A Nomogram for Planning Respiratory Therapy*

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A nomogram is presented which aids in monitoring and selecting therapy in the treatment of the patient with hypoxemia. It facilitates accurate bedside determination of total shunt fraction and estimation of the arterial oxygen tension (PaO₂) attainable at inspired oxygen concentrations of 21 percent to 100 percent. The nomogram permits rapid consideration of changes in hemoglobin concentration, arteriovenous oxygen content difference, and shunt fraction on the PaO₂. The uses of the nomogram in several therapeutic contexts are discussed.

Over the past 20 years the development of methods for quantifying relationships between ventilation and perfusion within the lung has used the concept of shunt fraction (Qs/Qt). True shunt is defined as the fraction of cardiac output passing through unventilated portions of the lung. A decrease in the arterial oxygen pressure (PaO₂) can be due to perfusion of unventilated alveoli (true shunt), perfusion of underventilated alveoli (VA/Q abnormality), or defects in diffusion. Total wasted blood flow or physiologic shunt is the equivalent fraction of cardiac output which, if going to unventilated alveoli, would cause a given decrease in PaO₂. In this report, unless otherwise noted, Qs/Qt refers to total venous admixture due to wasted blood flow.

The alveolar-arterial oxygen pressure difference (P[A-a]O₂) and PaO₂, which are often used as indicators of Qs/Qt, are influenced by several variables that do not directly reflect pulmonary function. These include the concentration of hemoglobin, the concentration of oxygen in the inspired gas (FIO₂), the arteriovenous oxygen content difference (C(a-v)O₂), and the position of the oxyhemoglobin dissociation curve, which, in turn, is most often affected by changes in pH. Alteration in one or more of these variables occurs in patients with acute respiratory failure; thus, a change in the P(A-a)O₂ or PaO₂ does not necessarily indicate a corresponding change in pulmonary function. This report presents a nomogram which permits rapid determination of Qs/Qt for any value of hemoglobin level, FIO₂, and C(a-v)O₂, and position of the oxyhemoglobin dissociation curve.

The delay in use of this approach in planning therapy can be attributed largely to the complexity of the equations. The shunt equation

\[ \frac{Q_s}{Q_T} = \frac{C_aO_2 - C_vO_2}{C_aO_2 - C_rO_2} \]

(where C_aO₂ is the oxygen content of the patient's arterial blood, C_vO₂ is the oxygen content of blood in equilibrium with oxygen, and C_rO₂ is the oxygen content of the patient's mixed venous blood) can be expanded and rearranged to show:

\[ 0.0031 \text{ PaO}_2 = 1.39 \text{ Hb (S}_O_2 - \text{ SaO}_2) + 0.0031 \text{ PaO}_2 - \frac{Q_s}{Q_T} \cdot C(a-v)O_2 \]

where Hb is the concentration of hemoglobin, PaO₂ is the ideal alveolar oxygen pressure in millimeters of mercury for a given FIO₂, SaO₂ is the oxygen saturation of the patient's arterial blood, and S_vO₂ is the calculated saturation of hemoglobin in ideal alveolar capillary blood. Since S_O₂ and SaO₂ are complicated nonlinear functions of PaO₂ and PaO₂, respectively, accurate estimation of the effect on PaO₂ of changes in the concentration of hemoglobin, the FIO₂, the C(a-v)O₂, or the position of the oxyhemoglobin dissociation curve (affecting S_O₂ and SaO₂) is impracticable without a computer.

CONSTRUCTION OF THE NOMOGRAM

Values for PaO₂ were calculated from the ideal alveolar gas equation, assuming a barometric pressure of 760 mm Hg, a respiratory quotient of 0.8, and an arterial carbon dioxide tension (PaCO₂) of 40 mm Hg. Using PaO₂, values for PaO₂ were calculated on a computer by solving

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the shunt equation and the equations given by Kelman for the standard oxyhemoglobin dissociation curve (at a temperature of 37°C, a pH of 7.40, and a PaCO₂ of 40 mm Hg). These values for PaO₂ were plotted on lines corresponding to different combinations of FiO₂ and the hemoglobin concentration with respect to the FiO₂ of 1.00 and hemoglobin concentration of 11.0 gm/100 ml, which were arbitrarily used as the reference axis for the nomogram. The nomogram (Fig 1) has been constructed so that only horizontal lines perpendicular to the vertical axes need be drawn; there is no need for angular alignment.

**DESCRIPTION OF THE NOMOGRAM**

The nomogram has been divided into the following three parts: (1) the left-hand section is used to adjust the relationship between PaO₂ and Qs/QT for various values of C(a-v)O₂; (2) the middle section shows, for a given Qs/QT and C(a-v)O₂, the effect on PaO₂ of changes in FiO₂ and the hemoglobin concentration; and (3) the lines on the extreme left and right are used for the accurate construction of horizontal lines to interrelate the two major parts of the nomogram.

Under each value of FiO₂ are three vertical lines labeled 7, 11, and 15 to represent values for hemoglobin expressed in grams per 100 milliliters. On these lines are plotted values of PaO₂. Given values of PaO₂ are connected by isopleths. The scale shown both at top and bottom can be used as a guide to add lines equivalent to values for hemoglobin levels other than 7, 11, and 15 gm/100 ml. The isopleths for PaO₂ are used to determine values for PaO₂ for these hemoglobin levels.

To use the nomogram, the following four steps should be followed: (1) locate the FiO₂ nearest the patient's actual FiO₂; (2) under this FiO₂, locate the vertical line for a hemoglobin level nearest the value for the patient, or draw one corresponding to the patient's hemoglobin; (3) locate on the line for the hemoglobin level the patient's measured PaO₂; and (4) through the point located as step 3, draw a line parallel to the horizontal axis of the nomogram, using the scales on the extreme left and right to be sure the line is parallel with the horizontal scale at top and bottom. The constructed line is used for the following:

First, the Qs/QT may be determined for known or assumed C(a-v)O₂. Find a vertical line at the left of the nomogram corresponding to the patient's measured or assumed C(a-v)O₂ where the horizontal line intersects this line for C(a-v)O₂ and read the value of the patient's Qs/QT.

Secondly, the constructed line may be used to predict the PaO₂ attainable at the different values of FiO₂ for constant Qs/QT and C(a-v)O₂. Read along the horizontal line under any chosen value of
for FIO2 the PaO2 on the line for the hemoglobin level appropriate to the patient. The user can also pick a desired PaO2 where the horizontal line crosses the patient's line for hemoglobin level and read the value for FIO2 needed to achieve this PaO2. It also, of course, allows the user to estimate the effect of a given change in FIO2.

Thirdly, the constructed line may be used to determine the effect of change in C(a-v)O2 on PaO2. Using the patient's or an assumed C(a-v)O2, locate the point corresponding to the patient's Qs/QT. A horizontal line through this point permits reading of PaO2 for any FIO2, as described previously. Then repeat the process by drawing a second horizontal line through the same value for Qs/QT on another line for C(a-v)O2.

A fourth use of the constructed line is to determine the effect of change in Qs/QT on PaO2. On the appropriate line for C(a-v)O2, locate points corresponding to various values for Qs/QT, and draw horizontal lines through these points. Values of PaO2 that would result from changes in Qs/QT can be read under any FIO2 on the appropriate line for hemoglobin level.

Fifth, the constructed line may be used to determine the effect of changes in C(a-v)O2 on Qs/QT. Read across the horizontal line the Qs/QT on the various lines for C(a-v)O2.

Sixth, C(a-v)O2 can be estimated from values for PaO2 and PfO2 as follows: Draw a horizontal line through the value for the patient's PaO2 as described previously. On the same line for hemoglobin level, locate the value of the patient's venous oxygen pressure, and draw a horizontal line through this point. Using the scale shown both at the extreme right and left, read the values at which the lines cross the scale. The absolute difference between values is the C(a-v)O2. Use of the nomogram for calculating C(a-v)O2 has potential for serious error because the steep slope of the oxyhemoglobin dissociation curve in the region in which the mixed venous oxygen pressure usually falls makes calculation of venous oxygen content from measured oxygen pressure inexact. Small errors in measurement of venous oxygen pressure and pH, combined with small shifts in location of the oxyhemoglobin dissociation curve, can result in large errors in estimating oxygen content. The additional error due to the approximate nature of the adjustment for pH when oxygen pressures are less than 40 mm Hg and the small inaccuracy due to possible errors in alignment in use of the nomogram make estimation of the C(a-v)O2 inexact. Estimation is best restricted to classifying the C(a-v)O2

![Ventilation-perfusion nomogram with lines drawn in as examples for using nomogram (see text).](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20996/ on 04/15/2017)
as large, average, or small. Clinically, such estimation of relative magnitude may be sufficient for narrowing the determination of $Q_s/Q_T$ to a small interval or for gauging the degree of extraction of oxygen by the tissues.

For values for hemoglobin levels above 15 gm/100 ml, the value can be interpolated by drawing extra lines using the scales to the right of lines for hemoglobin level of 15 gm/100 ml. Then the $PaO_2$ must be extrapolated. At low values of $PaO_2$, this extrapolation becomes increasingly inaccurate. Such a line is drawn in for a hemoglobin level of 17 gm/100 ml and $FIO_2$ of 0.4 in Figure 2, and two extrapolations for values of $PaO_2$ of 90 and 65 mm Hg are also drawn.

**Discussion**

A few examples will illustrate the usefulness of the nomogram in estimating the degree of pulmonary dysfunction and in using various therapeutic methods to increase $PaO_2$.

A patient has a measured hemoglobin level of 11 gm/100 ml, a $PaO_2$ of 65 mm Hg, and an $FIO_2$ of 40 percent, as shown by horizontal line 1 in Figure 2 (point A). If the assumption is made that the $C(a-v)O_2$ is 5 (point B), the $Q_s/Q_T$ would be determined as 0.24; however, many postinjury or septic patients have high cardiac outputs with low $C(a-v)O_2$. If the $C(a-v)O_2$ were only 3 (point C), the $Q_s/Q_T$ actually would have been 0.34. Conversely, if the cardiac output were low with large $C(a-v)O_2$ of 8 (point D), the $Q_s/Q_T$ would be only 0.16. If the cardiac output rose, lowering the $C(a-v)O_2$, while the $Q_s/Q_T$ remained the same, the $PaO_2$ would markedly increase to 73 mm Hg, with a $C(a-v)O_2$ of 4, or to 88 mm Hg with a $C(a-v)O_2$ of 3.

If we consider a sicker patient with a $PaO_2$ of 50 mm Hg on $FIO_2$ of 40 percent and hemoglobin level of 11 gm/100 ml (Fig 2, point E, line 2) with $C(a-v)O_2$ of 5, the $Q_s/Q_T$ is 0.35 but rises to 0.48 with a $C(a-v)O_2$ of 3. More striking is the finding that changing $FIO_2$ to 100 percent only raises $PaO_2$ to 88 mm Hg, clearly showing the futility of this method of treatment. If the $C(a-v)O_2$ increases from 5 to 8 due to depression of cardiac output while $Q_s/Q_T$ remains unchanged (point F, line 3), then with an $FIO_2$ of 0.4, the $PaO_2$ will be 44 mm Hg, and with an $FIO_2$ of 1.0, the $PaO_2$ will be only 50 mm Hg. On the other hand, if something such as the use of positive end-expiratory pressure decreases the shunt to 0.2 without depression of cardiac output or change in $C(a-v)O_2$, the $PaO_2$ (point G, line 4) will rise to 73 on an $FIO_2$ of 0.4.

Another use of the nomogram is to give some estimate of the amount of depression in the $PaO_2$ that results from true shunt vs the amount of depression in the $PaO_2$ that is due to hypventilation, rather than nonventilation of alveoli. If a horizontal line is drawn through any point for $PaO_2$, then one can read the predicted $PaO_2$ that might be expected from a given change in $FIO_2$. If only the $FIO_2$ administered to the patient is changed and a second analysis of blood gas levels deviates from the prediction, the discrepancy is most likely due to reduction of apparent shunt by providing adequate oxygen to saturate blood draining the alveoli with low $V_{A/O}$. Thus, by induction, one can have some idea of the nature of the ventilation-perfusion abnormality that is causing the fall in $PaO_2$. If a change in $FIO_2$ results in predicted changes in $PaO_2$, then the patient’s pulmonary dysfunction is primarily resulting from perfusion of unventilated alveoli. If the change is greater than predicted, then the depression of $PaO_2$ is, to a significant degree, due to hypventilation of some alveoli.

The nomogram can also be helpful in predicting when the assumption that $C(a-v)O_2$ is 5 will give a reasonable estimate of shunt. In a study of a large number of patients observed after trauma, Shapiro and associates showed that at a shunt of less than 0.22, the error in assuming that $C(a-v)O_2$ was 5 was less than 0.05. The error of assuming that $C(a-v)O_2$ is 5 rapidly increases as the $PaO_2$ falls. If the $PaO_2$ with the subject breathing room air is less than 60 mm Hg and on an $FIO_2$ of 1.0 is less than 235 mm Hg, it is essential to have a sample of mixed venous blood to predict accurately the $Q_s/Q_T$.

In addition, it must be emphasized that at very low values for $PaO_2$, any estimation of shunt or $C(a-v)O_2$ using measurements of blood oxygen pressure, rather than oxygen content is inaccurate. Unfortunately, most clinical laboratories do not measure oxygen content, and so the clinician is dependent on these less accurate values. Hopefully, familiarity with the nomogram and the crowding of numbers at the lower levels of $PaO_2$ will make the limitations in accuracy of blood oxygen pressure readily apparent.

**References**