shown in table IV. The last column shows the difference between the physiological deadspaces calculated when intubated, subtracted from those calculated when breathing through the mouthpiece. The mean difference, 27 ml, was highly significant (SD 14; P<0.001). The rate and depth of respiration was not significantly changed.

**DISCUSSION**

From Study II it was concluded that intubation reduced the physiological deadspace by a mean of 27 ml in comparison with mouth-breathing with the nose closed. Previous studies have measured the changes in anatomical deadspace with intubation, either by water displacement in cadavers (Nunn, Campbell and Peckett, 1959) or by Fowler's method (Thornton, 1960). In both groups a reduction of approximately 70 ml resulted from intubation. The physiological deadspace is a functional measurement, and the volume changes found in the present studies are a more realistic indication of the effective reduction of deadspace with intubation.

The physiological shunt refers to the quantity of venous blood that has been added to the arterial blood emerging from the lungs. The sources of this venous admixture when breathing 100 per cent oxygen are: firstly, the anatomical shunt of venous blood draining from the bronchial, pleural or Thebesian veins, or from direct arteriovenous anastomoses; secondly, the effective
Study I; patient No. 13. Chest radiographs taken 6 days pre-operatively and on the 1st, 3rd, and 13th postoperative days. These correspond to shunt measurements of 18, 30, 22 and 14 per cent respectively. Right lower lobe collapse is evident on the 1st and 3rd postoperative days.
shunt caused by unoxygenated blood from non-ventilated alveoli. In young healthy adults these factors amount to 1–2 per cent of the cardiac output (Cole and Bishop, 1963). But this probably increases with age (Raine and Bishop, 1963; Marshall and Millar, 1965) and in hospitalized patients (Berggren, 1942) and the pre-operative values found in this study reflect these factors in addition to the possible influence of premedication.

The results from Study I were unexpected in view of recent reports that shunting progressively increased during anaesthesia (Bendixen, Hedley-Whyte and Laver, 1963), although one other group has not confirmed these findings (Askrog et al., 1964). While it is difficult to reconcile these two reports it is worthy of note that neither group has measured the A-a Po₂ diff. pre-operatively; it is difficult to say that any subsequent determination during anaesthesia has become abnormal without this essential measurement, particularly as manipulations necessary during induction and intubation are likely to modify considerably the state of expansion of the lungs. In the present studies serial measurements were not made, and it is therefore not possible to affirm whether an initially reduced A-a Po₂ diff. was increasing at the time of measurement. However, it is possible to state that increased physiological shunting did not occur under the clinical conditions obtaining in this study, except in three instances.

In order to discuss these results it is necessary to examine more closely the assumptions made in the calculation of the shunt. The arteriovenous oxygen content difference may be calculated from the equation:

\[ a - \bar{v} \text{ diff.} = \frac{\bar{V}_o_2}{Q} \]

Assuming that patients anaesthetized with halo-

![Graph](https://academic.oup.com/bja/article-abstract/38/12/912/277195)

**Fig. 4**

On the left the relationships between cardiac output and arteriovenous oxygen content are plotted at three levels of oxygen uptake (\(V_o_2\)). On the right the relationships between the arteriovenous oxygen content and the physiological shunt percentage are plotted at five different values of the alveolar-arterial oxygen tension difference (A-a Po₂ diff.). The shaded area covers the values of the a-Vo₂ content difference (3.5 and 5 vols. per cent) used for the calculations in the present studies. (See text.)
thane after premedication and induction with thiopentone have an oxygen requirement approximately 85 per cent of basal (Nunn and Matthews, 1959) then the normal range of oxygen uptakes would be covered by volumes of 70, 100 and 130 ml/min/sq.m. If the a–v O₂ differences are then calculated at different cardiac outputs this may be plotted as in the lefthand side of figure 4. Assuming that oxygen concentrations approaching 100 per cent were breathed, the shunt may be calculated according to the equation described before based on the A-a Po₃ diff., and again lines may be plotted of the changes in physiological shunts that result from changes in the a–v O₂ content differences when the A-a Po₃ diff. remains constant; these are shown in the righthand side of figure 4 and also shown is a shaded area corresponding to the ranges of a–v O₂ content difference used in this study. The normal a–v O₂ content difference in adults at rest varies from 3 to 5.8 with a mean of 4.4 vols./100 ml (Cournand et al., 1945); during anaesthesia with halothane it is probable that the oxygen consumption and the cardiac output both fall to the same extent (Severinghaus and Cullen, 1958) and therefore the a–v O₂ content difference would remain unchanged. There is, however, considerable disagreement as to the effect of halothane on cardiac output (Payne, Gardner and Verner, 1959).

An excessive fall in cardiac output would result in an increase in a–v O₂ content difference, since the mixed venous blood would be more desaturated and thus the same percentage shunt would correspond to a higher A-a Po₃ diff. Thus from figure 4 a disproportionate fall of cardiac output of 50 per cent would result in an increased A-a Po₃ diff. of approximately 100 mm Hg if the true shunt percentage remained constant; if the a–v O₂ content difference is assumed to remain constant then the shunt will appear to have increased by approximately 6 per cent.

These considerations may explain the increased shunt found in patient No. 1 (table II) in whom the systolic blood pressure fell from 190 mm Hg pre-operatively to 70 mm Hg during the measurements. However, patient No. 13 showed a similar fall in arterial pressure with little change in A-a Po₃ diff. The final answer to these questions must await further study. No evidence for the occurrence of atelectasis during anaesthesia was found.

The physiological deadspace: tidal volume ratio was unchanged during halothane anaesthesia but the interpretation of this finding is complicated by intubation, the inhalation of 100 per cent oxygen, and by narcotic, vagolytic and barbiturate drugs, all or any of which may produce effects on the deadspace. In the case of atropine the anatomical deadspace has been shown to increase by 30–40 ml (Severinghaus and Stupfel, 1955). In the present investigation the net effect of all these changes left the ratio unchanged.

APPENDIX

The abbreviations used in this paper are in accordance with the recommendations of the committee on "The standardization of definitions and symbols in respiratory physiology" (Chairman: Pappenheimer, J. R.), Fed. Proc., 1950, 602.

\[ \begin{align*}
\text{Pi} & = \text{gas tension in inspired gas} \\
\text{Pf} & = \text{gas tension in mixed expired gas} \\
\text{Pa} & = \text{arterial gas tension} \\
\text{VA} & = \text{alveolar ventilation (l./min)} \\
\text{VT} & = \text{tidal volume} \\
\text{VDP} & = \text{physiological deadspace} \\
\text{VD_APP} & = \text{apparatus deadspace} \\
\text{Q} & = \text{cardiac output (vol/min)} \\
\text{QS} & = \text{physiological shunt (vol/min)} \\
\text{BTPS} & = \text{gas volume at body temp., pressure and saturated} \\
\text{A-a Po₃ diff.} & = \text{alveolar to arterial oxygen tension difference (mm Hg)} \\
\text{a–v O₂ diff.} & = \text{arterial to venous oxygen content difference (vols. %)} \\
\text{Cao₂} & = \text{arterial oxygen content per 100 ml blood} \\
\text{Cco₂} & = \text{oxygen content per 100 ml end-pulmonary capillary blood} \\
\text{Cvo₂} & = \text{oxygen content per 100 ml mixed venous blood} \\
\end{align*} \]

ACKNOWLEDGEMENTS

It is a pleasure to acknowledge the co-operation of anaesthetic, nursing and orthopaedic staff of the United Cambridge Hospitals.

I am grateful to Dr. I. A. Silver of the Sub-department of Veterinary Anatomy, University of Cambridge, for supplying the oxygen micro-electrodes, and to Dr. R. A. Millar of the Department of Anaesthetics, United Cambridge Hospitals, for his advice and criticism.

This work was supported by the Elmore Research Fund, University of Cambridge and the Board of Governors, United Cambridge Hospitals.

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SHUNT PHYSIOLOGIQUE ET ESPACE MORT
PENDANT LA RESPIRATION SPONTANEE
D'HALOTHANE: ANESTHESIE A L'OXYGENE
ET INFLUENCE DE L'INTUBATION SUR
L'ESPACE MORT PHYSIOLOGIQUE

SOMMAIRE
Chez 13 malades, on a mesure le rapport entre l'espace
mort physiologique et l'air courant ainsi que la dif-
fERENCE DE TENSION DE L'OXYGENE ALVEOLAIRE ET ARTERIEL,
immediatement avant et pendant l'anesthesie par
l'halothane dans l'oxygene apres 30-70 respirations
spontanees. Les chiffres moyens n'ont pas change et
on a conclu que le shunt physiologique etait egalement
inchange sauf dans deux cas que l'on presente. Dans
une autre serie de 10 malades, on a mesure une
reduction moyenne de l'espace mort physiologique de
27 ml, resultat de l'intubation intra-trachea.

PHYSIOLOGISCHE SHUNT-BILDUNG UND
totraum bei spontanatmung wahrend
der halothan-sauerstoff-narkose
und der einfluss der intubation auf
den physiologischen totraum

ZUSAMMENFASSUNG
Bei 13 Patienten wurde das Verhalten zwischen dem
physiologischen Totraum und dem Atemzugvolumen
und die Differenz des Sauerstoffdruckes zwischen
Alveole und Arterie unmittelbar vor und wahrend der
Halothan-Sauerstoff-Narkose nach 30- bis 70-minu-
tiger Spontanatmung gemessen. Die Mittelwerte
andersten sich nicht; es wurde daraus geschlossen,
da der physiologische Shunt sich ebenfalls nicht verander-
nete, abgesehen von zwei Fallen, die besprochen werden.
In einer weiteren Serie mit 10 Patienten wurde als
Ergebnis der endotrachealen Intubation eine durch-
schrittliche Vermindерung des physiologischen Tot-
raumes um 27 ml gemessen.