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-CO\(_2\)), 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 (\(\dot{V}_t = 17\) b.p.m.) and 0.75 litre (\(\dot{V}_t = 9\) b.p.m.), median values for \(V_{D}\) were 0.44 and 0.31. Increasing \(V_t\) and decreasing \(f\) did not change airway deadspace (\(V_{D}^{aw}\)) so that the fraction \(V_{D}^{aw}/\dot{V}_t\) was decreased (\(P<0.001\)). The alveolar deadspace fraction, \(V_{D}^{al}/\dot{V}_t\), was decreased in 93% of patients (\(P<0.001\)). These improvements with increasing \(V_t\) can be attributed to beneficial effects on gas distribution and diffusion time. Patients with large alveolar deadspaces had steeply sloping SBT-CO\(_2\) phase III, and increased expiratory time constants of the respiratory system. The median arterial-end-tidal \(P_{CO_2}\) difference, \((P_{CO_2} - P_{E'TCO_2})\), 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 \((P_{CO_2} - P_{E'TCO_2})\) values at large tidal volumes. When phase III slopes steeply, negative \((P_{CO_2} - P_{E'TCO_2})\) values may be observed in the presence of alveolar deadspace.

Physiological deadspace (\(V_{D}^{phys}\)) during anaesthesia with intermittent positive pressure ventilation (IPPV) is increased when compared with \(V_{D}^{phys}\) 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 (\(V_{D}^{aw}\)) and alveolar deadspace (\(V_{D}^{al}\)), 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 \(V_{D}^{phys}\), its constituents, and factors influencing them has been presented previously (Fletcher et al., 1981). The importance of not only end-tidal \(P_{CO_2}\) (\(P_{E'TCO_2}\)), but also of information from the entire tracing over a breath of expired \(P_{CO_2}\) (\(P_{E'TCO_2}\), or fraction, \(F_{CO_2}\)) against tidal volume, the single breath test for carbon dioxide (SBT-CO\(_2\)) (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 (\(V_{D}^{phys}/\dot{V}_t\)), was decreased significantly when ventilatory frequency (\(f\)) was decreased and \(V_t\) increased. The aim of the present study was to analyse changes in deadspace and SBT-CO\(_2\) 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...
FIG. 1. Left: The classic presentation of the single breath test for carbon dioxide. It is an original tracing displaying typical features at large $V_T$. After phase I, the carbon dioxide-free part of the breath, the expired volumes are designated effective volumes (uppermost ordinate). The slope of phase III is measured between 0.2 and 0.4 litre effective volume. Efficiency $V_{ET}$ is calculated from $FE_{CO2}$ and $VE_{CO2}$ at effective volume 0.4 litre. Efficiency $V_{ET}$ is calculated for the whole effective volume ($VE_{CO2}$). Right: SBT-CO$_2$ presented according to Langley and colleagues (1975). Expired carbon dioxide volume ($VE_{CO2}$) is on the abscissa. The interrupted line illustrates how the first fairly linear portion of the curve is extrapolated to give $VE_{CO2}^m$.

FIG. 2. Left: The phases of SBT-CO$_2$. Phase I, the carbon dioxide-free phase, contains compressed gas, c, during IPPV. Phase II is the S-shaped upswing, and phase III is the "alveolar plateau". The figures 0.2 and 0.4 refer to effective volumes, that is volumes measured from the onset of phase II. Right: Estimation of efficiency. Area X, the volume of carbon dioxide contained in the breath is related to area ABCDA, or the volume of carbon dioxide contained in a theoretical ideal lung with the same effective tidal volume and end-tidal $FE_{CO2}$. (Reproduced from Fletcher and colleagues (1981).)

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 CO$_2$ 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 CO$_2$ 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-CO$_2$ in two diagrams with expired volume on the ordinate and $FE_{CO2}$ or expired carbon dioxide volume on the abscissa. Expired volume was obtained by integration of flow. The volume of carbon dioxide was obtained by integration of (flow $\times FE_{CO2}$). 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, linearity 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-CO₂. Gas compression and apparatus deadspace influence phase I to a degree that is difficult to assess in routine IPPV. Delineation of VDₑ 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 (VTₑ). One measure of the shape of SBT-CO₂ 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 PₐCO₂ decreases the slope of phase III, the increase in PₐCO₂ per litre was divided by the mean PₐCO₂ 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 (VTₑ) given by the area X under the tracing. This can be compared with that volume which would be eliminated if the whole VTₑ had FₐCO₂ equal to FₑCO₂. The ratio VTₑ/\(\frac{VTₑ}{FₑCO₂}\) is that area X/area ABCDA, is designated efficiency (Fletcher et al., 1981). If SBT-CO₂ 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 (efficiencyVTₑ) and for the initial 0.4 litre of the effective volume (efficiency₀.₄). The volume of 0.4 litre was corrected for body height thus:

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

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, VDₑ, was calculated according to

\[
VDₑ = VT \times (1 - \frac{페CO₂}{PₐCO₂})
\]

Mixed expired PₐCO₂ (PₑCO₂) was calculated from SBT-CO₂ as \(VTₑ \times \text{barometric pressure (kPa)}/VT(\text{ml})\). VDₑ was obtained from:

\[
VDₑ = VDₑ + VDₑ
\]

Venous admixture. A rough estimate of venous admixture or right to left shunt fraction (Qₘ/Qₑ) was obtained from inspired F₂O₂ and P₂O₂ (Nunn, 1977). Assuming a mixed venous–arterial PₐCO₂ difference (PᵥCO₂ - PₐCO₂) of 1 kPa, pulmonary end-capillary PₑCO₂ (PₑCO₂) was calculated from the equation given by Mürzt (1961):

\[
PₑCO₂ = PₐCO₂ - (PᵥCO₂ - PₐCO₂) \times \frac{Qₘ/Qₑ}{1 - Qₘ/Qₑ}
\]

PₑCO₂ and PᵥCO₂ were used to obtain an approximate estimate of the extent to which shunt contributed to VTₑ/VT.

Time constants (RₑCₑ) for the respiratory system were calculated as the product of expiratory resistance, Rₑ (cm H₂O litre⁻¹ s) and compliance of the respiratory system, Cₑ (litre cm H₂O⁻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 (Paco2 - Pe'CO2), were normally distributed and could therefore be tested with Student’s t test. The difference for (Paco2 - Pe'CO2) was tested using the Wilcoxon test. n = 58 (for efficiency0.4 at small Vt, n = 50)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Small tidal volumes</th>
<th>Large tidal volumes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Var (ml)</td>
<td>Median 450</td>
<td>743</td>
</tr>
<tr>
<td>Var** (ml)</td>
<td>85</td>
<td>83</td>
</tr>
<tr>
<td>Var* (ml)</td>
<td>105</td>
<td>143</td>
</tr>
<tr>
<td>Var** (ml)</td>
<td>195</td>
<td>229</td>
</tr>
<tr>
<td>Var*/Var</td>
<td>0.19</td>
<td>0.11</td>
</tr>
<tr>
<td>Var*/Var**</td>
<td>0.29</td>
<td>0.22</td>
</tr>
<tr>
<td>Var*/Var**</td>
<td>0.44</td>
<td>0.31</td>
</tr>
<tr>
<td>(Paco2 - Pe'CO2) (kPa)</td>
<td>0.60</td>
<td>0.30</td>
</tr>
<tr>
<td>Efficiency0.4</td>
<td>0.79</td>
<td>0.82</td>
</tr>
<tr>
<td>EfficiencyVr**</td>
<td>0.80</td>
<td>0.83</td>
</tr>
<tr>
<td>Relative slope (litre^-1)</td>
<td>0.58</td>
<td>0.45</td>
</tr>
</tbody>
</table>

RESULTS

Data about tidal volume and its subdivisions are given in table II. Median airway deadspace was about 85 ml at both settings. Vd**/Vt was 0.19 and 0.11 at small and large Vt, respectively.

Median Vd** 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, Vd**/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 Vr** and efficiency were significantly greater at large tidal volumes.

The (Paco2 - Pe'CO2) difference was smaller at...
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Table III. The fraction $P_{CO_2}/P_{CO_2}$ at 0.2, 0.3 and 0.4 litre
effective volume was greater at small $V_T$ than at large. The figures
given are mean ± SD. $n = 58$ ($n = 51$ at 0.4 litre). The implications
of these findings are illustrated in figure 6.

<table>
<thead>
<tr>
<th>Effective volume (litre)</th>
<th>$P_{CO_2}/P_{CO_2}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small $V_T$</td>
<td>0.75±0.11</td>
</tr>
<tr>
<td>Large $V_T$</td>
<td>0.75±0.11</td>
</tr>
<tr>
<td>Difference</td>
<td>0.004</td>
</tr>
<tr>
<td>$P$</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

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 $P_{CO_2}$ after expiration of 0.2–0.4 litre
of the effective volume, was slightly closer to arterial
at small $V_T$ (table III; fig. 5). Six patients with small
$V_{D/VT}$ values (mean 0.19 at small $V_T$) deviated
from the above pattern: all had virtually horizontal
phase III (mean relative slope 0.25) and showed
little or no change in $V_{D/VT}$ 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 $V_T$
resulted in a lower inspiratory flow. Large tidal
volumes gave greater peak and pause airway pres-
sures 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, $RC_{f}$, 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></th>
<th>Large tidal volumes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Median or mean</td>
<td>5th</td>
<td>95th</td>
<td>Median or mean</td>
</tr>
<tr>
<td>Frequency (b.p.m.)</td>
<td>17.3</td>
<td>14.2</td>
<td>20.4</td>
<td>9.3</td>
</tr>
<tr>
<td>Minute ventilation</td>
<td>7.9</td>
<td>5.4</td>
<td>10.4</td>
<td>6.9</td>
</tr>
<tr>
<td>$P_{CO_2}$ (kPa)</td>
<td>4.2</td>
<td>3.2</td>
<td>5.1</td>
<td>4.2</td>
</tr>
<tr>
<td>Peak pressure (cm H$_2$O)</td>
<td>18</td>
<td>13</td>
<td>26</td>
<td>20</td>
</tr>
<tr>
<td>Pause pressure (cm H$_2$O)</td>
<td>11</td>
<td>8</td>
<td>16</td>
<td>14</td>
</tr>
<tr>
<td>$C_a$ (ml cm H$_2$O$^{-1})$</td>
<td>64</td>
<td>39</td>
<td>123</td>
<td>68</td>
</tr>
</tbody>
</table>
TABLE V. Correlation between airway deadspace (y) at small tidal volumes and height, pause pressure and $C_m$ (x) according to the formula $y = mx + b$. *RSD = residual standard deviation.

<table>
<thead>
<tr>
<th>Height (m)</th>
<th>Surface area (m²)</th>
<th>Pause pressure (cm H₂O)</th>
<th>$C_m$ (ml cm H₂O⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>m</td>
<td>123</td>
<td>-122</td>
<td>19</td>
</tr>
<tr>
<td>b</td>
<td>21</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>RSD</td>
<td>0.54**</td>
<td>0.37**</td>
<td>0.35**</td>
</tr>
</tbody>
</table>

TABLE VI. Correlation between deadspace variables (y) and age, $RC_{cr}$, late expiratory flow, and relative slope (x), 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>$RC_{cr}$ (s)</th>
<th>Late expiratory flow (litre⁻¹)</th>
<th>Relative slope (litre⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_{D^{LV}}/V_{T^{LV}}$ Small VT</td>
<td>m</td>
<td>0.0025</td>
<td>0.112</td>
<td>1.67</td>
</tr>
<tr>
<td></td>
<td>b</td>
<td>0.16</td>
<td>0.18</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>RSD</td>
<td>0.09</td>
<td>0.09</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>r</td>
<td>0.41**</td>
<td>0.39**</td>
<td>0.46**</td>
</tr>
</tbody>
</table>

| $V_{D^{LV}}/V_{T^{LV}}$ Large VT | m        | 0.0019        | 0.13                          | n.s.                      |
|                                | b        | 0.14          | 0.07                          | n.s.                      |
|                                | RSD      | 0.09          | 0.09                          | n.s.                      |
|                                | r        | 0.32*         | 0.42**                        | n.s.                      |

| $V_{D^{LV}}/V_{T^{LV}}$ Small VT | m        | 0.0026        | 0.099                         | 1.40                      |
|                                | b        | 0.29          | 0.33                          | 0.39                     |
|                                | RSD      | 0.07          | 0.08                          | 0.08                     |
|                                | r        | 0.48**        | 0.38**                        | 0.43**                   |

| Relative slope Small VT | m        | 0.0088        | 0.46                          | 8.18                      |
|                        | b        | 0.12          | 0.10                          | 0.30                      |
|                        | RSD      | 0.29          | 0.29                          | 0.24                      |
|                        | r        | 0.43**        | 0.47**                        | 0.66**                    |

| Relative slope Large VT | m        | 0.0067        | 0.41                          | 3.38                      |
|                        | b        | 0.06          | -0.12                         | 0.37                      |
|                        | RSD      | 0.26          | 0.25                          | 0.27                      |
|                        | r        | 0.38**        | 0.45**                        | 0.28*                     |

**FIG. 5.** SBT-CO₂ tracings constructed to show the typical differences found at the two different ventilator settings. Heavy line: $VT = 0.45, f= 17$ b.p.m.; thin line: $VT = 0.75, f= 9$ b.p.m. The dotted line represents $P_{ACO₂}$ or $P_{ECO₂}$. Increases in VT and decreases in $f$ decrease relative slope and $(P_{ACO₂} - P_{ECO₂})$. The extra ventilation at large tidal volumes is preferentially distributed to dependent, well perfused lung regions so that $V_{D^{LV}}/V_{T^{LV}}$ is decreased.

**FIG. 6.** The relationship between phase III slope (relative slope) and $V_{D^{LV}}/V_{T^{LV}}$ 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.

flow (the flow persisting at the very end of expiration) and relative slope (fig. 6). It was not correlated to pause pressure, $C_m$ or expiratory resistance. Relative slope was positively correlated to age, $RC_{cr}$ and late expiratory flow.

The $(P_{ACO₂} - P_{ECO₂})$ difference was related to
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Fig. 7. The relationship between efficiency $\nu_{\text{eff}}$ and $(P_{\text{aco}2} - P_{\text{evo}2})$ at small tidal volumes. The three patients represented by empty circles probably have increased alveolar deadspace of non-sequential origins.

Efficiency (fig. 7):

$$(P_{\text{aco}2} - P_{\text{evo}2}) = 4.9 - 5.36 \times \text{efficiency } \nu_{\text{eff}}$$

RSD = 0.30

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

Fig. 8. Left: The relationship between $(P_{\text{aco}2} - P_{\text{evo}2})/P_{\text{aco}2}$ and $V_{\text{Dav}}/V_{\text{Ta}}$ at large tidal volumes. The large scatter is caused by individual variation in the relationship between phase III and $P_{\text{aco}2}$ (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 \((P_{aco_2} - P_{e,co_2})/P_{aco_2}\) was related to \(V_{d,aw}/V_{t,aw}\) (fig. 8).

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

**DISCUSSION**

The purpose of this study was to analyse changes in deadspace and SBT-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 \(V_t\). 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 \(V_t\) (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. \(V_{d,aw}\) 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 \(V_{d,aw}\). These various influences obviously balanced each other when the setting was changed.

Estimates of \(V_{d,aw}\) based on the use of \(P_{e,co_2}\) (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 \(V_{d,aw}\) 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 \(V_{d,aw}/V_{t,aw}\) 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 \(V_t\) (Rehder, Sessler and Rodarte, 1977). The increased \(C_e\) at large \(V_t\) may be the result of (i) greater chest wall compliance at larger tidal volumes (Grimby, Hedenstierna and Löfström, 1975) and (ii) lung areas with poor ventilation at small tidal volumes taking a larger share of the ventilation at large \(V_t\), possibly by the opening up of almost closed units and a more efficient alveolar surfactant system at large \(V_t\).

Six younger patients with small deadspaces showed no improvement in \(V_{d,aw}/V_{t,aw}\) or \((P_{aco_2} - P_{e,co_2})\) at large tidal volumes. It may be speculated that they had, even at small \(V_t\), 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 \(V_{d,aw}/V_{t,aw}\) 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-CO\(_2\): the slope of phase III, and the relationship of phase III to \(P_{aco_2}\).

*The slope of phase III.* Patients with steep phase III slopes had large alveolar deadspaces (fig. 6). \(V_{d,aw}/V_{t,aw}\) and relative slope were decreased at large tidal volumes. This, together with the association between increased \(V_{d,aw}\) 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 \(R_{C_a}\), 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 \(V_{d,aw}\). 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
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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 VD/VT (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 PACO₂ and (ii) changes the time course of alveolar carbon dioxide evolution (Fletcher, 1980; Fletcher et al., 1981).

The relationship between PE'CO² and PACO². At small tidal volumes (PACO₂ - PE'CO²) 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%. (PACO₂ - PE'CO²) was thus strongly dependent upon VT, which is to be expected as phase III almost always has a positive slope. This dependence upon VT 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 (PACO₂ - PE'CO²) difference was observed during exercise by Jones, Robertson and Kane (1979). The existence of negative arterial-end-tidal PCO² gradients is understood if it is remembered that PACO₂ represents the temporal and spatial mean alveolar PCO₂ (Riley et al., 1946; Fletcher et al., 1981). The correlation between (PACO₂ - PE'CO²) and efficiency is of practical interest since it allows a non-invasive estimate of PACO₂ which should be more accurate than PE'CO² alone (Fletcher, 1980).

The PACO₂-phase III difference. Figure 5 shows that, although the PACO₂-phase III difference at given expired volumes was slightly greater at large VT (table III), the net effect of increasing VT was to decrease (PACO₂ - PE'CO²) and VD/VT.

The decrease in (PACO₂ - PE'CO²) at large tidal volumes was the result of several factors. Increases in VT and decreases in V/Q 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 VD/VT 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 (PACO₂ - PE'CO²) difference. Efficiency (i) 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 C₄ values (mean 108 ml cm H₂O⁻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 VD/VT and (PACO₂ - PE'CO²)/PACO₂. It has been stated previously (Fletcher et al., 1981) that the deviation from the line of identity approximates to phase III slope × VTₑ/VTₑ/2. At large tidal volumes, VTₑ increases more than slope decreases, and so the deviation from the line of identity is greater than at small VT. In a group of patients where both VT and phase III slope differ, (PACO₂ - PE'CO²)/PACO₂ is a poor index of the alveolar deadspace fraction.

Physiological deadspace. Although VDₑ was increased significantly at large tidal volumes (table II), VDₑ/VT 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 $Vd_{phys}/VT$ during anaesthesia with IPPV. However, we have shown previously a strong correlation between $Vd_{phys}/VT$ 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 $VT$ 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 $VT$ rather than small in as far as airway pressures allow.

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En utilisant le test de respiration unique au CO₂ (TRU - CO₂), les constituant de l’espace mort physiologique ont été étudiés au cours de l’anesthésie avec VPPI chez 58 patients. La ventilation comportait un débit inspiratoire en créneau avec une pause de fin d’inspiration (respectivement 25% et 10% de la durée du cycle). Pour des volumes courants de 450 ml (f = 17 r.p.m.) et 750 ml (f = 9 r.p.m.), les valeurs moyennes de Vdphys/VT étaient de 0,44 et 0,31. L’augmentation de VT et la diminution de f ne modifiaient pas l’espace mort des voies aériennes (Vdm) de telle sorte que le rapport Vdphys/VT s’abaissait (P < 0,001). La fraction d’espace mort alvéolaire Vtmphys/Vtmphys diminuait chez 93% des sujets (P < 0,001). Ces améliorations obtenues en augmentant le VT peuvent être attribuées aux effets bénéfiques sur la distribution des gaz et le temps de diffusion. Les patients dont les espaces morts alvéolaires étaient importants avaient les phases III du TRU – CO₂ très pentues, ainsi que des constantes de temps expiratoire du système respiratoire augmentées. La différence moyenne entre la PCO₂ artérielle et la PCO₂ de fin d’expiration (Paco₂ – PeCO₂) était de 0,6 kPa pour des volumes courants faibles et de 0,3 kPa pour des volumes courants importants (P < 0,001). Trois patients avaient des valeurs de (Paco₂ – PeCO₂) nulles et quatre valeurs négatives pour des volumes courants importants. Lorsque la phase III est très pentue, des valeurs négatives de (Paco₂ – PeCO₂) peuvent être observées en présence d’un espace mort alvéolaire important.

Bei 58 Patienten wurden mit dem Einzelatemzugs-Test für CO₂ (Single Breath Test = SBT-CO₂) die Komponenten des physiologischen Totraums während Narkose mit IPPV untersucht. Ein inspiratorischer Squarewave-Flow und eine endexspiratorische Pause (25% bzw 10% der Zykluszeit) wurden angewandt. Bei Zugvolumina von 0,45 litre (f = 17) und 0,75 litre (f = 9) waren die Mittelwerte für Vdphys/VT 0,44 und 0,31. Ansteigendes VT und abfallende Frequenz veränderten den Totraum der Atemwege (Vdm) nicht, so daß das Verhältnis Vtphys/VT abfiel (P < 0,001). Das alveolare Totraumverhältnis Vtphys/VT phys verringerte sich bei 93% der Patienten (P < 0,0001). Diese Verbesserungen mit ansteigendem VT können auf günstige Wirkungen der Gasverteilung und Diffusionszeit zurückgeführt wurden. Patienten mit großen alveolaren Toträumen haben steil abfallende Phasen III im SBT-CO₂, und ansteigende Expirationszeit-Konstanten des Atemsystems. Die mittlere arterielle endexspiratorische PCO₂-Differenz (Paco₂ – PeCO₂) war bei kleinen Zugvolumina 0,6 kPa und bei großen 0,3 kPa (P < 0,001). Drei Patienten hatten 0 und vier negative (Paco₂ – PeCO₂)-Werte bei großen Zugvolumina. Wenn die Phase III steil abfällt, können bei alveolarem Totraum negative (Paco₂ – PeCO₂)-Werte auftreten.

Mediante el ensayo de aliento único para el anhídrido carbónico (SBT-CO₂), se investigaron los componentes del espacio muerto fisiológico durante la anestesia con IPPV en 58 pacientes. Se usaron una corriente inspiratoria de onda cuadrada y una pausa inspiratorio-terminal (25% y 10% del tiempo del ciclo respectivamente). En volúmenes respiratorios de 0,45 litro (f = 17 b.p.m.) y 0,75 litro (f = 9 b.p.m.), los valores medios del Vdphys/VT eran de 0,44 y de 0,31. Al aumentar el VT y disminuir el f, no se registró ningún cambio en el espacio muerto de las vías respiratorias (Vdphys), de manera que la fracción (Vdphys/VT) disminuyó (P < 0,001). La fracción del espacio muerto alveolar Vtmphys/VTmphys disminuyó en un 93% de los pacientes (P < 0,001). Estas mejorías con un creciente VT pueden atribuirse a los efectos beneficiosos sobre la distribución y el tiempo de difusión de los gases. Los pacientes con espacios muertos alveolares importantes registraron una fase III fuertemente descendente del SBT-CO₂ y unas constantes crecientes del tiempo expiratorio del sistema respiratorio. La diferencia en el PCO₂ respiratorio-terminal–arterial medio (Paco₂ – PeCO₂) era de 0,6 kPa en pequeños volúmenes respiratorios y de 0,3 kPa en los grandes (P < 0,001). Tres pacientes demostraron valores en cero del (Paco₂ – PeCO₂) mientras que otros cuatro tenían valores negativos para volúmenes respiratorios importantes. Cuando la fase III baja abruptamente, se puede observar valores negativos (Paco₂ – PeCO₂) en presencia de espacio muerto alveolar.

En utilisant le test de respiration unique au CO₂ (TRU-CO₂), les constituant de l’espace mort physiologique ont été étudiés au cours de l’anesthésie avec VPPI chez 58 patients. La ventilation comportait un débit inspiratoire en créneau avec une pause de fin d’inspiration (respectivement 25% et 10% de la durée du cycle). Pour des volumes courants de 450 ml (f = 17 r.p.m.) et 750 ml (f = 9 r.p.m.), les valeurs moyennes de $V_{d\text{phys}}/VT$ étaient de 0,44 et 0,31. L’augmentation de $VT$ et la diminution de $f$ ne modifiaient pas l’espace mort des voies aériennes ($V_{dm}$) de telle sorte que le rapport $V_{d\text{phys}}/VT$ s’abaissait ($P < 0,001$). La fraction d’espace mort alvéolaire $V_{tm\text{phys}}/V_{tm\text{phys}}$ diminuait chez 93% des sujets ($P < 0,001$). Ces améliorations obtenues en augmentant le VT peuvent être attribuées aux effets bénéfiques sur la distribution des gaz et le temps de diffusion. Les patients dont les espaces morts alvéolaires étaient importants avaient les phases III du TRU–CO₂ très pentues, ainsi que des constantes de temps expiratoire du système respiratoire augmentées. La différence moyenne entre la PCO₂ artérielle et la PCO₂ de fin d’expiration ($P_{aco_2} – P_eCO_2$) était de 0,6 kPa pour des volumes courants faibles et de 0,3 kPa pour des volumes courants importants ($P < 0,001$). Trois patients avaient des valeurs de ($P_{aco_2} – P_eCO_2$) nulles et quatre valeurs négatives pour des volumes courants importants. Lorsque la phase III est très pentue, des valeurs négatives de ($P_{aco_2} – P_eCO_2$) peuvent être observées en présence d’un espace mort alvéolaire important.