ANALYSIS OF FACTORS WHICH AFFECT THE RELATIONSHIP BETWEEN INSPIRED OXYGEN PARTIAL PRESSURE AND ARTERIAL OXYGEN SATURATION

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SUMMARY

The adoption of pulse oximetry in anaesthesia and intensive care has resulted in oxyhaemoglobin saturation ($S_{aO_2}$) becoming an important and widely used clinical descriptor of gas exchange, although its full potential has not been realized. We have explored the effect of impaired gas exchange on the relationship between $P_{iO_2}$ and $S_{aO_2}$. A curve of oxygen content vs $P_{iO_2}$ for an ideal lung was constructed using the oxygen dissociation curve and allowing for the effect of dissolved oxygen in plasma. A plot of $P_{iO_2}$ vs $S_{aO_2}$ was derived from this content curve. The effect of impaired gas exchange (shunt and $V/Q$ effects) on this relationship was then modelled using for shunt the equation $\dot{Q}_s/\dot{Q}_t = (C'_{ca} - C_{vo})/(C'_{ca} - C_{vo})$ and for $V/Q$, a graphical method using the equation $V/Q = (P_{iO_2} - P_{aO_2})/(P_{iO_2} - P_{aO_2})$ and the oxygen content curve to model the effect of a spread of $V/Q$ ratios. A total of nine compartments were used to model the scatter of $V/Q$, and the size of these compartments were determined by their relative blood supply. Plots of $P_{iO_2}$ vs $S_{aO_2}$ were derived for different values of shunt and $V/Q$. The $P_{iO_2}$ vs $S_{aO_2}$ curve reflected the shape of the oxygen dissociation curve but lay to the right by $P_{iO_2} - P_{aO_2}$. Shunt caused a depression of the plateau of this curve with relatively little lateral movement, whereas a low mean $V/Q$ ratio caused a marked rightwards lateral displacement. Increased log $Q$ caused a flattening of the relationship. The combined effect of shunt and a spread of $V/Q$ ratios caused both lateral and vertical displacements. Thus a series of simple measurements of $P_{iO_2}$ and $S_{aO_2}$ gave information about both shunt and $V/Q$ abnormality.

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KEY WORDS

Oxygen: oxyhaemoglobin saturation, tension.

In an accompanying paper [1] we showed that the curve relating inspired oxygen partial pressure ($P_{iO_2}$) and oxyhaemoglobin saturation ($S_{aO_2}$) varies considerably in the perioperative period, and that when $P_{iO_2}$ lies on the steep part of the curve the particular patient is at increased risk of postoperative hypoxaemia and likely to display an unstable pattern of $S_{aO_2}$. The position and shape of this curve is likely to be altered by factors causing impaired gas exchange and hence it could be used as a non-invasive indicator of such factors, and a predictor of postoperative hypoxaemia. However, there has been no published analysis on how the curve may be affected by such changes. The purpose of this paper is to examine the effect of impaired gas exchange on the $P_{iO_2}$ vs $S_{aO_2}$ relationship using a theoretical model which incorporates shunt and distributions of ventilation/perfusion ratios ($V/Q$).

Various models have been described in an attempt to understand the physiology of impaired gas exchange. One is that of Riley [2], in which the lung is described in terms of three compartments: shunt, ideal alveoli and deadspace. In this model, units of different ventilation/perfusion ratio ($V/Q$) are rationalized into varying contributions to the three compartments. Using the Riley model, a value for shunt can be calculated for a given $V/Q$ abnormality. However, it has also been suggested that the amount of shunt attributable to a spread of $V/Q$ varies with $P_{iO_2}$ [3, 4]. Multiple inert gas elimination techniques have been used to estimate $V/Q$ distribution scatter [5] and we have used distributions similar to those derived using this technique to illustrate the working of our model.

THEORETICAL ANALYSIS

We have developed a simple model of $V/Q$ scatter involving nine compartments: true shunt ($V/Q = 0$), spread of $V/Q$ ratios (0.1, 0.3, 0.5, 1, 2, 4, 10) and deadspace ($V/Q = \infty$). This modelling in terms of true shunt, $V/Q$ distribution and deadspace uses compartments (albeit fewer) similar to those used in multiple inert gas elimination techniques. The size of each compartment in the model is variable and is determined by the percentage of the total blood flow to that compartment. When using data from multiple inert gas studies, the multiple compartments were rationalized into those of our model.

We assumed the following values in the model: haemoglobin concentration 15.27 g dl$^{-1}$; oxygen combining power of haemoglobin 1.31 ml g$^{-1}$; physical solution of oxygen in blood 0.0225 ml dl$^{-1}$ kPa$^{-1}$; dry barometric pressure 93.7 kPa, cardiac output 5 litre min$^{-1}$; oxygen consumption 250 ml min$^{-1}$. This results in an arterial to mixed venous oxygen...
content difference of 50 ml litre⁻¹. We based our oxyhaemoglobin dissociation curve on the tables of Kelman and Nunn [6].

### The $P_{to}$ vs $S_{ao}$ relationship

The oxygen dissociation curve gives the relationship between arterial oxygen partial pressure ($P_{ao}$) and arterial oxyhaemoglobin saturation ($S_{ao}$). In an "ideal" lung in which all alveoli have a $V/Q$ ratio of 1 and without any shunt, diffusion defect or alveolar deadspace, the relationship between alveolar oxygen partial pressure ($P_{ao}$) and $S_{ao}$ corresponds exactly to the shape and position of the oxygen dissociation curve. If one then considers the position of the relationship between inspired oxygen partial pressure ($P_{ao}$) and $S_{ao}$, this curve would also correspond to the shape of the oxygen dissociation curve but would lie to the right by an amount corresponding to the $P_{to} - P_{ao}$ gradient. The net result is the curve shown in figure 1; we refer to this curve as the "ideal" $P_{to}$ vs $S_{ao}$ relationship.

### Factors affecting the ideal $P_{to}$ vs $S_{ao}$ relationship

The oxygen dissociation curve. Any factor which affects the position of this curve (pH, carbon dioxide, temperature, 2,3-diphosphoglycerate, carboxyhaemoglobin) will cause a corresponding change in the position and shape of the $P_{to}$ vs $S_{ao}$ relationship [6].

As carbon dioxide and oxygen exchange are closely related, $P_{aco}$ is altered by gas exchange impairment that causes hypoaxaemia. This leads to Bohr effects causing a rightwards shift of the oxygen dissociation curve with increased $P_{aco}$. In addition, blood from low $V/Q$ units will have a greater $P_{aco}$, than blood from high $V/Q$ units. This produces a Bohr shift in the blood returning from low $V/Q$ units which is related to the magnitude of the gas exchange abnormality. The magnitude of the error introduced by not incorporating the effect of carbon dioxide exchange is very small (see Appendix 1).

### The $P_{to} - P_{ao}$ gradient

This is given by the alveolar gas equation in the form:

$$P_{to} - P_{ao} = P_{bar} \times \dot{V}_{o_2} / \dot{V}_A$$

(1)

Using the assumed values for $P_{bar}$ and $\dot{V}_{o_2}$, and with $\dot{V}_A = 4000$ ml:

$$P_{to} - P_{ao} = 5.86 \text{ kPa}$$

Any factor which increases this gradient causes a translocation of the curve to the right as an increased $P_{to}$ is required to produce a given $P_{ao}$. Increased oxygen consumption or reduced alveolar ventilation produces this effect.

### Hypoventilation

From equation (1) it can be seen that, for given $\dot{V}_{o_2}$, if $\dot{V}_A$ is reduced then $P_{to} - P_{ao}$ is increased. For the whole lung, it can be seen that hypoventilation behaves as a reduction of the $V/Q$ ratio of all units and produces a displaced $P_{to}$ vs $S_{ao}$ relationship. Figure 1 shows the ideal curve with a series of curves showing the shift produced by hypoventilation.

### Abnormalities of gas exchange

The effect of true shunt and $V/Q$ distribution abnormality are analysed separately.

### True shunt

Consider the effect of shunt in the absence of $V/Q$ distribution abnormality. In this situation, one may think in terms of a two-compartment model of true shunt and ideal alveoli. One may determine the effect of a shunt on the position of the $P_{ao}$ vs $S_{ao}$ relationship by calculating the resulting oxygen content when mixing end-capillary blood ($= P_{ao}$) with mixed venous blood in the proportion of the shunt at varying values of $P_{to}$. The well known shunt equation is used:

$$\frac{\dot{Q}_s}{\dot{Q}_t} = \frac{(C_{o_2} - C_{a_2})}{(C_{o_2} - C_{v_2})}$$

(2)

Arterial to mixed venous oxygen content difference is assumed to be 50 ml litre⁻¹ and this is incorporated into equation (2) by expressing it in the form:

$$C_{a_2} = C_{o_2} - \dot{Q}_s/\dot{Q}_t \times (C_{a_2} - C_{v_2})/(1 - \dot{Q}_s/\dot{Q}_t)$$

(3)

Repeating this procedure with varying degrees of shunt results in a series of oxygen content lines each displaced from the ideal line (zero shunt) by a factor of $(C_{o_2} - C_{v_2})/(1 - \dot{Q}_s/\dot{Q}_t)$. Converting to oxyhaemoglobin saturation results in virtual shunt isopleths as shown in figure 2. These are plotted as $P_{to}$ vs $S_{ao}$ and assume a $P_{to} - P_{ao}$ gradient of 5.86 kPa.

### $V/Q$ mismatch

Consider the effect of $V/Q$ mismatch in the absence of true shunt. The $V/Q$ ratios of alveoli in the lung are scattered and in theory could range from 0 to infinity. Estimations of the spread of $V/Q$ ratios in the lung using multiple inert gas elimination techniques [5] give results such as those illustrated in figure 3b [7]. This gives the blood flow and ventilation to units of differing $V/Q$ values; each point represents a different compartment (43 in total). In our model we have reduced the number of compartments to nine, each having a mean $V/Q$ of 0, 0.1, 0.3, 0.5, 1, 2, 4, 10 and infinity. The size of each compartment is dependent upon its blood supply and may be zero. When a particular distribution is applied to our model, the various points are assigned to the nearest compartment and
FIG. 2. Inspired oxygen partial pressure vs oxyhaemoglobin saturation, showing the calculated effect of different degrees of virtual shunt in accordance with the assumed model values given in the text. $P_{ti} - P_{A_{02}} = 5.86 \text{kPa}$.

FIG. 3. A: Oxygen content (—) and oxyhaemoglobin saturation (——) vs $P_{O_2}$ for an ideal lung and a lung with the $V/Q$ abnormality shown in figure 5B. B: Compartmental distribution of ventilation [□] and blood flow (●) with respect to ventilation–perfusion ratio. Results similar to this are obtained using the multiple inert gas elimination technique. (Redrawn from Dueck [7].)

FIG. 4. Diagrammatic representation of alveolar ventilation and perfusion showing how each can be represented by a Fick equation (see text).

FIG. 5. A: Oxygen partial pressure vs oxygen content assuming a haemoglobin concentration of 15.27 g litre$^{-1}$. The graphical solution to equation (5) is shown. (See text for discussion.) B: For given $P_{ti}$ and $C_vO_2$, a series of lines of differing gradient, representing the solution of equation (6) for varying $V/Q$, can be drawn. The intersection of these lines with the oxygen content curve give the end-capillary content of compartments of differing $V/Q$. Note that the horizontal line for zero $V/Q$ meets the ordinate at $C_vO_2$. In this example the $V/Q$ lines are set for $P_{ti}(Ji30 \text{kPa}$ and $C_vO_2 150 \text{ml litre}^{-1}$.

To ascertain the contribution of each compartment to the oxygen content of arterial blood, it is necessary to understand how the $P_{ti} - P_{A_{02}}$ gradient at any given $V/Q$ alters with $P_{ti}$. The diagram in figure 4 describes the dynamics of oxygen uptake from an alveolus into a capillary. Using the Fick principle it can be seen that, for the alveolar component (A):

$$P_{ti} - P_{A_{02}} = k \frac{V_{O_2}}{V_A}$$

Similarly for the capillary component (B):

$$C_cO_2 - C_vO_2 = \frac{V_{O_2}}{Q}$$

Combining these two equations in terms of $V_A/Q$:

$$V_A/Q = k(C_cO_2 - C_vO_2)/(P_{ti} - P_{A_{02}})$$
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This is therefore proportional to $V/Q$. When content is expressed in ml litre$^{-1}$ and partial pressure in kPa, the coefficient of proportionality is 0.113 [8]. A series of these lines may be constructed of differing gradients representing differing $V/Q$ values. They can be placed over a content curve (using a transparent overlay) for any values of $P_{tO_2}$ and $CvO_2$ and the intercept of the line with the oxygen content curve gives $C_{aO_2}$ for that $V/Q$ compartment. Figure 5B shows this for $P_{tO_2}$ 30 kPa and $CvO_2$ 150 ml litre$^{-1}$. This method was used to calculate the contribution of differing $V/Q$ compartments at differing $P_{tO_2}$ values. As the proportion of pulmonary blood flow to each compartment is known, from figure 5 the relative contributions of each compartment to the final $C_{aO_2}$ may be read and a weighted mean calculated; this is then converted to $S_{aO_2}$. This process can be repeated at different values of $P_{tO_2}$ to construct a curve of $P_{tO_2}$ vs $S_{aO_2}$ in the presence of any given spread of $V/Q$ mismatch (see Appendix 2 for details of working).

The arterial-to-mixed venous oxygen extraction of 50 ml litre$^{-1}$ must be held constant when the above graphical method is used. For each calculation, $CvO_2$ is estimated and the resulting $CaO_2$ calculated. If the result is such that $CaO_2 - CvO_2$ is not equal to 50 ml litre$^{-1}$, $CvO_2$ is re-estimated and the process repeated. We accepted a tolerance of ±2 ml litre$^{-1}$.

The effect of an abnormal $V/Q$ distribution can be considered in terms of the effect of mean $V/Q$ and the effect of $V/Q$ distribution. A lung with $V/Q = 1$, with little or no distribution about this value, gives an ideal $P_{tO_2}$ vs $S_{aO_2}$ curve. Modelling the effect of increasing log $SDQ$ with constant mean $V/Q = 1$ results in the curves shown in figure 6 (a flattening of the knee of the curve). Alterations in mean $V/Q$ to smaller values results in much larger movements of the curve to the right, as the effect is to increase the value of $P_{tO_2} - PaO_2$. Figure 7 shows a series of curves of different mean $V/Q$ using the above model with 100% of the blood flow through that particular compartment. These simply show the magnitude of the shift which occurs with alterations in mean $V/Q$ alone. They are quite hypothetical, as $V/Q$ is never limited to one value, but occurs as a wide scatter. The combination of low $V/Q$ (shift) and scatter of $V/Q$ (flattening) is seen in figure 3 using data obtained from a patient [7].

**Combined effect of $V/Q$ mismatch and shunt.** The effect of shunt is easily modelled by adding the shunt component to the compartment of $V/Q = 0$ in the above model.
We have described the relationship between $P_{1O_2}$ and $S_{A_{O_2}}$ and analysed the effect of various abnormalities of gas exchange on the shape and position of this relationship. The salient findings of the analysis are as follows.

1. The $P_{1O_2}$ vs $S_{A_{O_2}}$ curve reflects the shape of the oxygen dissociation curve, but lies to the right by the $P_{1O_2} - P_{A_{O_2}}$ difference (fig. 1).

2. Any factor which alters the position of the oxygen dissociation curve itself, or alters the value of $P_{1O_2} - P_{A_{O_2}}$, alters the degree of this lateral movement also.

3. The effect of true shunt considered alone is to produce the series of curves shown in figure 2, with a predominantly vertical depression of the plateau of the $P_{1O_2}$ vs $S_{A_{O_2}}$ curve and relatively little lateral movement. The slope of the plateau which occurs on these shunt isopleths is caused by increasing oxygen content of non-shunted blood, derived from increased dissolved oxygen at greater values of $P_{1O_2}$.

4. The effect of an increasing abnormal scatter of $V/Q$ ratios about a widening mean (increasing $log_{10} Q$) is to produce a flattening of the knee of the curve (fig. 6).

5. The effect of a reduced mean $V/Q$ ratio, as could be produced by changes in alveolar ventilation or cardiac output, is a marked rightwards lateral displacement of the relationship as a consequence of an increase in the $P_{1O_2} - P_{A_{O_2}}$ difference (fig. 7).

6. The combined effect of scatter of $V/Q$ ratios and reduced mean $V/Q$ is to produce a laterally displaced curve with flattening (fig. 8).

Riley and Courmand, in 1949 [2], described a three-compartment model of the lung involving shunt, ideal alveoli and deadspace. Using this model in its original form, the Dueck data (fig. 3b) would be resolved into a compartment of shunt and a compartment of ideal alveoli. This could then be incorporated into the multicompartmental model described in this paper by setting the size of all compartments to zero except $V/Q = 0$ (shunt) and $V/Q = 1$ (ideal alveoli). The resultant $P_{1O_2}$ vs $S_{A_{O_2}}$ curve would show a shunt effect, but would have no shift and no flattening. Modifying the model to include shunt, low $V/Q$ and high $V/Q$ compartments would improve the resolution, but the shape of the $P_{1O_2}$ vs $S_{A_{O_2}}$ curve would be dependent upon the position and size of these $V/Q$ compartments. Multiple inert gas determination of $V/Q$ distribution used a large number of compartments in order to describe the position and size of these distributions. Any number of compartments can be chosen to model the spread of $V/Q$ ratios, but we chose seven in addition to shunt ($V/Q = 0$) and deadspace ($V/Q = infinity$), as these enabled a reasonable sample of $V/Q$ ratios to be distinguished over the range in which published $V/Q$ ratios tend to occur over a log scale. They enable the position and spread of the $V/Q$ distribution to be modelled with sufficient accuracy to demonstrate the effects that these abnormalities have on the $P_{1O_2}$ vs $S_{A_{O_2}}$ curve. An infinite number of compartments would be required ideally to produce an accurate model of scatter. With fewer than nine compartments, the important effect of wide $V/Q$ scatter is progressively lost and, with more, little is gained in this respect but the model becomes increasingly cumbersome. Using the three-compartment model of Riley and Courmand [2], the flattening effect of an increased spread of $V/Q$ ratios would not be seen at all.

The use of the multiple inert gas technique for estimation of $V/Q$ results in a narrow peaked distribution in young healthy volunteers [9]. Widening of the $V/Q$ distribution (greater $log_{10} Q$) has been reported in middle-aged subjects under anaesthesia [10]. Much larger widening has been reported in aged patients with pulmonary disease [7]. A wide $V/Q$ distribution redrawn from the latter study is shown in figure 3b.

Figure 6 shows how increasing $V/Q$ mismatch (increased $log_{10} Q$) affects the relationship with a flattening of the knee of the curve, even without alterations in mean $V/Q$.

The effect of abnormal $V/Q$ distributions on oxygen content as derived from the graphical model described above is shown in figure 6. Note how the difference in content between blood from an ideal lung and blood from a lung with a $V/Q$ abnormality is small at large values of $P_{1O_2}$ and becomes increasingly large at smaller $P_{1O_2}$ values. This is in contrast with the content difference of blood from an ideal lung and blood from a lung with true shunt, which remains constant at varying $P_{1O_2}$ values. The increased content difference at small $P_{1O_2}$ with a $V/Q$ abnormality occurs because, as $P_{1O_2}$ is reduced, content in units of low $V/Q$ begins to occur on the steep part of the content curve, hence reducing $C_{A_{O_2}}$.

The corresponding $P_{1O_2}$ vs $S_{A_{O_2}}$ curves are shown also and illustrate the flattening displacement which occurs with this abnormality.

A reduction in mean $V/Q$ results in a rightwards displacement. This may be seen from equation (6) in which, for a constant $(P_{A_{CO_2}} - P_{A_{O_2}})$, a decrease in $V/Q$ will involve an increased $P_{1O_2} - P_{A_{O_2}}$ difference. This assumption of constant $(P_{A_{CO_2}} - P_{A_{O_2}})$ implies that these are “virtual” shunt and $V/Q$ model lines.

With small values of $P_{1O_2}$ there are confounding effects not otherwise taken into account in the model described. An abnormal $V/Q$ scatter which causes an impairment of oxygenation also causes an impairment of carbon dioxide transfer and an increase in $P_{A_{CO_2}}$ occurs. This would tend to exacerbate a rightwards shift as increased $P_{A_{CO_2}}$ causes a shift of the oxygen dissociation curve to the right by the Bohr effect. A gas exchange abnormality of the magnitude of that shown in figure 3b would result in a $P_{A_{CO_2}}$ of 5.47 kPa if $V/A$ remained constant. The Bohr shift occasioned by this would cause a further rightwards shift of approximately 0.06 kPa. However, this effect will be offset "in vivo" by an increase in $V/A$ causing an increase in $V/A_{Q}$ and decrease in $P_{A_{CO_2}}$.

When $P_{1O_2}$ is altered clinically, many other factors also influence the effect of a given $V/Q$ abnormality. Cardiac output would be unlikely to remain constant at progressive degrees of hypoxaemia and an increase would decrease the overall $V/Q$, causing a leftwards shift. Hypoxaemia is also often accompanied by
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progressive hyperventilation, which also alters overall $V/Q$ and causes an increasing rightwards shift. All of these factors could change at different $P_{O_2}$ during the construction of a $P_{O_2}$ vs $S_{A\O_2}$ curve, in which case the in vivo curves would deviate from the model curve. This does not invalidate the model, as a new curve can be constructed for any given value of pH, carbon dioxide, cardiac output and alveolar ventilation. What occurs is that the patient's $S_{A\O_2}$ moves from one model curve to another as various factors assumed constant for the construction of the original curve are changed.

It can be seen that the exact position of the steep part of the curve is altered by many factors which alter in the perioperative period. Alterations in overall $V/Q$ probably are the most important factors, as they can bring the steep part of the curve into the clinical range (fig. 7).

In figures 2, 3, 6 and 7, $P_{O_2}$ is shown on the x axis at values up to 100 kPa. This is to make clear the mathematical working of the model and to show how shunt and $V/Q$ differ in their effects at increased $P_{O_2}$. In practice, however, there will be confounding factors at increased $P_{O_2}$. Absorption atelectasis and reversal of hypoxic pulmonary vasoconstriction are both likely to alter the position of these curves at $P_{O_2}$ values > 80 kPa.

The widespread adoption of pulse oximetry has made the measurement of oxygen saturation simple and non-invasive. Knowledge of the $P_{O_2}$ vs $S_{A\O_2}$ relationship and the mechanisms underlying changes therein are important in interpreting the significance of single $S_{A\O_2}$ values. Before pulse oximetry was available, it was commonplace to judge oxygen requirements by measuring $P_{A\O_2}$ at a known $P_{O_2}$ and reading the degree of shunt from a series of isopleths [11]. This could be done similarly using shunt isopleths expressed in terms of saturation (fig. 2). However, our earlier data [1, 12] showed a marked deviation from these isopleths at smaller values of $P_{O_2}$. It had been suggested previously that the degree of shunt attributable to a given distribution of $V/Q$ increases as $P_{O_2}$ is reduced [3, 4]; this was confirmed by our model and was manifest in the $P_{O_2}$ vs $S_{A\O_2}$ curves as a lateral displacement and flattening of the steep part of the curve. This is of clinical significance as the steep part of the curve may move into the clinical range. In the study by Lawler and Nunn [13], some patients showed increased measured virtual shunt at smaller $P_{O_2}$. This could be explained by the effect of a $V/Q$ abnormality at smaller values of $P_{O_2}$. $P_{O_2}$ vs $S_{A\O_2}$ diagrams are sensitive to the difference in behaviour of true shunt and $V/Q$ maldistribution at varying $P_{O_2}$.

We have not attempted to quantify plateau depression and shift in this paper. Any attempt to do so using curves obtained from patient data must take into account the interactions of the various patterns seen. Shunt (plateau depression) is best revealed as the depression in $S_{A\O_2}$ at $P_{O_2}$ 50 kPa, as here the effects of small mean $V/Q$ (shift) and a large $\log_{10} Q$ (flattening) are minimalized (figs 6, 7). The effect of $V/Q$ abnormalities (mean $V/Q$ and $\log_{10} Q$) are absent at $P_{O_2}$ 100 kPa, but here the effect of shunts less than 30% are harder to ascertain from saturation readings. Low mean $V/Q$ (shift) is best revealed at $S_{A\O_2}$ 80%, as here the effect of shunt and large $\log_{10} Q$ are also minimized (figs 2, 6). This would be difficult to achieve clinically in most patients without the administration of subatmospheric partial pressures of oxygen. However, an indication of the low $V/Q$ effect (shift) may be obtained by plotting a series of $S_{A\O_2}$ values over a wide range and detecting deviation from the shunt isopleth with smaller $P_{O_2}$.

In conclusion, measurement of the $P_{O_2}$ vs $S_{A\O_2}$ relationship is a simple, non-invasive extension of the use of pulse oximetry. It gives useful information regarding both the aetiology of hypoxaemia and the likely behaviour of oxygen saturation in the perioperative period. Reference to the oxygen dissociation curve alone when interpreting oxygen saturation results can be very misleading, as the position of the $P_{O_2}$ vs $S_{A\O_2}$ relationship may alter widely in the perioperative period.

APPENDIX 1

Carbon dioxide exchange is related to $V/Q$ by the following equation, which is derived using the same principles as equation (6):

$$\dot{V}_A/\dot{Q} = (C_{CO_2} - C_{A\O_2})/P_{A\O_2}$$  \(7\)

The solution to this equation is also by the graphical method, but using the carbon dioxide dissociation curve. Figure 8 (reproduced from Fahri [8]) shows how $P_{A\O_2}$ and $C_{A\O_2}$ vary with $V/Q$. Table 1 shows the effect of reducing $V/Q$ from 1 to 0.5 on $P_{A\O_2}$ (assuming [8] that $P_{A\O_2} = P_{A\O_2}$ at low $V/Q$). The effect on pH, derived using a Siggaard-Andersen nomogram is shown, and the effect that the consequent Bohr shift will have on a $P_{A\O_2}$ of 6 kPa (chosen as this is on the steep part of the oxyhaemoglobin dissociation curve).

<table>
<thead>
<tr>
<th>$P_{A\O_2}$ (kPa)</th>
<th>5.07</th>
<th>5.47</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.40</td>
<td>7.39</td>
</tr>
<tr>
<td>$P_{A\O_2}$ (kPa)</td>
<td>6</td>
<td>5.94</td>
</tr>
</tbody>
</table>
ILLUSTRATION OF THE WORKING OF THE MODEL

The Dueck data (fig. 3B) have been rationalized into the following compartments (table II).

<table>
<thead>
<tr>
<th>Blood flow (%)</th>
<th>0</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>V/Q = 0</td>
<td>100</td>
<td>90</td>
<td>80</td>
<td>70</td>
<td>60</td>
<td>50</td>
<td>40</td>
<td>30</td>
<td>20</td>
<td>10</td>
<td>0</td>
</tr>
</tbody>
</table>

To estimate $S_{a}O_{2}$ at $P_{O_{2}} 25$ kPa

Mixed venous oxygen content is estimated at 110 ml litre$^{-1}$. Using the transparency of figure 5B over the oxygen content curve: for a mixed venous oxygen content 110 ml litre$^{-1}$ the line of $V/Q = 0$ on the transparency is set at 110 on the ordinate and for $P_{O_{2}} 25$ kPa the line of $V/Q = \infty$ on the transparency is set at 25 on the abscissa. The intersection of the $V/Q$ lines on the transparency with the oxygen content curve gives the end-capillary oxygen content of blood leaving each compartment (table III).

<table>
<thead>
<tr>
<th>$V/Q$</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>4</th>
<th>10</th>
<th>$\infty$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$C_{c}O_{2}$ (ml litre$^{-1}$)</td>
<td>125</td>
<td>158</td>
<td>180</td>
<td>196</td>
<td>198</td>
<td>200</td>
</tr>
</tbody>
</table>

A weighted mean of these $C_{c}O_{2}$ values is calculated using the percentage of the total blood flow to each compartment as the weighting. For the above data this is 161.6 ml litre$^{-1}$, giving an oxygen saturation of 80.4%. Note how this gives an arterial-to-mixed venous oxygen content difference of 51.6 ml litre$^{-1}$ which is acceptable, as our tolerance is 48-52 ml litre$^{-1}$. If it had been outside this range, $C_{v}O_{2}$ would have to be re-estimated and the above working repeated. The above process is repeated over a wide range of $P_{O_{2}}$ values to construct a curve of $P_{O_{2}}$ vs $S_{a}O_{2}$.

REFERENCES