RELATIONSHIP BETWEEN ALVEOLAR DEADSPACE AND ARTERIAL OXYGENATION IN CHILDREN WITH CONGENITAL CARDIAC DISEASE†

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Intracardiac right-to-left (RL) shunts produce differences between alveolar and arterial partial pressures for oxygen and carbon dioxide. The alveolar–arterial differences in $PcO_2$ are deliberately ignored in the conventional calculation of the shunt fraction $Qs/Qt$. The calculation is based on the contents of oxygen in arterial, mixed venous and pulmonary end-capillary blood ($CaO_2, CvO_2$ and $CcO_2$, respectively):

$$\frac{Qs}{Qt} = \frac{(CcO_2 - CaO_2)}{(CcO_2 - CvO_2)} \quad (1)$$

$CcO_2$ is estimated from the haemoglobin dissociation curve and $PcO_2$, the $Po_2$ of pulmonary end-capillary blood; the latter is estimated from the ideal alveolar gas equation:

$$PcO_2 = PoO_2 = PiO_2 - \frac{PaCO_2}{R} \quad (2)$$

where $PaO_2$ and $PaCO_2$ are the ideal alveolar partial pressures of oxygen and carbon dioxide; $R$ = respiratory quotient; $PiO_2$ = inspired partial pressure of oxygen. $PaCO_2$ is assumed to be equal to $PaCO_2$ (the arterial $PcO_2$), although for any appreciable $Qs/Qt$ this cannot be true. However, the assumption can give only a small error in the estimate of $PaO_2$; if $PaO_2$ is increased by a sufficiently increased $PiO_2$, end-capillary haemoglobin is fully saturated and the resulting error in the estimate of $CcO_2$ is negligible. The dissolved oxygen, the only component which can be affected by the error, makes only a small contribution to total $CcO_2$.

SUMMARY

Fifty-eight children were studied during nitrous oxide in oxygen and fentanyl anaesthesia before undergoing closed or open cardiac surgery. $FiO_2$ was 0.5. Alveolar deadspace was measured using the carbon dioxide single breath test (SBT-CO$_2$) obtained from a computerized on-line system for monitoring expired CO$_2$ and airway flow, based on the Servo ventilator. Arterial blood was sampled simultaneously for measurement of $PaCO_2$ and $PaO_2$. There was a marked reciprocal relationship between $PaO_2$ and the alveolar deadspace fraction. In children with a normal pulmonary circulation and good oxygenation, alveolar deadspace fraction was approx. 0.05. Shunts which reduced $PaO_2$ to 70 kPa produced a deadspace fraction of 0.15. When $VaO_2$ was 3-4 kPa, alveolar deadspace fraction was approx. 0.4. In well-oxygenated children, alveolar deadspace fraction was only slightly greater than predicted by a model of the effects of pure right-to-left shunting. In severely cyanotic children, the discrepancy between predicted and observed $VD/VT$ was greater. The mean arterial–end-tidal CO$_2$ difference was zero in children in whom $PaO_2$ was greater than 10 kPa, despite a measurable alveolar deadspace. In severely hypoxic children, the difference was 1–2 kPa. In a retrospective analysis of published data from anaesthetized adults without intracardiac shunting, no relationship was found between alveolar deadspace and $PaO_2$.

However, for any appreciable $Qs/Qt$, there is an appreciable alveolar–arterial $PcO_2$ difference. Although it results from a circulatory disturbance, a RL shunt reduces the efficiency of elimination of carbon dioxide. This effect can be expressed as a
contribution to the alveolar deadspace fraction of alveolar tidal volume:

\[
\frac{V_{D}^{alv}}{V_{T}^{alv}} = \frac{P_{a}CO_{2} - P_{a}CO_{2}}{P_{a}CO_{2} - P_{1}CO_{2}}
\]  

(3)

or, if \( P_{1}CO_{2} \) is zero:

\[
\frac{V_{D}^{alv}}{V_{T}^{alv}} = 1 - \frac{P_{a}CO_{2}}{P_{a}CO_{2}}
\]

(4)

In a real lung, the alveolar gas is inhomogeneous. \( P_{a}CO_{2} \) cannot, therefore, be represented by the \( P_{a}CO_{2} \) of gas sampled in any selected part of the alveolar space, or any instant in time (for example, end-tidal \( P_{a}CO_{2} \), \( P_{e}CO_{2} \))), and it is not necessarily represented by the mean \( P_{a}CO_{2} \) of the alveolar part of the expired tidal volume, \( P_{a}CO_{2} \). Nevertheless, if \( P_{a}CO_{2} \) is used in equation (3) instead of \( P_{a}CO_{2} \), one does obtain a useful measure of inefficiency of elimination of carbon dioxide:

\[
\frac{V_{D}^{alv}}{V_{T}^{alv}} = 1 - \frac{P_{a}CO_{2}}{P_{a}CO_{2}}
\]

(5)

The average concentration of any gas in the alveolar portion of the expirate can be measured by use of single breath analysis. This concept, introduced by Aitken and Clarke-Kennedy [1] and re-iterated later by Fowler [2], has been developed [3-6] as a computerized on-line technique for monitoring carbon dioxide elimination during ventilator treatment. It is used routinely in this department during paediatric cardiac anaesthesia. The single breath test for carbon dioxide (SBT-CO\(_2\)), is obtained by combining signals for airway flow and expired carbon dioxide, both of which are becoming recognized as desirable facets of routine monitoring.

This paper describes the use of SBT-CO\(_2\) to estimate \( V_{D}^{alv}/V_{T}^{alv} \) in a group of children about to undergo cardiac surgery. Many of the children were cyanotic, with right-to-left (RL) or mixed RL and left-to-right (LR) shunts. The paper examines particularly the inverse relationship between \( V_{D}^{alv}/V_{T}^{alv} \) and \( P_{a}O_{2} \), which was found in these children; this contrasts with the lack of relationship seen in a group of adults studied in a similar way (unpublished data from Fletcher and Jonson [3]). The observations are compared with the results of a theoretical model of the \( V_{D}^{alv}/V_{T}^{alv} \) v. \( P_{a}O_{2} \) relationship which should follow from pure RL shunting.

**THEORY**

**Relationship between \( V_{D}^{alv}/V_{T}^{alv} \) and \( P_{a}O_{2} \) for different RL shunts**

For a pure RL shunt, \( Q_{s}/Q_{t} \) is related to the oxygen contents \( C_{a}O_{2} \), \( C_{V}O_{2} \) and \( C_{c}O_{2} \) by equation (1). This principle can be applied equally to carbon dioxide contents:

\[
\frac{Q_{s}}{Q_{t}} = \frac{(C_{a}CO_{2} - C_{c}CO_{2})}{(C_{V}CO_{2} - C_{c}CO_{2})}
\]

(6)

Rearranging equations (1) and (6), it can be shown that, for a given \( Q_{s}/Q_{t} \), the arterial-end-capillary differences can be calculated from arteriovenous differences:

\[
(1 - \frac{Q_{s}}{Q_{t}}). (C_{a}O_{2} - C_{c}O_{2}) = \frac{Q_{s}}{Q_{t}}. (C_{a}O_{2} - C_{V}O_{2})
\]

(7)

and

\[
(1 - \frac{Q_{s}}{Q_{t}}). (C_{a}CO_{2} - C_{c}CO_{2}) = \frac{Q_{s}}{Q_{t}}. (C_{V}CO_{2} - C_{c}CO_{2})
\]

(8)

Thus the expected arterial-end-capillary differences can be calculated from arteriovenous differences for a given shunt. Dividing equation (8) by equation (7), it also follows that the respiratory quotient \( R \) describes, not only the ratio of arteriovenous differences in carbon dioxide and oxygen contents, but also the ratio of end-capillary-arterial and end-capillary-mixed venous differences. Thus:

\[
\frac{(C_{a}CO_{2} - C_{c}CO_{2})}{(C_{c}CO_{2} - C_{a}CO_{2})} = R
\]

(9)

If ventilation is controlled to maintain a constant \( P_{a}CO_{2} \), and if \( F_{I}O_{2} \) is increased to 0.4 or more, \( C_{a}CO_{2} \) and \( C_{c}CO_{2} \) will be fixed. \( C_{c}CO_{2} \) and \( C_{a}CO_{2} \) will vary with \( Q_{s}/Q_{t} \). The corresponding \( P_{a}CO_{2} \) and \( P_{a}O_{2} \) can be determined by applying the appropriate oxygen and carbon dioxide dissociation curves for blood of appropriate haemoglobin content. Finally, \( V_{D}^{alv}/V_{T}^{alv} \) can be calculated from equation (3) and plotted against \( P_{a}O_{2} \) to give the theoretical relationship between the two variables. If the oxygen and carbon dioxide dissociation curves were simple linear relationships, the \( P_{a}CO_{2} \) v. alveolar deadspace fraction relationship would also be linear. The non-linearity of the Hb dissociation curve is reflected in the non-linearity of the \( V_{D}^{alv}/V_{T}^{alv} \) v. \( P_{a}O_{2} \) relationship.
There are several possible contributions to the \( V_{D}^{alv}/V_{T}^{alv} \) v. \( P_{aO_2} \) relationship, in addition to that of a pure RL shunt (see discussion). This paper attempts to assess their importance by comparing the theoretical relationship with observations in children with cyanotic and acyanotic heart disease.

**PATIENTS AND METHODS**

Fifty-eight children about to undergo closed or open cardiac surgery were studied in the supine position during undisturbed fentanyl-nitrous oxide anaesthesia with intermittent positive pressure ventilation (IPPV). Eight of the children also received 0.5% halothane. \( F_{O_2} \) was 0.50. The ventilator, a Servo 900C, was set to give constant-flow, volume-controlled ventilation at a frequency of 25–35 b.p.m., depending on body weight, and an inspiratory time of 25% with 10% pause. Minute volume was adjusted to give moderate hypocapnia (\( P_{aCO_2} 4–5 \) kPa). Positive end-expiratory pressure was not used.

The children (some of whose deadspace values have been described previously [6]) were allocated to four groups according to the characteristics of their central circulation, determined by preoperative angiography, intracardiac pressure and saturation measurements, echocardiography, chest x-ray and haemoglobin determinations. Thus 13 were shown to have normal lungs and pulmonary circulation, 17 had pure left-to-right (LR) intracardiac shunting, 18 had pure RL shunting and 10 probably had mixed RL–LR shunting. Diagnoses, anthropomorphic details and some gas exchange variables are given in tables I and II.

Measurements were made using an on-line system for monitoring expired carbon dioxide [7]. A Facit computer received signals for airway flow and pressure from the ventilator, and signals for expired \( P_{CO_2} \) from a Siemens-Elema CO\(_2\) Analyzer 930 [8]. As the transducer of the carbon dioxide analyser is placed directly across the airway ("in-line") there is no sampling lag and consequently, airway flow and carbon dioxide signals are virtually synchronous [5]. The com-

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### Table I. Diagnoses in the children. ASD, VSD = atrial, ventricular septal defects; PDA = patent ductus arteriosus; TGA = transposition of the great arteries; AVC = common atrioventricular canal; TAPV = total anomalous pulmonary venous drainage.

<table>
<thead>
<tr>
<th>Normal circulation</th>
<th>LR shunts</th>
<th>RL shunts</th>
<th>Mixed shunts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary stenosis</td>
<td>3 ASD</td>
<td>6 TGA</td>
<td>5 &quot;Acyanotic Fallot&quot;</td>
</tr>
<tr>
<td>Aortic coarctation</td>
<td>3 VSD</td>
<td>3 Pulmonary</td>
<td>1 TGA</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>3 Large PDA</td>
<td>8 stenosis or TAPV, ASD</td>
<td>1</td>
</tr>
<tr>
<td>Small PDA</td>
<td>5 Total</td>
<td>17 VSD, pulmonary hypertension 2</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>17 TGA</td>
<td>18 Mixed shunts</td>
</tr>
</tbody>
</table>

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### Table II. Ages, weights and some ventilatory variables in the children (mean (SD or range)). Significantly different from group with normal circulation, after Bonferroni correction: **P < 0.01; *P = 0.05.**

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>( V_T ) (ml)</th>
<th>( P_{aO_2} ) (kPa)</th>
<th>( \frac{V_{D}^{alv}}{V_{T}^{alv}} )</th>
<th>( P_{aCO_2} - P_{CO_2}^{alv} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal pulmonary circulation (( n = 13 ))</td>
<td>5.5 (3.7)</td>
<td>19.8 (9.1)</td>
<td>170 (69)</td>
<td>33.8 (5.5)</td>
<td>0.05 (0.03)</td>
</tr>
<tr>
<td>LR shunt (( n = 17 ))</td>
<td>3.6 (3.0)</td>
<td>13.7 (6.9)</td>
<td>119 (44)</td>
<td>26.8 (6.2)**</td>
<td>0.05 (0.05)</td>
</tr>
<tr>
<td>RL shunt (( n = 18 ))</td>
<td>2.4 (2.0)**</td>
<td>9.2 (5.3)**</td>
<td>79 (38)**</td>
<td>7.3 (2.5)**</td>
<td>0.25 (0.12)**</td>
</tr>
<tr>
<td>Mixed shunt (( n = 10 ))</td>
<td>1.3 (1.3)**</td>
<td>8.8 (4.0)**</td>
<td>80 (31)**</td>
<td>13.2 (1.3)**</td>
<td>0.12 (0.07)</td>
</tr>
</tbody>
</table>
ALVEOLAR DEADSPACE IN CARDIAC DISEASE

Fig. 1. Typical carbon dioxide single breath test from a child with a large RL shunt. Area Z represents the airway deadspace. $V_{E}^{alv}/V_{T}^{alv}$ can be represented by $Y/(X+Y)$.

puter presented the single breath test for carbon dioxide (SBT-CO$_2$) (the plot of expired carbon dioxide against expired volume) and calculated deadspace from a supplied value for $P_{A}CO_2$. Figure 1 shows a typical single breath test from a child with a large RL shunt. The shape of the curve can be described as follows: the initial expire has zero carbon dioxide content (phase I). Thereafter follows a sharp increase in $F_E^{CO_2}$ (phase II) as alveolar gas begins to reach the airway opening. Phase III is sometimes referred to as the alveolar plateau. It consists of alveolar gas, and normally has a slight slope, caused by (a) the continuing output of carbon dioxide into the alveoli, and (b) spread of ventilation/perfusion (V/Q) values; areas of high V/Q normally empty before those of low V/Q [4]. The contribution of (a) to phase III slope during mechanical ventilation in children can be shown to be small [4].

The area under the curve is the volume of carbon dioxide in the breath, $V_{T}^{CO_2}$. The airway deadspace, $V_{D}^{aw}$, was obtained as the volume of the expirate when $F_E^{CO_2}$ was 50% of early phase III $F_C^{CO_2}$. (This construction is feasible only in children, in whom phase III slope is small.) Physiological deadspace was obtained from:

$$V_{D}^{phys}/V_{T} = \frac{F_{A}^{CO_2}-F_{E}^{CO_2}}{F_{A}^{CO_2}-F_{I}^{CO_2}}$$

(10)

where $F_{E}^{CO_2}$ mixed expired $F_C^{CO_2}$, was obtained from $V_{T}^{CO_2}/V_{T}$. Inspired $F_C^{CO_2}$ ($F_{I}^{CO_2}$) is not zero because approximately 3 ml of apparatus deadspace gas is inspired from the Y-piece. $F_{A}^{CO_2}$ was estimated by dividing the carbon dioxide content of this gas (regarded as being end-tidal gas) by the alveolar tidal volume ($V_{T}^{alv}$) which is given by ($V_{T}^{alv}-V_{D}^{aw}$). $F_{A}^{CO_2}$, the $F_C^{CO_2}$ of a gas in equilibrium with arterial blood, was calculated by the computer from a temperature-corrected value for $P_{A}CO_2$. Body temperature was measured by an oesophageal probe. $V_{D}^{alv}$ was obtained from ($V_{D}^{phys}-V_{D}^{aw}$). The slope of phase III was measured as the increase in $F_E^{CO_2}$ between expired volumes of 5% and 10% of predicted total lung capacity, divided by the mean $F_E^{CO_2}$ between these points.

Data from adults for comparison were taken from the patients presented by Fletcher and Jonson [3]. That study examined $V_{D}^{alv}/V_{T}^{alv}$ and several different variables, although not $P_{A}O_2$.

Deadspace variables were obtained as the mean of three breaths, sampled at the same time as arterial blood for blood-gas analysis (ABL2; Radiometer, Copenhagen) was withdrawn slowly from an indwelling femoral or radial artery catheter. (None of the children had RL shunting through a patent ductus arteriosus, therefore choice of sampling site should not influence the $P_{A}O_2$ obtained.) The carbon dioxide analyser was calibrated daily against a test gas which had been checked by tonometry against the blood-gas analyser. Blood-gas values were corrected to body temperature, according to Kelman and Nunn [9].

Deadspace variables were corrected for a tidal volume-dependent error in carbon dioxide measurement [6].

Student's $t$ test was used for statistical comparisons. For the multiple comparisons in table I, the Bonferroni correction was applied.

Predicted alveolar deadspace–$P_{A}O_2$ relationship

In order to compare the observations with those that could be expected if RL shunting were the sole cause of alveolar deadspace, a model was constructed. The approach described in the theory section (above) was applied. $V_{D}^{alv}/V_{T}^{alv}$ and $P_{A}O_2$ were calculated for nine different values of $Q_{S}/Q_{T}$ (fig. 2) assuming:

$F_{I}^{O_2} = 0.5$

Hb concentration = 16 g dl$^{-1}$ (representing a mean value for all the children)

Oxygen combining capacity = 1.34 ml g$^{-1}$

The oxyhaemoglobin dissociation curve of Kelman and Nunn [9]

The carbon dioxide dissociation curve given by Nunn [10]
Normal pH and temperature

An RQ of 0.8

Two different values of arteriovenous difference in oxygen content: 4.4 and 6.3 ml dl⁻¹.

Linear regression of the predicted values for $V_{DAIV}/V_{TAIV}$ against $1/PaO_2$ (kPa) gave the following:

$V_{DAIV}/V_{TAIV} = 1.10/PaO_2 - 0.030$;
$r = 0.98$ for the lesser $(a - v)$ difference;
$V_{DAIV}/V_{TAIV} = 1.22/PaO_2 - 0.028$;
$r = 0.99$ for the greater $(a - v)$ difference (fig. 2).

RESULTS

Children

There was an inverse relationship between $V_{DAIV}/V_{TAIV}$ and temperature-corrected $PaO_2$ (kPa), described by the regression equation:

$V_{DAIV}/V_{TAIV} = -0.007 + 1.607/PaO_2$;
$r = 0.87$; residual standard deviation

(RSD) = 0.059.

The observations and the regression lines (solid line) are plotted in figure 3. For comparison, the
figure also shows the relationship (broken lines) predicted by the theoretical model (fig. 2) for the two (a — v) oxygen differences. There were significant correlations between 1/PaO₂ and V_{D}^{alv}/V_{T}^{alv} in the LR and RL groups:

LR: \( V_{D}^{alv}/V_{T}^{alv} = 0.023 + 1.97/PaO₂ \);
\( r = 0.49, P = 0.05 \)

RL: \( V_{D}^{alv}/V_{T}^{alv} = 0.023 + 1.79/PaO₂ \);
\( r = 0.81, P = 0.0001 \)

In the normal circulation \( r = -0.35 \) and mixed shunt groups \( r = 0.44 \) the correlations were not significant. \( PaO₂ \) correlated positively with age in the children with normal circulation and those with LR shunts \( r = 0.38, P = 0.04 \).

In the well-oxygenated children (LR and normal circulation groups), but not the others, the slope of phase III of SBT-CO₂ correlated negatively with \( PaO₂ (r = -0.36, P = 0.04) \). The relationship between \( PaO₂ \) and the arterial-end-tidal \( PCO₂ \) difference \( (PaCO₂ - PE'CO₂) \) is shown in figure 4.

**Adults**

Results from the adults are shown in figure 5. \( FE'O₂ \) was 0.35. There was no relationship between \( V_{D}^{alv}/V_{T}^{alv} \) and \( PaO₂ (r = 0.13, P = 0.3) \), or \( 1/PaO₂ \), \( (r = -0.14, P = 0.3) \).

**DISCUSSION**

The alveolar deadspace fraction of alveolar tidal volume \( (V_{D}^{alv}/V_{T}^{alv}) \) is a notional representation of imperfections in pulmonary gas exchange which create a difference between \( PAE_{CO₂} \) and \( PCO₂ \), by decreasing the former or increasing the latter, or both. \( PAE_{CO₂} \) is measured readily in a sample of arterial blood, but it is necessary to use SBT-CO₂ to measure \( PAE_{CO₂} \) because no single sample of alveolar expirate can represent the mean
\(P_{CO_2}\) of alveolar expirate. SBT-\(CO_2\) has been shown to measure expired carbon dioxide accurately during controlled ventilation [11], although it is necessary to correct for a small systematic error which occurs at tidal volumes of less than 200 ml [6].

The concept of alveolar deadspace is regarded usually as representing wasted or inefficient ventilation, caused by ventilation of poorly- or non-perfused alveoli. The primary effect of this is to reduce \(P_{A_{CO_2}}\) and therefore to increase alveolar deadspace, with no obligatory effect on \(P_{A_{O_2}}\). However, circulatory inefficiency, such as intra-cardiac RL shunting or perfusion of poorly- or non-ventilated areas (with low or zero ventilation/ perfusion (\(V/Q\)) ratios), also produces a measurable alveolar deadspace (which does not, however, represent ventilation of any compartment). Such circulatory inefficiency increases \(P_{A_{CO_2}}\) and reduces \(P_{A_{O_2}}\).

The primary effect of ventilatory inefficiency is thus to increase \(V_{D_{alv}}/V_{T_{alv}}\) with no obligatory effect on \(P_{A_{CO_2}}\), whereas circulatory inefficiency affects both \(V_{D_{alv}}/V_{T_{alv}}\) and \(P_{A_{O_2}}\). These primary effects may be accompanied by secondary effects from disturbances in \(V/Q\) distribution; if, for instance, ventilation is diverted to deadspace, some regions of low \(V/Q\) may be created. Similarly, if perfusion is diverted to a RL shunt, some regions of high \(V/Q\) (pulmonary hypoperfusion) may occur. The general effect of secondary effects is to add to the \(P_{A_{CO_2}} - P_{A_{CO_2}}\) difference created by primary defects, and this may be accompanied by some reduction in \(P_{A_{O_2}}\) (see below).

The occurrence of secondary effects depends on whether or not the ventilatory and circulatory inefficiencies are accompanied by overall respective increases in \(V\) or \(Q\). If there is deadspace ventilation, a compensatory increase in overall ventilation can prevent the secondary creation of low \(V/Q\) regions. Similarly, in RL shunting, a compensatory increase in total \(Q\) may prevent the appearance of regions of high \(V/Q\).

The children with RL shunts were younger and weighed less than those with normal circulation. Cyanotic children usually require surgery at an earlier age than those with non-cyanotic lesions, and is difficult to see how this age difference can be avoided in a clinical investigation. The smaller children may have a greater tendency to atelectasis [12], and in the present study, \(P_{A_{O_2}}\) increased with age in the acyanotic children. However, this should not greatly affect the results, as any reduction in \(P_{A_{O_2}}\) produced by RL shunting via atelectasis should be accompanied by an increase in alveolar deadspace.

The shape of the observed \(V_{D_{alv}}/V_{T_{alv}}\) v. \(P_{A_{O_2}}\) curve in children agrees well with the prediction (figs 2 and 3). The curve is approximately hyperbolic because the oxyhaemoglobin dissociation curve, which relates oxygen tension to content, is also approximately hyperbolic. (If \(V_{D_{alv}}/V_{T_{alv}}\) is plotted against oxygen saturation (or content), a straight line is obtained (Fletcher, unpublished results)). However, compared with the prediction, the observed \(V_{D_{alv}}/V_{T_{alv}}\) v. \(P_{A_{O_2}}\) curve is displaced upwards by approximately 0.04 at \(P_{A_{O_2}}\), 33.8 kPa (the mean value of the normal group), and by 0.07–0.08 at 7.3 kPa (RL group mean). At 5 kPa, the discrepancy increases further to approximately 0.10.

In the well-oxygenated children, the discrepancy between observed and expected \(V_{D_{alv}}/V_{T_{alv}}\) is small; some of it may result from systematic error which is produced when the Siemens–Elema 930 \(CO_2\) Analyzer is used at paediatric tidal volumes. This arises because the zero calibration is reset during each inspiration, when the transducer is flushed with fresh gas. Small tidal volumes fail to remove carbon dioxide completely. Because the new zero is established before the (logarithmic) carbon dioxide signal is linearized, carbon dioxide molecules remaining in the transducer at this stage have a disproportionately great effect. The zero error reduces the measured \(P_{CO_2}\) of the next expiration. Correction factors, obtained from two large series of observations [6], have been applied to \(V_{T_{CO_2}}\) and \(F_E\). If this factor should prove to be too small, values for \(V_{D_{alv}}\) in this study will have been systematically overestimated. In well oxygenated children, a 1% error in measurement of \(P_{A_{CO_2}}\) or \(P_{A_{CO_2}}\) gives an error of 0.01 in \(V_{D_{alv}}/V_{T_{alv}}\); in the cyanotic children, the effect is less.

Calculation of deadspace in the children (equation (10)) included insertion of a value for \(F_{TCO_2}\) arising from carbon dioxide rebreathed in the Y-piece. The effect of this on \(V_{D_{alv}}\) is less than 0.01, even in the smallest children. Other reasons for the discrepancy between observed and predicted \(V_{D_{alv}}/V_{T_{alv}}\) may be temporal deadspace or imperfect gas mixing. Temporal deadspace implies temporal mismatching of ventilation and perfusion. This may occur during positive pressure ventilation; pulmonary blood flow is maximal at
end-expiration, when ventilation is least. Some small degree of imperfect gas mixing cannot be ruled out, as even normal children have a slightly positive phase III slope. Finally, there is the possibility of secondary effects of ventilatory and, probably more importantly, circulatory inefficiency (see below).

In the children with RL shunts there is a very strong \( V_{D} / V_{T} \) ratio greater than normal. This was predicted. RL shunts are often regarded as causing "pulmonary hypoperfusion"—that is, inadequate pulmonary circulation giving rise to increased \( V/Q \) ratios greater than optimal. Such pulmonary hypoperfusion was discussed above as being a secondary effect of circulatory inefficiency. There is some evidence for hypoperfusion in the present observations, as the discrepancy between observed and expected \( V_{D} / V_{T} \) in these children increases somewhat with decreasing \( P_{A_{o}} \). However, cyanotic children have an increased cardiac output, and so areas of increased \( V/Q \) may perhaps be created only when the shunt is extremely large; for instance, a child with a shunt of 0.3 \((P_{A_{o}} = 7-8 \text{kPa})\) would have a normal pulmonary blood flow if its cardiac output was increased to 143% of normal. However, in order to confirm the existence of such secondary effects, it is necessary to compare pulmonary vein blood and alveolar gas carbon dioxide tensions in severely cyanotic children; this has not yet been done.

As a rule, children with LR shunting have excellent gas exchange. They differ from those with normal circulation only in having a slightly reduced \( P_{A_{o}} \) and an increased phase III slope; these variables being negatively correlated with each other. An increased phase III slope implies increased \( V/Q \) spread, which in turn implies an increase in \( V_{D} / V_{T} \) ratio. However, it is not possible to demonstrate any such increase in the present material; it is probably of too small a magnitude. Pulmonary artery \( P_{O_{2}} \) is increased in these children and, therefore, perfusion of low or zero \( V/Q \) areas does not affect \( P_{A_{o}} \) to the same extent as in normal children. Furthermore, perfusion of low \( V/Q \) areas does not necessarily imply under-perfusion of other areas, and therefore secondary effects are probably unimportant.

Much of the alveolar deadspace demonstrated in these children with congenital cardiac disease may thus be explained by RL shunting. The residual deadspace may result from several factors, including a possible systematic measurement error, temporal deadspace, and deadspace produced by imperfect gas mixing in the lung. In children with severe cyanotic disease, deadspace may be created also by secondary effects of the RL shunt—that is, failure of perfusion of the lung parenchyma.

The arterial-end-tidal carbon dioxide difference shows a relationship to \( P_{A_{o}} \) which is approximately similar to that of \( V_{D} / V_{T} \). However, \( (P_{A_{o}} - P_{E_{CO_{2}}}) \) is approximately zero for all \( P_{A_{o}} \) values of 10 kPa and greater, whereas \( V_{D} / V_{T} \) is approximately 0.15 at 10 kPa. The reason for this is that, as stated above, in the range of \( P_{A_{o}} \) greater than 10 kPa, phase III slope increases with decreasing \( P_{A_{o}} \); these two features being associated with increased pulmonary perfusion. A sloping phase III allows a zero arterial-end-tidal carbon dioxide difference, even when the alveolar deadspace is increased. This is seen occasionally in healthy adults undergoing ventilation with large tidal volumes [3].

Finally, figure 5 shows that there is no relationship between alveolar deadspace and oxygenation during anaesthesia in an adult general surgical population. This suggests that much of the alveolar deadspace is produced by primary ventilatory inefficiencies. There are probably also secondary effects. In general, increased alveolar deadspace in adults is the result of airways disease, which causes an increased spread of \( V/Q \) ratios and an increase in phase III slope. Phase III slope in a middle-aged individual is usually much steeper than that seen in children.

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**REFERENCES**


