DEADSPACE AND THE SINGLE BREATH TEST FOR CARBON DIOXIDE DURING ANAESTHESIA AND ARTIFICIAL VENTILATION

Effects of tidal volume and frequency of respiration

R. FLETCHER AND B. JONSON

SUMMARY

Using the single breath test for carbon dioxide (SBT-CO₂), the components of physiological deadspace were investigated during anaesthesia with IPPV in 58 patients. A square-wave inspiratory flow and an end-inspiratory pause (25% and 10% of cycle time, respectively) were used. At tidal volumes of 0.45 litre (f = 17 b.p.m.), and 0.75 litre (f = 9 b.p.m.), median values for Vb²⁰ were 0.44 and 0.31. Increasing Vr and decreasing f did not change airway deadspace (Vb²⁰) so that the fraction Vb²⁰/Vr was decreased (P < 0.001). The alveolar deadspace fraction, Vb²⁰/Vr²⁰, was decreased in 93% of patients (P < 0.001). These improvements with increasing Vr can be attributed to beneficial effects on gas distribution and diffusion time. Patients with large alveolar deadspaces had steeply sloping SBT-CO₂ phase III, and increased expiratory time constants of the respiratory system. The median arterial-end-tidal Pco₂ difference, (Paco₂ - Pe'CO₂), was 0.6 kPa at small and 0.3 kPa at large tidal volumes (P < 0.001). Three patients had zero and four had negative (Paco₂ - Pe'CO₂) values at large tidal volumes. When phase III slopes steeply, negative (Paco₂ - Pe'CO₂) values may be observed in the presence of alveolar deadspace.

Physiological deadspace (Vdphys) during anaesthesia with intermittent positive pressure ventilation (IPPV) is increased when compared with Vdphys during spontaneous breathing in the awake state (Campbell, Nunn and Peckett, 1958). In particular, it is increased in the elderly, in the presence of minor degrees of respiratory disease (Cooper, 1967), and during arterial hypotension (Eckenhoff et al., 1963). It is affected by ventilatory pattern (Watson, 1962; Lyager, 1970; Baker, Collis and Cowie, 1977) and may, therefore, be a useful measure of ventilatory efficiency. Analysis of its components, airway deadspace (Vdphys) and alveolar deadspace (Vdphys), may provide information about disturbances of gas exchange (Nunn and Hill, 1960; Olsson et al., 1980; Fletcher et al., 1981).

A unified theoretical concept of Vdphys, its constituents, and factors influencing them has been presented previously (Fletcher et al., 1981). The importance of not only end-tidal Paco₂ (Pe'CO₂), but also of information from the entire tracing over a breath of expired Paco₂ (PeCO₂, or fraction, FECO₂) against tidal volume, the single breath test for carbon dioxide (SBT-CO₂) (Comroe, 1962) was emphasized (fig. 1). The shape of this tracing allows some conclusions to be drawn about the nature of alveolar deadspace, and the distribution of ventilation/perfusion (V/Q) ratios.

Cooper (1967) showed that the deadspace/tidal volume ratio (Vdphys/Vt), was decreased significantly when ventilatory frequency (f) was decreased and Vt increased. The aim of the present study was to analyse changes in deadspace and SBT-CO₂ brought about by this manoeuvre.

PATIENTS AND METHODS

Fifty-eight patients (median age 64 yr, range 13–84 yr), undergoing elective major orthopaedic, neuro- or general surgery were studied. The trachea was intubated in all patients and the lungs ventilated with nitrous oxide 65% in oxygen. Neuromuscular blockade was produced and fentanyl or pethidine administered i.v. All patients were in the supine position. A radial artery catheter allowed withdrawal of arterial blood. The study was approved by the local Ethics Committee.

No patient had incapacitating pulmonary disease. Nine had suspected or confirmed histories of asthma, emphysema, pulmonary emboli, cardiac failure, recent myocardial infarction or repeated pneumonia, and a further 12 had minor changes seen on chest x-ray.

Arterial blood-gas tensions were analysed (Instrumentation Laboratory 413) and were corrected...
to body temperature (nasopharyngeal probe (Ellab)). The analyser was checked on alternate days by tonometry.

A Servo Ventilator 900 B delivering a square wave flow was used with a CO₂ Analyzer 930 (Olsson et al., 1980) and a lung mechanics calculator (Jonson et al., 1975). The deadspace between the tracheal tube and the cuvette of the CO₂ Analyzer was 12 ml. A Bennet humidifier and the ventilator tubing created a compressible volume of 1900 ml. The tubing was checked to exclude possible leaks.

The carbon dioxide signal and the expiratory flow signal from the ventilator were transmitted to a computer (Digital Equipment PDP8) via a tape recorder (Philips Minilog 4). The computer presented SBT-CO₂ in two diagrams with expired volume on the ordinate and FE CO₂ or expired carbon dioxide volume on the abscissa. Expiratory volume was obtained by integration of flow. The volume of carbon dioxide was obtained by integration of (flow × FE CO₂). The sampling frequency was 100 Hz. Daily tape recordings were made of calibration signals representing flow of nitrous oxide in oxygen measured against a gas meter, and carbon dioxide concentration of a known gas.

**Corrections.** Values for carbon dioxide concentrations and volumes were corrected using the method of Fletcher and colleagues (1983). Corrections were
made for: rebreathing in the Y-piece, the effects of nitrous oxide, analyser delay, alinearity of carbon dioxide analysis, variations in temperature and water content of the expired gas in the tubings, the effects of compressed gas in the tubings, and barometric pressure.

The shape of SBT-CO$_2$. Gas compression and apparatus deadspace influence phase I to a degree that is difficult to assess in routine IPPV. Delineation of $V_D^{aw}$ is difficult at the bedside. The onset of phase II is distinct and this point has been suggested as a point of reference on the volume axis (fig. 2, left) (Olsson et al., 1980; Fletcher et al., 1981). Volumes expressed after this point contain carbon dioxide and are referred to as effective volumes. The sum of phases II and III is called the effective tidal volume ($V_T^e$). One measure of the shape of SBT-CO$_2$ is the slope of phase III, measured between 0.2 and 0.4 litre effective volume (corrected for body height). Because hyperventilation to a low $P_E CO_2$ decreases the slope of phase III, the increase in $P_E CO_2$ per litre was divided by the mean $P_E CO_2$ between 0.2 and 0.4 litre effective volume. This gave a measure that related more closely to the spread of $V/Q$ ratios that it represents. This variable, "relative slope", has the units litre$^{-1}$.

In figure 2 (right), the breath eliminates a volume of carbon dioxide ($V_{TCO_2}$) given by the area X under the tracing. This can be compared with that volume which would be eliminated if the whole $V_T^{eff}$ had $P_E CO_2$ equal to $P_E CO_2$. The ratio $V_{TCO_2}/(P_E CO_2 \times V_T^{eff})$, that is area X/area ABCDA, is designated efficiency (Fletcher et al., 1981). If SBT-CO$_2$ approaches a rectangular shape, efficiency comes close to 1.0; a ramp form would have an efficiency of 0.5. Efficiency was calculated for the total effective volume (efficiency$_{VT^e}$) and for the initial 0.4 litre of the effective volume (efficiency$_{0.4}$). The volume of 0.4 litre was corrected for body height thus:

$$\text{corrected volume} = \frac{\text{height (m)}}{1.75} \times 0.4$$

The relationship between arterial and alveolar $P_CO_2$ was expressed as the fraction $P_E CO_2/P_A CO_2$. It was measured at effective volumes of 0.2, 0.3 and 0.4 litre.

The subdivisions of physiological deadspace are represented graphically in figure 3. Area X = tidal elimination of carbon dioxide, that is, effective ventilation; Area Y represents wasted ventilation as a result of $V_D^{aw}$, and Area Z represents wasted ventilation as a result of $V_D^{aw}$.

**Airway deadspace** was estimated by the method of Langley and colleagues (1975) (fig. 1). In eight patients where the transition between phases II and III was unclear, similar extrapolation was attempted at both tidal volumes.

Physiological deadspace, $V_D^{phys}$, was calculated according to

$$V_D^{phys} = V_T \times (1 - \frac{P_E CO_2}{P_A CO_2})$$

Mixed expired $P_CO_2$ ($P_E CO_2$) was calculated from SBT-CO$_2$ as $V_{TCO_2} \times (\text{barometric pressure (kPa)})/V_T(\text{ml})$. $V_D^{av}$ was obtained from:

$$V_D^{av} = V_D^{phys} - V_D^{aw}$$

**Venous admixture.** A rough estimate of venous admixture or right to left shunt fraction ($Q_s/Q_t$) was obtained from inspired $F_O_2$ and $P_A CO_2$ (Nunn, 1977). Assuming a mixed venous--arterial $P_CO_2$ difference ($P_V CO_2 - P_A CO_2$) of 1 kPa, pulmonary end-capillary $P_CO_2$ ($P_c CO_2$) was calculated from the equation given by Müritz (1961):

$$P_c CO_2 = P_A CO_2 - (P_V CO_2 - P_A CO_2) \times \frac{Q_s/Q_t}{1 - Q_s/Q_t}$$

$P_c CO_2$ and $P_A CO_2$ were used to obtain an approximate estimate of the extent to which shunt contributed to $V_D^{phys}/V_T$.

**Time constants ($R_C$** for the respiratory system were calculated as the product of expiratory resistance, $R_e$ (cm H$_2$O litre$^{-1}$ s) and compliance of the respiratory system, $C_r$ (litre cm H$_2$O$^{-1}$) as obtained digitally from the lung mechanics unit.

**Procedure**

During the anaesthetic, ventilation was volume controlled with constant inspiratory flow and a fixed
TABLE I. Typical ventilator settings at small and large Vt. The relative duration of inspiration and end-inspiratory pause are unchanged (25% and 10% of cycle time, respectively).

<table>
<thead>
<tr>
<th></th>
<th>Frequency (b.p.m.)</th>
<th>Total volume (litre)</th>
<th>Minute ventilation (litre)</th>
<th>Respiratory cycle (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small tidal volumes</td>
<td>17</td>
<td>0.45</td>
<td>8.0</td>
<td>0.9</td>
</tr>
<tr>
<td>Large tidal volumes</td>
<td>9</td>
<td>0.74</td>
<td>7.0</td>
<td>1.7</td>
</tr>
</tbody>
</table>

TABLE II. Results of these variables are not normally distributed. The figures given are the median and the 5th and 95th percentiles. The differences between the pairs of observations at small and large Vt, except for \( (P_{acO_2} - P_eCO_2) \), were normally distributed and could therefore be tested with Student’s t test. The difference for \( (P_{acO_2} - P_eCO_2) \) was tested using the Wilcoxon test. \( n = 58 \) (for efficiency 0.4 at small Vt, \( n = 50 \)).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Small tidal volumes</th>
<th>Medium</th>
<th>5th</th>
<th>95th</th>
<th>Large tidal volumes</th>
<th>Medium</th>
<th>5th</th>
<th>95th</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT (ml)</td>
<td>450</td>
<td>373</td>
<td>546</td>
<td></td>
<td>743</td>
<td>591</td>
<td>903</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vb**(ml)</td>
<td>85</td>
<td>58</td>
<td>127</td>
<td></td>
<td>83</td>
<td>55</td>
<td>127</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vb**(ml)</td>
<td>105</td>
<td>60</td>
<td>197</td>
<td></td>
<td>143</td>
<td>83</td>
<td>283</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>VT**(ml)</td>
<td>195</td>
<td>138</td>
<td>300</td>
<td></td>
<td>229</td>
<td>155</td>
<td>382</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Vb**(VT)</td>
<td>0.19</td>
<td>0.13</td>
<td>0.26</td>
<td></td>
<td>0.11</td>
<td>0.08</td>
<td>0.16</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Vb**(VT)</td>
<td>0.29</td>
<td>0.17</td>
<td>0.49</td>
<td></td>
<td>0.22</td>
<td>0.13</td>
<td>0.43</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>VT**(VT)</td>
<td>0.44</td>
<td>0.32</td>
<td>0.58</td>
<td></td>
<td>0.31</td>
<td>0.22</td>
<td>0.49</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>(Paco2 - Pe'CO2) (kPa)</td>
<td>0.60</td>
<td>0.20</td>
<td>1.40</td>
<td></td>
<td>0.30</td>
<td>-0.10</td>
<td>1.10</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Efficiency 0.4</td>
<td>0.79</td>
<td>0.72</td>
<td>0.87</td>
<td></td>
<td>0.82</td>
<td>0.74</td>
<td>0.90</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Efficiency Vt eff</td>
<td>0.80</td>
<td>0.72</td>
<td>0.85</td>
<td></td>
<td>0.83</td>
<td>0.72</td>
<td>0.88</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Relative slope (litre⁻¹)</td>
<td>0.58</td>
<td>0.25</td>
<td>1.20</td>
<td></td>
<td>0.45</td>
<td>0.13</td>
<td>1.00</td>
<td>&lt;0.001</td>
<td></td>
</tr>
</tbody>
</table>

The cycle: 25% inspiration, 10% end-inspiratory pause (EIP), and 65% expiration. Positive end-expiratory pressure was not used. Minute ventilation and respiratory frequency were set according to one of the alternatives in table I so as to give \( P_{ecO_2} \) values in the range 4.0–4.5 kPa. At steady state, as judged by stable \( P_{e'}CO_2 \) and carbon dioxide minute production \( (\dot{V}CO_2) \), 5 ml of arterial blood was slowly withdrawn into a heparinized glass syringe. At the same time the values on the digital displays of the carbon dioxide analyser and lung mechanics calculator were noted, and \( \dot{V}e \) and \( P_{ecO_2} \) signals for several breaths were recorded on the tape recorder. A new steady state at a different frequency was then achieved (table I), after a change in minute ventilation which was intended to minimize changes in alveolar ventilation.

Statistical analysis of the results was made using Student’s t test for paired observations, the Wilcoxon test, and linear regressions.

RESULTS

Data about tidal volume and its subdivisions are given in table II. Median airway deadspace was about 85 ml at both settings. \( Vb**(VT) \) was 0.19 and 0.11 at small and large VT, respectively.

Median \( Vb**(VT) \) was 105 ml at small tidal volumes and increased by 38 ml at large tidal volumes \( (P < 0.001) \). Thus physiological deadspace was greater at large VT. The median alveolar deadspace fraction, \( Vb**(VT) \), was 0.29 at small tidal volumes and decreased significantly to 0.22 at large.

Relative slope was significantly less at large tidal volumes. Efficiency \( Vt eff \) and efficiency 0.4 were significantly greater at large tidal volumes.

The \( (Paco2 - P_{ecO2}) \) difference was smaller at
large tidal volumes (table II; fig. 4). Four patients (7%) had negative and three had zero differences at large tidal volumes. However, alveolar \( P_{CO_2} \), as judged from \( PE_{CO_2} \) after expiration of 0.2–0.4 litre of the effective volume, was slightly closer to arterial at small VT (table III; fig. 5). Six patients with small \( VD^A/VT^A \) values (mean 0.19 at small VT) deviated from the above pattern: all had virtually horizontal phase III (mean relative slope 0.25) and showed little or no change in \( VD^A/VT^A \) or \((P_{CO_2} - PE_{CO_2})\) when the ventilator setting was changed (fig. 4). Their mean age was 49 yr.

Table IV gives some variables describing lung ventilation. As the inspiratory phase was always 25% of cycle time, the lower ventilation at large VT resulted in a lower inspiratory flow. Large tidal volumes gave greater peak and pause airway pressures and greater \( C_a \). The same mean \( P_{CO_2} \) was achieved at both ventilator settings (4.2 kPa).

Airway deadspace was positively correlated to body height, surface area and \( C_a \) and was greatest in patients with low airway pressures (table V).

Alveolar deadspace was not correlated to body size. The alveolar deadspace fraction (table VI) was positively correlated to age, \( RC^- \), late expiratory

### Table IV. Ventilatory variables. Figures given are the median, and the 5th and 95th percentiles. Frequency, minute ventilation and \( P_{CO_2} \) were normally distributed: the figures here are mean ± 2SD, closely corresponding to the range of 95% of observations. (n = 58)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Small tidal volumes</th>
<th>Large tidal volumes</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Median or mean</td>
<td>5th</td>
<td>95th</td>
</tr>
<tr>
<td>Frequency (b.p.m.)</td>
<td>17.3</td>
<td>14.2</td>
<td>20.4</td>
</tr>
<tr>
<td>Minute ventilation (litre)</td>
<td>7.9</td>
<td>5.4</td>
<td>10.4</td>
</tr>
<tr>
<td>( P_{CO_2} ) (kPa)</td>
<td>4.2</td>
<td>3.2</td>
<td>5.1</td>
</tr>
<tr>
<td>Peak pressure (cm H₂O)</td>
<td>18</td>
<td>13</td>
<td>26</td>
</tr>
<tr>
<td>Pause pressure (cm H₂O)</td>
<td>11</td>
<td>8</td>
<td>16</td>
</tr>
<tr>
<td>( C_a ) (ml cm H₂O⁻¹)</td>
<td>64</td>
<td>39</td>
<td>123</td>
</tr>
</tbody>
</table>
TABLE V. Correlation between airway deadspace (y) at small tidal volumes and height, pause pressure and \( C_{\text{av}} \) (x) according to the formula \( y = mx + b \). \( RSD \) = residual standard deviation. *(P<0.05); **(P<0.01). (n=58)

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Height (m)</th>
<th>Surface area (m(^2))</th>
<th>Pause pressure (cm H(_2)O)</th>
<th>( C_{\text{av}} ) (ml cm H(_2)O(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>( m )</td>
<td>123</td>
<td>42.1</td>
<td>-3.39</td>
<td>0.37</td>
</tr>
<tr>
<td>( b )</td>
<td>-122</td>
<td>12.8</td>
<td>125</td>
<td>63</td>
</tr>
<tr>
<td>RSD</td>
<td>19</td>
<td>21</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>( r )</td>
<td>0.54**</td>
<td>0.37**</td>
<td>-0.35**</td>
<td>0.39**</td>
</tr>
</tbody>
</table>

TABLE VI. Correlation between deadspace variables (y) and age, \( R_{C_{\text{av}}} \), late expiratory flow, and relative slope (s), according to the formula \( y = mx + b \). \*P<0.05; **P<0.01, respectively. (n=58)

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>Age (yr)</th>
<th>( R_{C_{\text{av}}} )</th>
<th>Late expiratory flow (litre(^{-1}))</th>
<th>Relative slope (litre(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_{\text{E,alv}}/V_{T,alv} ) Small ( V_T )</td>
<td>0.0025</td>
<td>0.112</td>
<td>1.67</td>
<td>0.22</td>
</tr>
<tr>
<td>( m )</td>
<td>0.16</td>
<td>0.18</td>
<td>0.24</td>
<td>0.17</td>
</tr>
<tr>
<td>( b )</td>
<td>0.09</td>
<td>0.09</td>
<td>0.08</td>
<td>0.06</td>
</tr>
<tr>
<td>RSD</td>
<td>0.41**</td>
<td>0.39**</td>
<td>0.46**</td>
<td>0.74**</td>
</tr>
<tr>
<td>( r )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( V_{\text{E,alv}}/V_{T,alv} ) Large ( V_T )</td>
<td>0.0019</td>
<td>0.13</td>
<td>n.s.</td>
<td>0.214</td>
</tr>
<tr>
<td>( m )</td>
<td>0.14</td>
<td>0.07</td>
<td>n.s.</td>
<td>0.15</td>
</tr>
<tr>
<td>( b )</td>
<td>0.09</td>
<td>0.09</td>
<td>n.s.</td>
<td>0.07</td>
</tr>
<tr>
<td>RSD</td>
<td>0.32*</td>
<td>0.42**</td>
<td>n.s.</td>
<td>0.64**</td>
</tr>
<tr>
<td>( r )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( V_{\text{E,alv}}/V_{T,alv} ) Relative slope Small ( V_T )</td>
<td>0.0026</td>
<td>0.099</td>
<td>1.40</td>
<td>0.18</td>
</tr>
<tr>
<td>( m )</td>
<td>0.29</td>
<td>0.33</td>
<td>0.39</td>
<td>0.33</td>
</tr>
<tr>
<td>( b )</td>
<td>0.07</td>
<td>0.08</td>
<td>0.08</td>
<td>0.06</td>
</tr>
<tr>
<td>RSD</td>
<td>0.48**</td>
<td>0.38**</td>
<td>0.43**</td>
<td>0.70**</td>
</tr>
<tr>
<td>( r )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( V_{\text{E,alv}}/V_{T,alv} ) Relative slope Large ( V_T )</td>
<td>0.0088</td>
<td>0.46</td>
<td>8.18</td>
<td>—</td>
</tr>
<tr>
<td>( m )</td>
<td>0.12</td>
<td>0.10</td>
<td>0.30</td>
<td>—</td>
</tr>
<tr>
<td>( b )</td>
<td>0.29</td>
<td>0.29</td>
<td>0.24</td>
<td>—</td>
</tr>
<tr>
<td>RSD</td>
<td>0.43**</td>
<td>0.47**</td>
<td>0.66**</td>
<td>—</td>
</tr>
<tr>
<td>( r )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

![FIG. 5. SBT-CO\(_2\) tracings constructed to show the typical differences found at the two different ventilator settings. Heavy line: \( V_T = 0.45, f = 17 \text{ b.p.m.} \); thin line: \( V_T = 0.75, f = 9 \text{ b.p.m.} \). The dotted line represents \( P_{a\text{CO}_2} \) or \( P_F\text{CO}_2 \). Increases in \( V_T \) and decreases in \( f \) decrease relative slope and \( P_{a\text{CO}_2} - P_F\text{CO}_2 \). The extra ventilation at large tidal volumes is preferentially distributed to dependent, well perfused lung regions so that \( V_{\text{E,alv}}/V_{T,alv} \) is decreased.](https://academic.oup.com/bja/article-abstract/56/2/109/326606)

![FIG. 6. The relationship between phase III slope (relative slope) and \( V_{\text{E,alv}}/V_{T,alv} \) at small tidal volumes. The broken lines indicate the 95% confidence limits for the observations (Snedecor, 1956). The three patients (empty circles) whose values lie above the upper confidence limit may be regarded as having some alveolar deadspace of non-sequential origin, such as is caused by pulmonary embolism.](https://academic.oup.com/bja/article-abstract/56/2/109/326606)

Flow (the flow persisting at the very end of expiration) and relative slope (fig. 6). It was not correlated to pause pressure, \( C_{\text{av}} \) or expiratory resistance. Relative slope was positively correlated to age, \( R_{C_{\text{av}}} \) and late expiratory flow.

The \( (P_{a\text{CO}_2} - P_F\text{CO}_2) \) difference was related to
DEADSPACE DURING ANAESTHESIA

2.0
1.5
1.0
0.5
0
0.65 0.70

0.75

0.80

0.85

9.70

Efficiency

FIG. 7. The relationship between efficiency $V_{Teff}$ and $(P_{aCO_2} - P_{eCO_2})$ at small tidal volumes. The three patients represented by empty circles probably have increased alveolar deadspace of non-sequential origins.

efficiency (fig. 7):

$(P_{aCO_2} - P_{eCO_2}) = 4.9 - 5.36 \times \text{efficiency } V_{T_{eff}}$

$RSD = 0.30$

$R = -0.64 (P < 0.01).$

FIG. 8. Left: The relationship between $(P_{aCO_2} - P_{eCO_2})/P_{aCO_2}$ and $V_{oalv}/V_{T_{alv}}$ at large tidal volumes. The large scatter is caused by individual variation in the relationship between phase III and $P_{aCO_2}$ (Fletcher et al., 1981). Right: At small $V_T$ there is a more normal distribution, the regression equation being $y = 0.13 + 1.14x$ $(r = 0.95).$ The broken lines indicate the 95% confidence limits for the observations.
The fraction \((F_{\text{CO}_2} - P_{\text{E}'}\text{CO}_2)/P_{\text{CO}_2}\) was related to \(V_{\text{D}^{\text{aw}}}/VT^{\text{aw}}\) (fig. 8).

Venous admixture contributed about 0.02 to \(V_{\text{D}^{\text{aw}}}/VT\) at both settings, the largest value being 0.05.

**DISCUSSION**

The purpose of this study was to analyse changes in deadspace and SBT-\text{CO}_2 during anaesthesia which included IPPV with a strictly controlled ventilatory pattern. In particular, we wished to study one factor on which opinions differ: the use of large or small tidal volumes in routine IPPV. Thus, the principal changes made in the settings of the ventilator were those in \(f\) and \(VT\). The two ventilatory patterns were characterized by a "square wave" or constant inspiratory flow at small tidal volumes/high frequency and large tidal volumes/low frequency.

Inspiration, pause and expiration were always 25, 10 and 65% of the cycle. This meant that the duration of each phase was increased at large \(VT\) (table I). To avoid repetition in this discussion, reference to small and large tidal volumes implies these associated changes.

Airway deadspace was the same at the two ventilator settings (table II). It was related to factors reflecting body size such as height, surface area and compliance, but not to factors associated with sequential emptying, such as phase III slope. \(V_{\text{D}^{\text{aw}}}^{\text{aw}}\) increases with distending pressure (Hedenstierna and Lundberg, 1975) and with increasing end-inspiratory lung volume (Shephard et al., 1957). As presented previously (Fletcher et al., 1981), low inspiratory flow, and a long end-inspiratory pause should decrease \(V_{\text{D}^{\text{aw}}}^{\text{aw}}\). These various influences obviously balanced each other when the setting was changed.

Estimates of \(V_{\text{D}^{\text{aw}}}^{\text{aw}}\) based on the use of \(P_{\text{E}'}\text{CO}_2\) (Hedenstierna, 1975; Hedenstierna and McCarthy, 1975) are wrong in concept (Fletcher et al., 1981). Therefore they should not be compared with the present findings, which agree roughly with those of Thornton (1960), and Hedenstierna and Lundberg (1975), and with those of Nunn and Hill (1960) when allowance is made for differences in sampling methods.

We divided \(V_{\text{D}^{\text{aw}}}^{\text{aw}}\) into one phase when no carbon dioxide was recorded (phase I) and a later mixing phase. The former was about 40 ml at both ventilator settings and no significant information was obtained by this subdivision.

Alveolar deadspace was significantly greater at large tidal volumes, but \(V_{\text{D}^{\text{aw}}}^{\text{aw}}/VT^{\text{aw}}\) was significantly smaller. This improvement is probably attributable to the availability of more time for gas distribution within and between units (Fletcher et al., 1981), and to a more even global distribution of inspired gas which occurs with increasing \(VT\) (Rehder, Sessler and Rodarte, 1977). The increased \(C_m\) at large \(VT\) may be the result of (i) greater chest wall compliance at larger tidal volumes (Grimby, Hedenstierna and Lofström, 1975) and (ii) lung areas with poor ventilation at small tidal volumes taking a larger share of the ventilation at large \(VT\), possibly by the opening up of almost closed units and a more efficient alveolar surfactant system at large \(VT\).

Six younger patients with small deadspaces showed no improvement in \(V_{\text{D}^{\text{aw}}}^{\text{aw}}/VT^{\text{aw}}\) or \((F_{\text{CO}_2} - P_{\text{E}'}\text{CO}_2)/P_{\text{CO}_2}\) at large tidal volumes. It may be speculated that they had, even at small \(VT\), good \(V/Q\) distribution, and that any further gains made in the distribution of ventilation at large tidal volumes were counteracted by the adverse effects on the pulmonary circulation of the increase in intrathoracic pressure.

Increases in airway pressure have been shown to increase deadspace (Folkow and Pappenheimer, 1955; Landmark et al., 1977), but in this study \(V_{\text{D}^{\text{aw}}}^{\text{aw}}/VT^{\text{aw}}\) was not correlated to airway peak or pause pressure. These pressures were possibly too low to cause a marked effect on the distribution of perfusion.

Alveolar deadspace can be demonstrated by one or both of the following properties of SBT-\text{CO}_2: the slope of phase III, and the relationship of phase III to \(P_{\text{CO}_2}\).

The slope of phase III. Patients with steep phase III slopes had large alveolar deadspaces (fig. 6). \(V_{\text{D}^{\text{aw}}}^{\text{aw}}/VT^{\text{aw}}\) and relative slope were decreased at large tidal volumes. This, together with the association between increased \(V_{\text{D}^{\text{aw}}}^{\text{aw}}\) and late expiratory flow, suggests that the major causes of alveolar deadspace were time-dependent ones. These are (i) between units \(V/Q\) mismatching secondary to increased and unevenly distributed \(R_{\text{Ca}}\), or (ii) within units \(V/Q\) mismatching secondary to defective alveolar mixing, or both (Fletcher et al., 1981). Temporal \(V/Q\) mismatching as a result of phasic variations in \(V\) and \(Q\) should be greater at large tidal volumes (Fletcher et al., 1981) and, therefore, seems not to be an important cause of \(V_{\text{D}^{\text{aw}}}^{\text{aw}}\). The apparent deadspace attributable to shunt was small and constant at the two settings.

The shape of phase III is also of interest: in the
awake state during spontaneous breathing, it is always convex upwards. Anaesthesia with IPPV produces a more uniform, almost linear phase III in most subjects (Fletcher, 1980), but in five of our subjects an upward concavity was seen. These five had increased $V_d/V_T$ (mean 0.53 at small tidal volumes). This appearance was probably caused by the flow pattern during passive expiration, which (i) may favour early emptying of low time constant, hyperventilated units with low $P_{ACO_2}$ and (ii) changes the time course of alveolar carbon dioxide evolution (Fletcher, 1980; Fletcher et al., 1981).

The relationship between $P_{E'CO_2}$ and $P_{ACO_2}$. At small tidal volumes ($P_{ACO_2} - P_{E'CO_2}$) was always positive (median 0.6 kPa). At large volumes it was almost always less (median 0.3 kPa) (figs 4 and 5), was negative in 7% of patients and zero in 5%. ($P_{ACO_2} - P_{E'CO_2}$) was thus strongly dependent upon $V_T$, which is to be expected as phase III almost always has a positive slope. This dependence upon $V_T$ has also been demonstrated in the awake state by Poppius and co-workers (1975), and during anaesthesia with IPPV by Takki, Aromaa and Kauste (1972). A tidal volume-dependent, negative ($P_{ACO_2} - P_{E'CO_2}$) difference was observed during exercise by Jones, Robertson and Kane (1979). The existence of negative arterial–end-tidal $CO_2$ gradients is understood if it is remembered that $P_{ACO_2}$ represents the temporal and spatial mean alveolar $PCO_2$ (Riley et al., 1946; Fletcher et al., 1981). The correlation between ($P_{ACO_2} - P_{E'CO_2}$) and efficiency is of practical interest since it allows a non-invasive estimate of $P_{ACO_2}$ which should be more accurate than $P_{E'CO_2}$ alone (Fletcher, 1980).

The $P_{ACO_2}$–phase III difference. Figure 5 shows that, although the $P_{ACO_2}$–phase III difference at given expired volumes was slightly greater at large $V_T$ (table III), the net effect of increasing $V_T$ was to decrease ($P_{ACO_2} - P_{E'CO_2}$) and $V_{D^{TV}}/V_T$.

The decrease in ($P_{ACO_2} - P_{E'CO_2}$) at large tidal volumes was the result of several factors. Increases in $V_T$ and decreases in $f$ imply (i) that gas from "slow" alveoli can reach the mouth, whereas previously it remained in the airways; (ii) relatively better ventilation of dependent, well perfused alveoli. Froese and Bryan (1974) showed that, during anaesthesia with IPPV and the patient in the supine position, displacement of the dependent part of the diaphragm increased with increasing tidal volume. They also presented evidence that this was associated with greater dependent regional lung volume changes at larger tidal volumes. Mechanisms (i) and (ii) above are essentially the same as those that decrease $V_{D^{TV}}/V_T$ at large tidal volumes, and are intimately related to sequential emptying (and even preferential filling) (Rehder, Sessler and Rodarte, 1977). Small tidal volumes are conversely associated with relative overventilation of non-dependent, high $V/Q$ regions, which is the cause of the ($P_{ACO_2} - P_{E'CO_2}$) difference. Efficiency$_{CO_2}$ is increased at large tidal volumes, reflecting a sharper phase II–III transition. This may be the result of better filling of low $V/Q$ regions which then contribute more to initial expiration, and of more efficient washout of the airway as a result of turbulence at greater expiratory flows (Nunn, 1977). Perhaps three individuals (figs 6 and 7) in this study had significant alveolar deadspace of non-sequential causes—that is, an above-average alveolar deadspace associated with an almost horizontal phase III.

These patients had large $C_*$ values (mean 108 ml cm $H_2O^{-1}$). This pattern may be seen in the presence of ventilated, non-perfused lung segments, such as pulmonary embolism.

The arterial–end-tidal carbon dioxide difference as an index of alveolar deadspace. Figure 8 describes the relationship between $V_{D^{TV}}/V_T$ and ($P_{ACO_2} - P_{E'CO_2}$)/$P_{ACO_2}$. It has been stated previously (Fletcher et al., 1981) that the deviation from the line of identity approximates to phase III slope $\times V_{D^{TV}}/2$. At large tidal volumes, $V_{D^{TV}}$ increases more than slope decreases, and so the deviation from the line of identity is greater than at small $V_T$. In a group of patients where both $V_T$ and phase III slope differ, ($P_{ACO_2} - P_{E'CO_2}$)/$P_{ACO_2}$ is a poor index of the alveolar deadspace fraction.

Physiological deadspace. Although $V_{D^{TV}}$ was increased significantly at large tidal volumes (table III), $V_{D^{TV}}/V_T$ was decreased greatly, allowing a 13% decrease in total ventilation for the same alveolar ventilation. Comparison with other workers' results is of limited value since most authors who have studied similar patients do not give clear details of the ventilatory patterns used. However, the results presented compare with those of Hedenstierna and McCarthy (1975) who ventilated patients younger than ours and who used an accelerating flow pattern. They are also similar to those of Cooper (1961, 1967) who used a flow pattern that approximated to a square wave.

With three exceptions (emphysema, pneumonia, small areas of atelectasis), deadspace in patients with a history of pulmonary disease was distributed, with respect to age, in a manner similar to
that obtained in the other patients. Thus, the information available from routine preoperative investigation is of little help in identifying patients who will have increased $V_D^{phys}/V_T$ during anaesthesia with IPPV. However, we have shown previously a strong correlation between $V_D^{phys}/V_T$ during anaesthesia with IPPV and age for smokers. Non-smokers showed no such correlation (Fletcher and Jonson, 1981).

The lower deadspace ratio observed at large $V_T$ has in itself little importance in individuals with fairly normal lungs. However, the more efficient gas exchange suggests that various lung compartments are more evenly ventilated and that the tendency to airway obstruction or closure may be decreased. Such factors may be of importance in the prevention of complications such as infection, compromised clearance of mucus from airways, and atelectasis. The present study lends some support to the use of large $V_T$ rather than small in as far as airway pressures allow.

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RESUMEN

En utilizan el test de respiración unico al CO₂ (TRU-CO₂), los
constituyentes del espacio mort fisiolópico y los gas de la
anestesia con VPPI en 58 pacientes. La ventilación
comportaba un débit inspiratorio en creócalc con una pausa de fin
de inspiración (respectivamente 25% y 10% de la duración del ciclo).
Por dos volumenes courants de 450 ml (f = 17 r.p.m.) y 750 ml
(f = 9 r.p.m.), los valores medios de VD'*/VT eran de 0,44 y
0,31. La reducción de VT y la disminución de f no
modificaban que el espacio mort alveolar VD'*/VT disminuye 93% desde
sujets (P < 0,001). Así estos anécdotos obtenidos en aumentando el VT
peuen fixture los efectos beneficiosos sobre la distribución de
gas y el tiempo de perfusión. Los pacientes donde los espacios
mort alveópicos estaban aumentando estaban en el phase III del
TRU-CO₂ que era de 0,6 kPa para dos volumenes courants
faibles y de 0,3 kPa para los volumes courants
3kPa en los grandes
(P < 0,001). Tres pacientes mostraron valores en cero del
(Paco₂ - PaCO₂) mientras que otros cuatro tenían valores
negativos para volúmenes respiratorios importantes. Cuando la
fase III baja abruptamente, se puede observar valores negativos
(Paco₂ - PaCO₂) en presencia de espacio mort alveolar.