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THE OXYGEN-CARBON DIOXIDE DIAGRAM

Hermann Rahn
Wallace O. Fenn

The University of Rochester

Statement A
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August 1953

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THE OXYGEN-CARBON DIOXIDE DIAGRAM

Hermann Rahn
Wallace O. Fenn
University of Rochester

August 1953

Aero Medical Laboratory
Contract No. AF 18(600)-17
RDO No. 696-61

Wright Air Development Center
Air Research and Development Command
United States Air Force
Wright-Patterson Air Force Base, Ohio
FOREWORD

This summary of experimental investigations and theoretical development in respiratory physiology presented in this report were carried out over a period of many years in the Department of Physiology, School of Medicine and Dentistry, University of Rochester, New York, and were conducted under the provision of various Air Force contracts. This present summary was written under the provision of Contract AF 18 (600)-17 with Dr. J.W.Wilson and Dr. W.Hull of the Physiology Branch, Aero Medical Laboratory acting as project monitors. Work was performed under RDO No. 696-61, "High Altitude Physiology."
ABSTRACT

This report presents the $O_2$-$CO_2$ diagram which is useful in charting and predicting $O_2$ and $CO_2$ tension, blood gas content, blood pH, respiratory exchange ratios and alveolar ventilation as a function of changes of one or more parameters which may be encountered under various physiological stresses such as high altitude. The applications of this diagram are illustrated.

PUBLICATION REVIEW

This report has been reviewed and is approved.

FOR THE COMMANDER:

ROBERT H. BLOUNT
Colonel, USAF (MC)
Chief, Aero Medical Laboratory
Directorate of Research
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PREFACE

It is our purpose to present the oxygen-carbon dioxide diagram and some of its many applications in convenient form for study and consultation. We have found this diagram so helpful in the solution of our own daily problems that we believe it can be made equally useful to others. With oxygen plotted as abscissae and carbon dioxide as ordinates, it can be used to represent the simultaneous quantities of these two gases found in tissues, blood or lungs in any respiratory maneuver. In L. H. Henderson's classical monograph on Blood seven respiratory variables were considered and to represent the interrelations of these variables 105 different charts were presented as theoretically necessary for completeness. While all of these presented essentially the same basic facts, not all were equally useful. Similarly the O₂-CO₂ diagram is only one of many useful schemes for presenting the complicated processes involved in respiration. This particular diagram is unique, however, in its versatility and convenience and perhaps particularly its ready applicability to tissues, blood and pulmonary gases. The whole oxygen transport system can therefore be represented on a single diagram from the point of inspiration to the point of combustion in the tissues.

Like any other chart this one is highly forbidding at first acquaintance and even the most mathematically-minded physiologists are inclined to resent this particular type of symbolism as an unnecessary complication -- and they therefore put it aside in expectation of the day when the necessary facts can be stated in a few words with such simplicity that the atrocities of our diagrams can be neglected. This, unhappily, is a false hope for the only alternative is merely a rather complicated equation which to most people is still less meaningful. In fact, the two most awkward equations, those for alveolar air and alveolar ventilation, are fortunately plotted as families of straight lines on this diagram. When these lines are drawn as a transparent chart appropriately superimposed on the CO₂-O₂ diagram, the desired parameters can be read off graphically and therefore do not require special calculations. Indeed we have found increasing numbers of physiologists making consistent use of this device for straight thinking and some have copied our transparent charts to save the labor of lengthy calculations. We are encouraged to believe, therefore, that with the aids provided here anyone can master its various intricacies even though he is allergic to equations. Once mastered it provides a precise and comprehensive framework into which all the various pieces of the respiratory jig-saw puzzle can be fitted so that the interrelations stand out clearly.

In the Diagrams I - X we have built up the main outlines of the subject. With the legends it is hoped that they are self-explanatory. These diagrams include some familiar nomograms such as Diagrams VI and VII from Dill and his associates, which are so useful that they could not well be omitted from any set of working charts. These are printed large enough, however, so that they can really be used to obtain reliable figures for practical problems.
Unlike these Diagrams the various text figures included in the remainder of the book are intended chiefly to aid the reader in understanding some of the many possible applications of the $O_2-CO_2$ diagram. Our selection of these applications has been quite arbitrary and could be extended almost indefinitely to include such subjects as breath holding, drowning, explosive decompression, diving operations, and oxygen poisoning. Perhaps the most important omission, however, is the very fundamental problem of "distribution" and "diffusion" in the lung which has been so superbly presented by Riley and Cournand (14) using the $O_2-CO_2$ diagram. Our consideration will be given to the inclusion of these subjects if we ever have an opportunity in a second edition to correct the many errors (minor ones only, we hope) which must inevitably have escaped our notice in this volume. Meanwhile, we hope that our indulgent readers will be kind enough to call our attention to any such inaccuracies which may come to their attention.

Almost all the work represented herein has been generously supported by the Aero Medical Laboratory, Wright Air Development Center over the last ten years and we take pleasure at this time in expressing our gratitude and appreciation to the officers at that institution.

We are particularly indebted also to our many colleagues, past and present. Among the former, special mention should be made of Dr. Arthur B. Otis and, at an earlier date, Dr. Leigh E. Chadwick, both of whom made important contributions.

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August 21, 1953
Rochester, New York
INTRODUCTION TO DIAGRAMS

The ten diagrams which follow are designed to contain much of the data necessary for the solution of problems in respiratory gas exchange and gas transport. These diagrams themselves will suffice for many of the common problems which are frequently encountered, particularly those concerned with subjects breathing air or pure oxygen either at ground level or altitude. When other gas mixtures are involved, it will be necessary to replace Diagrams VIII and IX and to construct special diagrams. A work sheet is provided for that purpose. The methods of making these constructions are described in a special section at the end of the diagrams and the necessary equations are provided in the Equations section. Two of the diagrams are reproduced on transparent charts so that they can be superimposed on other diagrams at the proper position depending upon the value of the inspired oxygen pressure. For convenience we have provided a table of the barometric pressures at different altitudes (see special section) as well as the familiar nomograms of Dill and his associates. The latter are slightly simplified and are printed large enough for easy reading.

The physiological principles exemplified by these diagrams can easily be expressed in simple words but it is not possible in words to convey an accurate quantitative picture of the changes involved. It is in this way that the diagrams will be able to make their greatest contribution if one masters the habit of making frequent use of them. There is no respiratory maneuver involving changes in tensions of the respiratory gases in any part of the body which cannot be studied in terms of this diagram and the attempt to do so can hardly fail to prove profitable.

Of particular significance for the usefulness of these diagrams is the fact that within the limitations described they are applicable to any animal or person, the equations being independent of the size of the person, his metabolic rate, or the volume of his lungs.
Diagram I

Work Sheet. Abscissa, $pO_2$ - mm Hg. Ordinate, $PCO_2$ - mm Hg.
Diagram II

Combined O₂ and CO₂ dissociation curves of whole blood for man at sea level. Data obtained from Nomogram of Dill — see Diagram No. VI.

The oxyhemoglobin capacity is 20 vol. % (ml. STPD per 100 ml. of blood). The isopleths for various contents are indicated in red color. The slope of these isopleths represents the Bohr Effect.

The isopleths for CO₂ content represent the total CO₂ content of whole blood (ml. STPD per 100 ml. of blood) and are indicated in blue color. The slope of these isopleths represents the Haldane Effect.

The chart is useful in determining the CO₂ and O₂ contents of arterial blood if the CO₂ and O₂ tensions of the alveolar air are known, or vice versa if the alveolar-arterial O₂ gradient is disregarded or supplied.
Diagram III

Combined oxyhemoglobin saturation and plasma pH curves for man residing at sea level. Data obtained from Nomogram of Dill.

The isopleths of oxygen contents from the previous Diagram II are converted to per cent oxyhemoglobin saturation — \( \% \text{HbO}_2 \) and are indicated in red color.

The isopleths for serum pH are indicated in blue. These curves shift upwards at the left due to the Haldane Effect.
AT SEA LEVEL

%HbO₂

P₀₂

pH (s)

90

95

97.5

7.3

7.4

7.5

7.6

7.7
Diagram IV

Combined O₂ and CO₂ dissociation curves of whole blood attained by permanent residents living at 17,500 ft. in the Andes at barometric pressure of 401 mm Hg. These miners have an alveolar pCO₂ of 25.6 and a pO₂ of 42.3 mm Hg. Data obtained from the Nomogram of Dill — Diagram VII.
AT SEA LEVEL

\( P_{O_2} \) vs. \( %HbO_2 \) and pH

- \( P_{O_2} \) values range from 70 to 140
- \( %HbO_2 \) values range from 90 to 97.5
- pH values range from 7.3 to 7.7
Diagram V

Combined oxyhemoglobin saturation and plasma pH curves for permanent residents living at 17,500 ft, in the Andes. From Nomogram of Dill — Diagram VII.
MINERS RESIDING AT 17,500 FT. (5.3 km)
3.4 Km) AT A PRESSURE OF 401 mm Hg
Diagram VI

The blood Nomogram of Dill for man residing near sea level. The oxyhemoglobin values and CO$_2$ contents have been converted to vols. %. The R.Q. lines have been added.
MAN AT SEA LEVEL

Dill, Edwards, Consolazio
J.B.C. 118, 1937
Diagram VII

The blood Nomogram of Dill for permanent residing at 17,500 ft. altitude.
MINERS RESIDING AT 17,500 FT. (5.34 Km)

Dill, Talbott, Consolazio
J. Bio. Chem. 118, 1937
MINERS RESIDING AT 17,500 FT. (5.34 Km)

Dill, Talbott, Consolazio
J. Bio. Chem. 118, 1937
Diagram VIII

Isopleths of gas Exchange Ratio. A transparent copy of this diagram is supplied (mounted on the back cover), which is designed to be superimposed upon a CO₂-O₂ diagram like Diagram II, so that the base line of the transparency coincides with the base line or O₂ axis of the diagram and so that the point P₁₀₂ from which the R lines radiate is at the correct value for the inspired oxygen pressure. As gas exchange occurs in the alveoli the composition of the gas changes is indicated by these lines according to the exchange ratio, R. In a steady state, R = the R.Q. or respiratory quotient. Due to the nitrogen in the lungs the (negative) slope of these lines is not exactly equal to $\Delta V_{CO₂}/\Delta V_{O₂} = R$ but is $R/ (1 - F_{ICO₂}^O₂ (1 - R))$ in accordance with the alveolar air equation. The chart is a graphic representation of the alveolar air equation (see Equation 1). This chart applies only when O₂ fraction in the inspired air ($F_{ICO₂}^O₂$) is .209, the $F_{ICO₂}^O₂ = 0$, and the scale of ordinates and abscissae are equal. $P₁₀₂$ varies with the barometric pressure and is equal to ($P₈₋₄₇$) .209. The less the $N₂$ in the inspired air, the less these lines radiate until at $F_{IN₂} = 0$ they all coincide at the $R = 1$ line (1).

The chart is useful (a) in determining the R.Q. when the composition of the inspired and the alveolar air or expired air is known, or (b) in determining the alveolar O₂ tension for a given R when the inspired air composition and the alveolar CO₂ tension are known. It can be applied to a subject breathing air at any altitude.
Isopleths of gas exchange ratio $R$

$\alpha = R$

$P_{O_2}$
Diagram IX

Isopleths of Alveolar Ventilation. A transparent copy of this diagram (mounted on the back cover) is supplied to be used much like that of Diagram VIII. When the base line is properly superimposed on the \( O_2 \) axis of a \( CO_2-O_2 \) diagram with the point \( P_{IO_2} \) at the proper value for the inspired oxygen tension, the family of straight lines indicates the values of the alveolar ventilation \( (V_A) \) which would occur for a rate of oxygen consumption \( (V_{O_2}) \) of 100 ml., STPD, per minute. Thus for \( V_{O_2} = 300 \) ml. per minute, all values must be multiplied by 3. This family of lines is a graphic representation of Equation 2. It is applicable to a subject breathing air at any altitude provided the scale of the chart is 10 mm per inch. As the nitrogen content of the inspired air diminishes the (negative) slopes of these lines increase until in pure \( O_2 \) they coincide with the \( R = 1 \) diagonal (1).

If this chart and diagram VIII are superimposed simultaneously on diagram II, for example, there is a representation of 6 parameters, \( R \), alveolar \( CO_2 \) and \( O_2 \) tensions, arterial \( CO_2 \) and \( O_2 \) contents, and the ventilation/oxygen consumption or \( \dot{V}_A/\dot{V}_{O_2} \) ratio. Determination of any two of these parameters will fix the alveolar point and permit the values of all the others to be read from the chart. It is assumed only that there is such an entity as alveolar air of uniform composition. The values are independent of the size of the subject or the dimensions of his lungs.
\[ \dot{V}_A = \frac{L/min \text{ (B.T.P.S.)}}{100 \text{ ml } O_2 \text{ uptake (S.T.P.D.)}} \]
Diagram X

Combined oxyhemoglobin saturation and total blood CO₂ dissociation curves for the dog. These data were obtained by equilibrating dog blood with various O₂ and CO₂ tensions and analyzing for their O₂ and CO₂ content by the Van Slyke method. By extrapolation and interpolation of these data, as well as data previously obtained by A. Krogh, this composite chart has been drawn. The oxygen capacity of our dogs averaged 20 vols. %. (H. Rahn and H. T. Bahnson).
ION CURVES OF THE DOG

% HbO₂

85  90  95  97  98

total CO₂ Vol. %
53
49
45
41
37
33
29

P₀₂

70  80  90  100  110  120  130  140
All equations apply to the gas pressures or volumes after the gases have been warmed and saturated with water vapor at 37° C. Therefore the total pressures of the dry gases in the lung are equal to the barometric pressure, $P_B = 47$ mm Hg H$_2$O. The fraction of O$_2$, $F_{I02}$, is 0.209 in room air. Thus the inspired O$_2$ tension after it has been warmed and saturated is 0.209 ($P_B = 47$) when room air is breathed, and ($P_B = 47$) when pure O$_2$ is breathed. The fraction of CO$_2$, $F_{I02}$, in room air is considered to be 0.

In case of the alveolar ventilation equations the alveolar ventilation is expressed in l/min at B.T.P.S. (Body temperature, pressure, saturated), but the oxygen uptake is expressed in ml/min at S.T.P.D. (Standard temperature, pressure, dry). The difference in volume unit as well as changing fraction of gases to its partial pressure (in the derivation of the equation) introduces a constant .864.

**R Lines**

(a) When the inspired CO$_2$ tension is 0. The barometric pressure and the inspired O$_2$ fraction must be known. Assign any value to R and calculate the $P_{Ao2}$ (from Equation 1) for any arbitrary $P_{Aco2}$ value. The intersection of the O$_2$ and CO$_2$ tension on the diagram connected to the inspired O$_2$ tension, $P_{I02}$, yields the desired R line. Or in other words all points on this line can only be the result of this particular exchange ratio, $R$. This equation applies to all O$_2$-N$_2$ mixtures, however the slope of any particular R line (except $R = 1$) varies with the O$_2$-N$_2$ ratio.

(b) When CO$_2$ is present in the inspired gas. In this case the procedure is similar but Equation 6 must be used and the CO$_2$ concentration of the inspired air must be known.

There is one exception where the slopes of the R lines breathing air can be used when breathing CO$_2$. This is the case where the CO$_2$ has been added to air. For discussion of this case see Applications of O$_2$-CO$_2$ Diagram, section (q).

**V$_A$ Lines**

(a) When the inspired CO$_2$ tension is 0. The procedure is similar. The barometric pressure, the inspired O$_2$ fraction and the oxygen uptake, $V_{O2}$, must be known. Assign any value to R and calculate $P_{Aco2}$ (from Equation 2) for any arbitrary $P_{Aco2}$ value. Repeat this performance for a different $P_{Aco2}$ value. Connect the 2 intersections on the O$_2$-CO$_2$ diagram which yields the desired V$_A$ line. All V$_A$ lines are parallel to each other. When V$_A$ is infinity, it intersects the abscissae at the $P_{I02}$ point. This line is the same as that for infinite R. This equation applies to all O$_2$-N$_2$ mixtures, however the slope of the V$_A$ lines varies...
with the $O_2-N_2$ ratio. For the total ventilation, $V_E$, the product of (dead space x frequency) must be added.

Equation 3 is a very useful and much simpler equation. It expresses $V_A$ in terms of $R$ and $P_{Aco2}$, where $\dot{V}_{o2} \times R = \dot{V}_{co2}$. As long as $V_{co2}$ is constant the $V_A$ lines are merely horizontal lines across the $O_2-CO_2$ diagram and they do not require any $O_2$ values. They are therefore also independent of the inspired $O_2$ value.

(b) When $CO_2$ is present in the inspired gas. In this case the procedure is similar to the above procedure except that equation 7 or its alternate solution (in terms of $O_2$ and $CO_2$ only) must be used.

There is one very useful exception where the $V_A$ lines breathing room air are the same as when $CO_2$ is breathed. This is the case when $CO_2$ has been added to the room air. For discussion of this case see Application of $O_2-CO_2$ Diagram, section (q).
1. **Alveolar air equation (1).** Values of the alveolar O₂ tension, \( P_{A_O_2} \), are expressed in terms of the exchange ratio, \( R \), the inspired O₂ tension, \( P_{I_O_2} \), and the alveolar CO₂ tension \( P_{A_CO_2} \). The inspired fraction of O₂ is \( F_{I_O_2} \) and the inspired CO₂ tension is considered to be zero.

\[
P_{A_O_2} = P_{I_O_2} + \left( \frac{P_{A_CO_2} \cdot F_{I_O_2} \cdot (1 - R)}{R} \right) - \frac{P_{A_CO_2}}{R}
\]

or it may be rearranged to solve for \( R \):

\[
R = \frac{P_{A_CO_2} \cdot (1 - F_{I_O_2})}{P_{I_O_2} - P_{A_O_2} - F_{I_O_2} \cdot P_{A_CO_2}}
\]

2. **Alveolar ventilation equation (1).**

\[
P_{A_O_2} = P_{I_O_2} - \frac{0.864 \cdot V_{O_2} \cdot (1 - F_{I_O_2}) \cdot F_{I_O_2} \cdot P_{A_CO_2}}{V_{A}}
\]

where \( V_{O_2} = \text{ccO}_2 \text{ per min. STPD} \)

\( V_{A} = \text{lit/min BTPS alveolar ventilation (i.e., total ventilation less dead space ventilation).} \)

3. Alveolar ventilation may also be expressed in terms of alveolar pCO₂ and the exchange ratio, \( R \).

\[
V_{A} = \frac{0.864 \cdot V_{O_2} \cdot R}{P_{A_CO_2}}
\]

4. **Ventilation - perfusion equation (2).** By combining the Fick equation for blood flow with the alveolar ventilation equation the alveolar gas concentration may be expressed in terms of blood flow through the lung in lit/min, \( Q \) as well as \( V_{A} \).

Thus

\[
P_{A_CO_2} = \frac{Q}{V_{A}} \quad 0.864 \cdot R \cdot (C_{a_o_2} - C_{v_o_2})
\]

where \( C_{a_o_2} - C_{v_o_2} \) is the arterial-venous O₂ difference in ml/lit.

5. **Alveolar air and alveolar ventilation equations in terms of N₂.** These are similar to equations 1 and 2 expressed in terms of alveolar \( N_2, P_{A_N_2} \), when air is breathed.
5. (Continued)

\[ P_{An_2} = 0.791 \frac{P_{AcO_2}}{R} (1-R) + P_{In_2} \]

\[ V_A = 0.683 \frac{V_{O_2}}{(1-R)} \frac{P_{An_2} - P_{In_2}}{F_{Ico_2} (1-R) + R} \]

6. **Alveolar air equation when CO\textsubscript{2} is in the inspired gas mixture (1).**

\[ P_{AO_2} = P_{IO_2} \cdot R + P_{ACO_2} \cdot F_{IO_2} \cdot (1-R) + P_{Ico_2} - P_{ACO_2} \]

\[ F_{Ico_2} \cdot (1-R) + R \]

where \( F_{Ico_2} \) is the fraction of the inspired CO\textsubscript{2}

and \( P_{Ico_2} \) is the CO\textsubscript{2} tension of the inspired gas.

7. **Alveolar ventilation equation when CO\textsubscript{2} is in the inspired gas mixture (1).**

When the ventilation is expressed in terms of R and the alveolar and inspired CO\textsubscript{2} for a given O\textsubscript{2} uptake we have

\[ V_A = 0.864 \frac{V_{O_2}}{(R + F_{Ico_2} (1-R))} \frac{P_{ACO_2} - P_{Ico_2}}{P_{ACO_2} - P_{Ico_2}} \]

\( V_A \) expressed in terms of O\textsubscript{2} and CO\textsubscript{2} only may be found elsewhere (1).
### ALTITUDE-PRESSURE TABLE

<table>
<thead>
<tr>
<th>Altitude Ft.</th>
<th>PB mm Hg</th>
<th>(PB-47) mm Hg</th>
<th>(PB-47) mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>760</td>
<td>713</td>
<td>149</td>
</tr>
<tr>
<td>2000</td>
<td>707</td>
<td>660</td>
<td>138</td>
</tr>
<tr>
<td>4000</td>
<td>656</td>
<td>609</td>
<td>127</td>
</tr>
<tr>
<td>6000</td>
<td>609</td>
<td>562</td>
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<tr>
<td>8000</td>
<td>564</td>
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<td>10000</td>
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<td>100</td>
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<td>12000</td>
<td>483</td>
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<td>91</td>
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<tr>
<td>14000</td>
<td>446</td>
<td>399</td>
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<td>365</td>
<td>76</td>
</tr>
<tr>
<td>18000</td>
<td>379</td>
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<td>69</td>
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<td>20000</td>
<td>349</td>
<td>302</td>
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<td>22000</td>
<td>321</td>
<td>274</td>
<td>57</td>
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<td>24000</td>
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<td>247</td>
<td>52</td>
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<td>26000</td>
<td>270</td>
<td>223</td>
<td>47</td>
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<td>28000</td>
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<td>200</td>
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<td>30000</td>
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<td>159</td>
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<tr>
<td>34000</td>
<td>187</td>
<td>140</td>
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<td>36000</td>
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<td>38000</td>
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<td>40000</td>
<td>141</td>
<td>94</td>
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<td>42000</td>
<td>128</td>
<td>81</td>
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<td>44000</td>
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<td>106</td>
<td>59</td>
<td>12</td>
</tr>
<tr>
<td>48000</td>
<td>96</td>
<td>49</td>
<td>10</td>
</tr>
<tr>
<td>50000</td>
<td>87</td>
<td>40</td>
<td>8</td>
</tr>
<tr>
<td>63000</td>
<td>47</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

(All pressure figures rounded off to the nearest mm).

PB = Barometric Pressure - U.S. Standard Atmosphere

47 = $P_{H_2O}$ vapor pressure, at 37° C.

(PB-47) = Total pressure of the dry gases after the inspired gas has been saturated with water vapor. It is also equal to the inspired $O_2$ tension, $P_{O_2}$, when pure oxygen is inspired.

$.209$ = Fraction of $O_2$ in dry air at any pressure

$.209$ (PB-47) = Inspired $O_2$ tension, $P_{O_2}$, when air is breathed.

CONVERSION FACTORS:

1 Atmosphere = 14.7 p.s.i. = 760 mm Hg = 29.9 inches Hg
1 Foot = 0.305 meter

WADC TR 53-255 16
APPLICATIONS OF THE O₂-CO₂ DIAGRAM

The O₂-CO₂ diagram (1) allows the simultaneous visualization of the various parameters which determine the gas tensions in the blood and the lung. The whole O₂ and CO₂ transport between the inspired gas tensions and the venous blood gas tension can be described and the changes which must occur whenever one or more parameters are altered can be quantitatively predicted. The observed gas tensions in the lung and blood are primarily a function of: 1. the inspired O₂ tension, 2. the rate at which the air pump works, the alveolar ventilation, and 3. the rate of the heart pump or cardiac output.

The following applications are designed to illustrate the practical operations which are required in using the preceding diagrams to represent these problems of gas transport on the O₂-CO₂ diagram.

a.) Alveolar Air - Determination of ventilation and R from the composition of the alveolar air.

For general orientation we may take a subject with an alveolar O₂ and CO₂ tension of 100 and 40 mm Hg, respectively, when the barometric pressure is 747 mm Hg (see Figure 1). The moist, inspired O₂ tension (Pio₂) will be equal to (747-47) × 209 or 146 mm Hg. The transparent chart (No. VIII) for R, the gas exchange ratio, and for Vₐ, the alveolar ventilation (No. IX) placed at this value indicated that this alveolar gas tension is the result of an exchange ratio (R.Q.) of .85 and an alveolar ventilation of 1.83 L/min for an O₂ consumption of 100 cc/min (S.T.P.D.). If we assume an oxygen uptake of 300 cc, then the alveolar ventilation would be 1.83 × 3 or 5.5 L/min (B.T.P.S.). Vₐ chart cannot be used on Figure 1 where scale is reduced. Replot on Work Diagram I instead.

b.) Expired Air

Expired air may be regarded as a mixture of inspired air and alveolar air and consequently, on the CO₂-O₂ diagram, the composition of expired air must lie somewhere on the straight line which connects the inspired point and the alveolar point, the exact position depending on the relative amounts of dead space air and alveolar air. If E represents the expired air in Figure 1, then in accordance with the Bohr equation the linear distances AE/Al = dead space / tidal volume, and the distance EI represents alveolar volume. The greater the dead space the greater the distance between A and E. The chart demonstrates that if the composition of the expired and inspired air is known, the R diagonal can be drawn. The point representing the alveolar air on this line can be determined if the dead space and the tidal volume are known. In this indirect way the composition of alveolar air can be determined without the necessity of obtaining a sample for analysis.

c.) Arterial and Venous Blood Gas Tensions

For alveolar CO₂ and O₂ tensions of 40 and 100 mm, respectively, the
arterial tensions may be estimated as 40 mm and 95 mm, respectively. This assumes an average alveolar-arterial gradient \( (P_{A02} - P_{A02}) \) of 5 mm and no difference between the alveolar and arterial \( CO_2 \). Using these values the arterial point is represented in Fig. 1 as point a. The combined \( O_2 \) and \( CO_2 \) dissociation curves of the blood (Diagram II) indicate that at this arterial tension for \( O_2 \) and \( CO_2 \) we have an \( O_2 \) and \( CO_2 \) content of 19.4 and 48.0 vols. %, respectively; or if we glance at the blood pH and oxygen saturation chart (Diagram III), we find an \( O_2 \) saturation of 97% and a pH of slightly above 7.4.

From this arterial point one may now determine the mixed venous blood (\( \bar{v} \)) tensions or contents. Since the exchange ratio, \( R_a \), in the lungs in our example is .85, \( R_b \) for the blood must also be .85. The dotted line between a and \( \bar{v} \) in Figure 1 is the blood R line of .85. The arterial-venous content difference for \( CO_2 \) and \( O_2 \) between any point on this line and the fixed arterial point has a ratio of .85. (This line can be obtained by placing a straight edge on the proper arterial \( O_2 \) and \( CO_2 \) tensions of the nomogram (Diagram VI). By pivoting the straight edge around the point where it intersects \( R = .85 \) pairs of simultaneous \( pO_2 \) and \( pCO_2 \) values for this particular blood R value can be read off and plotted).

The mixed venous blood must therefore be somewhere on this \( R_b \) line, the exact point being determined by the cardiac output. If the blood flow is 6 L/min. (the oxygen uptake being 300 cc), the A-V difference for \( O_2 \) is 5 vol. %. The venous point is therefore at the intersection of the blood R line and the \( O_2 \) content isopleth of 14.4 vols. % (19.4-5.0). The oxygen saturation and pH of the venous blood can be obtained from the other chart (Diagram III). The total tension available may be divided into fractions the most important being the inspired-alveolar fraction, the magnitude of which depends upon the pulmonary ventilation, and the arterial-venous fraction which depends upon the cardiac output. In the steady state both depend also upon the metabolic rate. In addition one might recognize a pulmonary diffusion fraction, if the arterial blood and alveolar air are not in complete equilibrium, and a tissue diffusion fraction. Each of these fractions can vary independently but in determining them one must start with the inspired air which is the fixed point in the system and work backwards.

Mixed venous blood as found in the pulmonary artery is a mixture of many components from various organs which differ widely in \( O_2 \) and \( CO_2 \) content. This is illustrated in Figure 2 for a man at rest. The arterial blood, represented by a solid dot is assumed to have a \( pO_2 \) of 100 mm, a \( pCO_2 \) of 40 mm and an \( O_2 \) content of 19.5 vols. %. From this point a family of blood R lines have been drawn (as described above) anticipating the various respiratory quotients to be encountered from 1.0 in the brain to nearly 0.7 in resting muscle. The venous blood of each organ must be represented by a point located somewhere on the blood R line appropriate to that organ. The distance of any point from the arterial point is determined by the ratio of its \( O_2 \) metabolic rate to its perfusion rate or the A-V oxygen content difference. The individual values for various organs have been
### FIG. 1

<table>
<thead>
<tr>
<th>Organ</th>
<th>Wt.-kg</th>
<th>( V_{O_2} )-ml</th>
<th>Q-1it</th>
<th>R.Q.</th>
<th>(A-V)(O_2)-vols%</th>
<th>Venous (O_2)-vols%</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Lung</td>
<td>1.20</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>(2) Kidney</td>
<td>.27</td>
<td>22</td>
<td>1.33</td>
<td>.66</td>
<td>1.65</td>
<td>17.85</td>
</tr>
<tr>
<td>(3) Brain</td>
<td>1.50</td>
<td>68</td>
<td>.84</td>
<td>1.00</td>
<td>8.10</td>
<td>11.40</td>
</tr>
<tr>
<td>(4) Heart</td>
<td>.33</td>
<td>28</td>
<td>.20</td>
<td>.97</td>
<td>13.00</td>
<td>6.50</td>
</tr>
<tr>
<td>(5) G.I. Tract, Liver, Spleen</td>
<td>3.20</td>
<td>54</td>
<td>1.50</td>
<td>.00</td>
<td>3.0</td>
<td>15.90</td>
</tr>
<tr>
<td>(6) Muscle</td>
<td>40.00</td>
<td>50</td>
<td>1.00</td>
<td>.70</td>
<td>5.0</td>
<td>14.50</td>
</tr>
<tr>
<td>(7) Con. Tissue, Skin, Fat</td>
<td>22.00</td>
<td>20</td>
<td>.63</td>
<td>.80</td>
<td>3.2</td>
<td>13.00</td>
</tr>
<tr>
<td>(8) End. Glands, Marrow</td>
<td>1.50</td>
<td>30</td>
<td>.50</td>
<td>.05</td>
<td>6.0</td>
<td>13.50</td>
</tr>
<tr>
<td>Total or Weighted Means</td>
<td>70.00</td>
<td>270</td>
<td>6.00</td>
<td>.05</td>
<td>4.5</td>
<td>15.00</td>
</tr>
</tbody>
</table>

### FIG. 2 VENOUS BLOOD GAS TENSIONS
derived from various sources in the literature as indicated below Figure 2. The resultant **mixed venous point**, indicated by the other solid dot, is the sum of the **total \( O_2 \)** consumed by each of the organs (i.e., its A-V \( O_2 \) difference \( \times \) its blood flow) divided by the total flow or cardiac output.

d.) **Ventilation - Perfusion Ratio**

For a given composition of the mixed venous blood and inspired air the alveolar or arterial gas tensions are determined by the ratio of alveolar ventilation/ perfusion or the \( V_A/Q \) ratio, (See Equation 4), and are independent of the metabolic rate. This concept can be applied to the whole lung or to an individual alveolus. In the former case \( Q \), the perfusion rate is equal to the cardiac output and \( V_A \) to the total alveolar ventilation.

Equation 4 can be solved graphically (2) and is illustrated in Figure 3. It is necessary to fix the two points which represent the inspired gas tensions and the mixed venous gas tensions (See solid dots). Radiating from each of these points is a family of \( R \) lines, the pulmonary \( R \) lines from the inspired air point and the blood \( R \) lines from the mixed venous point. The blood \( R \) lines are determined as described above except that one starts with the straight edge at the p\( CO_2 \) and p\( O_2 \) of the mixed venous point on the nomograms. The \( R \) lines for \( .6, 1.0, 1.5 \) and \( 3.0 \) are indicated in Figure 3. Clearly there is only one point where a given blood \( R \) line intersects the alveolar \( R \) line of the same value. The tensions of \( O_2 \) and \( CO_2 \) indicated by this point represent therefore the only values at which blood and alveolar air can exchange at this particular exchange ratio. For example, gas and blood can only exchange at an \( R = 1.0 \) when the alveolar composition has an \( O_2 \) and \( CO_2 \) tension of 112 and 37 mm, respectively. (In such an ideal case it is assumed that no arterial-alveolar gradients exist.) Equation 4 allows one to calculate the \( V_A/Q \) ratio of 1.5 which would be necessary for this exchange.

When the intersections of the various possible blood and gas \( R \)'s are plotted, they can be connected to form the heavy solid line between the inspired and venous point, shown in Figure 3. This line represents all the possible combinations of \( O_2 \) and \( CO_2 \) tensions which could exist in any alveolus, each depending on a particular \( V_A/Q \) ratio. Some of these values have been indicated by the large numerals opposite various \( R \) line intersections. An alveolus with no perfusion or infinite ventilation is represented by the inspired air point and an alveolus with no ventilation or infinite perfusion rate is indicated by the venous point.

e.) **Alveolar-Arterial \( N_2 \)**

The \( O_2-CO_2 \) diagram has so far omitted any reference to the \( N_2 \) tensions in the alveolar air. Actually the changes which occur in alveolar or arterial \( N_2 \) are reflected in the \( O_2-CO_2 \) diagram. This fact can best be appreciated by making first an extension of this diagram to include the \( N_2 \) and water vapor as shown in Figure 4.
On the abscissa we have the inspired gas tensions, $P_{\text{O}_2} + P_{\text{In}_2} + P_{\text{H}_2\text{O}} = P_B$, the barometric pressure (760 mm Hg). If now the metabolic $\text{CO}_2$ is added and $\text{O}_2$ is taken up in the expired or alveolar air, the $P_{\text{n}_2}$ in the expired or alveolar air no longer remains equal to the inspired nitrogen, $P_{\text{In}_2}$, except when $R = 1.0$. In the latter case the $\text{CO}_2$ added is equal to the $\text{O}_2$ uptake and the net volume of the gas remains the same. However, when $R$ is less than 1.0, that is when the $\text{O}_2$ uptake exceeds the $\text{CO}_2$ output, the inspired $\text{N}_2$ becomes concentrated by the volume shrinkage. On the other hand, with an $R$ greater than 1.0, the $\text{CO}_2$ output exceeds the $\text{O}_2$ uptake and thereby dilutes the inspired $\text{N}_2$ and increases the inspired gas volume.

Figure 4 illustrates 3 different alveolar gas concentrations representing $R$ values of .5, 1.0 and 2.0. (These large $R$ differences were chosen simply to magnify the $\text{N}_2$ changes to be represented on the very small scale of the abscissae). One will note that the horizontal distance (from 0 to the $P_B$ line) through each alveolar point + the vertical distance must always be equal to the total barometric pressure. The absolute tensions of the lower, middle, and upper alveolar points are indicated in the table below including the changes which occur between the inspired and alveolar $\text{N}_2$ as well as the factor by which the volume of the inspired air changes. This factor is equal to $(P_{\text{In}_2}/P_{\text{An}_2})$. Thus, for example, the inspired volume of 1000 ml is reduced to 935 ml in the lung under the conditions of the upper alveolar air point. At altitude this factor increases inversely with the total pressure since $P_{\text{In}_2}$ becomes reduced but the difference between $P_{\text{In}_2}$ and $P_{\text{An}_2}$ remains the same for comparable $R$ and $\text{CO}_2$ values.

<table>
<thead>
<tr>
<th>$P_{\text{CO}_2}$</th>
<th>$P_{\text{O}_2}$</th>
<th>$P_{\text{N}_2}$</th>
<th>$P_{\text{H}_2\text{O}}$</th>
<th>$P_B$</th>
<th>$\Delta P_{\text{N}_2}$</th>
<th>Vol. cor. Factor $(P_{\text{In}<em>2}/P</em>{\text{An}_2})$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insipired gas</td>
<td>0</td>
<td>149</td>
<td>564</td>
<td>47</td>
<td>760</td>
<td>1.00</td>
</tr>
<tr>
<td>Lower Pt. $R = 1.0$</td>
<td>30</td>
<td>119</td>
<td>564</td>
<td>47</td>
<td>760</td>
<td>0</td>
</tr>
<tr>
<td>Middle Pt. $R = 2.0$</td>
<td>40</td>
<td>125</td>
<td>548</td>
<td>47</td>
<td>760</td>
<td>-16</td>
</tr>
<tr>
<td>Upper Pt. $R = 0.5$</td>
<td>50</td>
<td>60</td>
<td>603</td>
<td>47</td>
<td>760</td>
<td>+39</td>
</tr>
</tbody>
</table>

It is now important to point out that the changes of the $P_{\text{In}_2}$ can be directly obtained for any alveolar point by simply measuring the horizontal distance between this point and the $R = 1.0$ line. This interval represents the partial pressure of $\text{N}_2$ gained or lost from the inspired $\text{N}_2$. For practical purposes, therefore, it is not necessary to extend the $\text{O}_2-\text{CO}_2$ diagram in order to evaluate the changes in $\text{N}_2$. This can be further verified by placing the $R$ chart on Figure 1. One will note that the horizontal distance between the alveolar point A and the $R = 1.0$ line is 8 mm. Therefore, the alveolar $P_{\text{N}_2}$ is equal to $P_{\text{In}_2} + 8$. It will also be noted that a similar procedure for the expired air composition (point E - Figure 1) will yield
FIG. 3 VENTILATION-PERFUSION RATIOS

FIG. 4 THE $O_2-CO_2-N_2$ DIAGRAM
a smaller $N_2$ tension than found in the alveolar air. Thus the $P_{An2}$ is not only a function of $R$ but of $P_{CO_2}$ as well (see Equation 5). During the unsteady state large fluctuations in alveolar $N_2$ may be encountered. Some of these are indicated under the section (p), entitled the $N_2$--$CO_2$ Diagram.

f.) Breathing of Pure $O_2$ at Altitude

When the inspired gas is pure $O_2$, the ventilation and R.Q. isopleths of diagrams VIII and IX are all coincidental with the $R = 1$ diagonal having a slope of -1. The alveolar point must lie on this diagonal regardless of the actual exchange ratio. This is necessary because with only two gases present, in addition to water vapor, the sum of their tensions must always be equal to $P_B$. For every altitude or every value of $P_B$, therefore, there must be a single "altitude diagonal" starting from the inspired $P_{O_2}$ tension and having a slope of -1 (1). Several such altitude diagonals are shown in Figure 5. The curve connects the alveolar points which have been observed as average values on each of these diagonals or at each of these altitudes. For any altitude the intersection of the appropriate altitude diagonal with this curve will identify the $CO_2$ and $O_2$ tensions of the average alveolar point. With the aid of diagrams II and III the corresponding values of $HbO_2$ $\%$, $CO_2$ and $O_2$ contents and pH can be read off. Diagrams VIII and IX are not applicable when pure $O_2$ is used as the inspired gas.

When breathing pure $O_2$ at barometric pressures less than 196 mm (33,000 ft), the inspired $O_2$ tension is less than at sea level breathing air. At altitudes below this the use of $O_2$ in partial or complete substitution for air will restore the alveolar $p_{O_2}$ to normal values. At altitudes higher than this the alveolar $p_{O_2}$ will be below normal in spite of the use of pure $O_2$ unless pressure breathing is used.

g.) Pressure Breathing

When pressure breathing is instituted, the pressure is theoretically applied to both the $CO_2$ and $O_2$ of the lung. But the respiratory center does not tolerate a higher $CO_2$ tension. Thus for practical purposes all the pressure is applied to the $O_2$ tension and the alveolar point moves to another altitude diagonal (3,4) without change in $p_{CO_2}$. In Figure 5 the subject has an alveolar air at point A at 46,000 ft. With 22 mm Hg pressure breathing his alveolar point moves over 22 mm Hg to the 42,000 ft. diagonal to point B. Since pressure breathing is usually accompanied by an additional hyperventilation, he moves down the altitude diagonal to point C. The corresponding changes in the arterial blood gas content resulting from: 1. positive pressure alone, and 2. from the additional hyperventilation can be readily seen on diagrams II and III.

h.) Hypoxia at Air Breathing Altitude

At an air breathing altitude (Figure 6) which has the same inspired $O_2$ tension as an oxygen breathing altitude (for example, 46,000 ft. on $O_2$ and 22,000 ft. on $CO_2$, the $P_{An2}$ is 70 mm Hg while the alveolar tension is 20 mm Hg.)
FIG. 5 ALTITUDE DIAGONALS – PRESSURE BREATHING

FIG. 6 ACUTE EXPOSURE TO 22000'
on air, PB = 105 and 321, respectively), the alveolar point is no longer bound to the R = 1.0 line because of the presence of N₂. Instead we have a family of altitude diagonals, each one depending upon the R and the alveolar ventilation. Only when the R breathing air is equal to 1, all other things being equal, will the alveolar pO₂ be the same at these two altitudes. At any other R on air the alveolar pO₂ will be lower or higher, depending upon an R lower or higher than 1, respectively. Upon acute exposure to low O₂ the ensuing hyperventilation blows off CO₂ stores of the blood and tissues and thus raises temporarily the R. The steady state at 22,000 ft. is not reached in less than 30 minutes. Figure 6 describes the changes observed in man upon exposure to 22,000 ft. altitude (5). Point A would represent the alveolar concentration man would have if he maintained his normal alveolar sea level ventilation of 6 1/min and R. = .8. The open circles represent at five-minute intervals the observed concentrations which are produced by the anoxic hyperventilation. At the end of 5 minutes (first point) the alveolar ventilation has more than doubled with a reduction in pCO₂ and an R value of 1.2. If this new ventilation were maintained the alveolar point would slowly move down on the particular ventilation isopleth with the exhaustion of the CO₂ stores until the R line of 0.8 was reached at a pCO₂ of 16 mm with the establishment of a new steady state. Actually the initial acapnia counteracts the hypoxic drive and the alveolar ventilation is slowly reduced to about 10 1/min after 30 minutes of exposure with an R value of slightly over 0.9 at point B. The predicted HbO₂ saturation at point A would have been 30% but the observed alveolar values predict an HbO₂ of 70%. (Actually the ear oximeter indicated a value of 65%. This would indicate an A-a gradient of about 3 mm O₂).

After 30 minutes’ exposure (point B) the subjects were quickly recompressed to ground level and the recovery pathway is indicated from point B to A. The reduced ventilatory drive to less than 5 1/min allows for the gradual recovery of the previously lost CO₂ stores with R values below 0.6.

i.) Acclimatization to Altitude

When after an acute exposure to anoxia the steady state has been reached (1/2 - 1 hour), then the alveolar air values must come to rest somewhere along the R = .85 diagonal of that particular altitude (if the true metabolic R Q is 0.85). In Figure 7 the upper curve represents the alveolar values found at various altitudes after the steady state is attained (6).

If man now remains longer at these altitudes, as has been verified by residence in the mountains, he very slowly increases his ventilation. Thus the CO₂ is lowered over several days, and the alveolar point progresses down along the R.Q. = .85 diagonal with proportional increase in alveolar pO₂ until the acclimatized curve of Figure 7 is reached. The degree by which his blood oxygen saturation increases during this acclimatization process can be predicted from the oxygen saturation curves since they are not appreciably altered by the slow increase in
hemoglobin. On the other hand the pH, O₂ and CO₂ content isopleths are no longer valid and require new nomograms for their construction. The only complete data available for permanent residents are those of Dill et al obtained on miners living in the Andes at an altitude of 17,500 ft. (P_B = 401). The diagrams, Nos. IV, V, and VII, include their nomogram as well as two O₂-CO₂ diagrams with the isopleths for O₂, CO₂ blood content and pH and HbO₂ saturation. The average alveolar pO₂ and pCO₂ tensions for these people are 42 and 26, respectively. The inspired O₂ tension at this altitude is .209 (401-47) or 74 mm Hg. The R chart (Diagram VIII gives these people at rest an R of .8. The V_A chart yields an alveolar ventilation of 2.7 L/min/100 ml of O₂ uptake. This is a 50% increase over what would be required if they maintained their pCO₂ at 40 mm Hg. Furthermore, it can be seen that their arterial O₂ content of 23.5 vols. % is actually above that for man at sea level in spite of the fact that their saturation is only 79%. The pH is just slightly above 7.4.

j.) Equivalent Altitudes

The problem of equivalent altitudes breathing air and oxygen can be best appreciated by placing, for example, the R chart at the inspired pO₂ of 94 in Figure 5 which is also equal to the inspired pO₂ of 40,000 ft. when O₂ is breathed. As has been pointed out above the alveolar gas is restricted to the slope of -1 as long as O₂ is breathed. Breathing air an inspired pO₂ of 94 will be found at 11,200 ft. where P_B = 497 and P₁O₂ = .209 (497-47). But in this case man is not restricted to the R = 1 line, but can theoretically be at any R depending upon his ventilation. In fact as pointed out in section (b) his R will vary considerably with acute exposure until he reaches his new steady state. For example, at this particular air altitude with a pCO₂ of 40 mm and an R of 0.7 the alveolar point will actually be on the 42,000 ft altitude diagonal for pure oxygen. With an R greater than 1 he will at an O₂ altitude less than 40,000 ft and only when his R = 1.0 may his equivalent altitude be regarded as the same as that predicted from the inspired oxygen tension.

The advantage of a high R breathing air compared to breathing O₂ at equivalent inspired O₂ tensions can actually be demonstrated in survival studies of animals acutely exposed to very low O₂ tensions (7).

k.) Psycho-motor Performance at Altitude

Man's performance at altitude is affected by the composition of the arterial or alveolar gases. At each altitude there exists an optimum alveolar gas composition for the best performance. This optimum may vary for different types of performance. The effects of anoxia and acapnia seem to be additive rather than antagonistic in their effects (8,9). Figure 8 indicates the approximate boundaries of normal and impaired performance as well as the normal alveolar gas composition encountered with changes in altitude. The arterial gas composition in any performance region may be approximately gaged by reference to Diagrams II
FIG. 7 ACCLIMATIZATION TO ALTITUDE

FIG. 8 PERFORMANCE AND ALTITUDE
and III. Two oxygen saturation isopleths and two altitude diagonals have been added for orientation.

1.) Oxygen Transport and Cardiac Output at Altitude

By means of the $O_2$-$CO_2$ diagram it can be shown that the hyperventilation and lowered alveolar $pCO_2$ which occurs at altitude is advantageous for the transport of oxygen because it permits the maintenance of the same mean venous oxygen tension with minimum increase in cardiac output.

This is demonstrated in Figure 9. The vertical line at a $pO_2$ of 30 mm represents the venous oxygen tension which is to be maintained. On this line 5 sample points are selected. The $CO_2$ and $O_2$ contents of each of these points can be determined from the background grid of more or less horizontal and vertical lines. The corresponding alveolar points are connected to the venous points by broken lines. Each alveolar point is 6 vols. % of $O_2$ higher and 5 vols. % of $CO_2$ lower than the venous points. This assumes, therefore, that the exchange ratio is 0.83 and the A-V oxygen difference is 6 vols. % with a cardiac output taken as 100%. The heavy curve to the right on which the alveolar points lie represents the alveolar requirement for a venous $pO_2$ of 30 mm.

The meaning of this curve is better demonstrated in Figure 10 where similar alveolar requirement curves are shown for different cardiac outputs between 100 and 300% of normal. The same venous $pO_2$ can be maintained with a lower alveolar $pO_2$ if the cardiac output is increased above the normal value. Suppose further that the alveolar point is at the upper intersection of the 120% cardiac output curve and the 42,000 foot altitude diagonal. The subject is breathing pure oxygen, the arterial saturation is 85% and the alveolar $pCO_2$ about 35 mm. If he now increases his ventilation, the alveolar point will travel down the 42,000 foot diagonal to the right. In so doing it will come closer to the 100% cardiac output curve and further from the 120% cardiac output curve. Thus the same venous $pO_2$ can now be maintained with a cardiac output which has decreased from 120 to about 110% of the normal. Indeed the 93% saturation line appears to pass through the minimum or bend of each of these alveolar requirement curves. Thus it can be seen that for the transport of oxygen (1) hyperventilation will minimize the cardiac output requirement so long as the percentage saturation is not higher than 93% and (2) the higher the altitude the lower the value of the alveolar $pCO_2$ which is minimum for cardiac output and therefore optimum for $O_2$ transport.

The same problem can also be approached from a consideration of the position of the venous point as illustrated in Figure 11. The alveolar point may be considered as located somewhere along the R.Q. diagonal of approximately 0.8 at an inspired $pO_2$ of 80 mm and an altitude, breathing air, of about 18,000 feet. Three such sample points have been selected from which blood $R = .8$ lines (dotted) have been drawn. These are so drawn that the $CO_2$ increases 0.8 vols. % for every $O_2$ decrease of 1 vol. %. Points on these dotted curves which are 5 vols. % $O_2$ lower than the alveolar points are arbitrarily connected by the 100% cardiac output curve.
**Fig. 9**

**O₂ TRANSPORT & CARDIAC OUTPUT**

![Graph showing the relationship between O₂ partial pressure (P₂O₂) and venous carbon dioxide pressure (PCO₂). The graph illustrates cardiac output at 1.0 when A-V diff. = 6 VOLS. % O₂ and 5 VOLS. % CO₂. Resulting arterial O₂ is also shown.]

**Fig. 10**

![Graph showing arterial O₂ saturation and pCO₂ in relation to arterial pO₂ and equivalent altitude. The graph displays cardiac output at 100% and 120%.]
Similarly, the 50% cardiac output curve lies at an A-V O$_2$ difference of 10 vols. %. This family of curves shows for this altitude, therefore, (1) how much the venous pO$_2$ can be increased by an increase in cardiac output without change in the ventilation, or (2) what change in the venous point can be expected if the ventilation is changed without change in the cardiac output, and (3) that increase in ventilation will increase the venous pO$_2$ until the alveolar pCO$_2$ falls to about 20 mm after which there is either no change or a slight fall (as indicated on the 50% cardiac output curve). (At ground level the venous pO$_2$ falls if hyperventilation causes any fall of alveolar pCO$_2$ below the normal value as shown below in Figure 14.)

Similar venous point curves can be drawn for different altitudes as illustrated in Figure 12. Six different altitude diagonals are illustrated each with a value of R = 0.8. The corresponding venous point curves are illustrated at the left, each labelled with the appropriate value of the inspired oxygen tension, varying from 50 to 149 mm. The points on each of the altitude diagonals where the arterial saturation falls below 100% is indicated by a circle. Dotted lines from these points to the 75% saturation line indicate the corresponding venous points where the A-V differences are all 5 vols. % and the A-V CO$_2$ differences are 4 vols. %. These are the points where the venous point curves coincide with the 75% saturation line. The solid circles on the venous point curves represent the points which correspond to the average alveolar points observed at each of the altitudes indicated — the corresponding alveolar points are not indicated in order to avoid making the diagram too complicated. From this chart it can be concluded (1) that for a given cardiac output and A-V oxygen difference the maximum venous pO$_2$ can be obtained at an alveolar pCO$_2$ which is lower as the altitude increases and (2) that the venous pO$_2$ increases with increasing ventilation to a maximum value and then decreases slightly and (3) at altitudes where the inspired pO$_2$ is 100 mm or less, the venous pO$_2$ can be increased by an increase of ventilation beyond that which normally occurs. (Note that the venous point curves move further to the right below the solid circles except at P$_{O2}>100$ mm.)

A slightly different representation of venous points and tissue points at a altitude is illustrated in Figure 13 where again six different 0.8 altitude diagonals have been drawn (for air breathing). Each of these terminates at the average observed alveolar point lying on the alveolar curve. From these alveolar points dotted lines are drawn to the venous points. Each of these dotted lines represents a blood R.Q. line with an A-V oxygen difference of 5 vols. % and an A-V CO$_2$ difference of 4 vols. %. These venous points correspond to the solid circles in Figure 9 and indicate the value of the venous gas tensions if there is no increase in the cardiac output above the normal ground level value. From the venous points so selected other dotted lines are drawn to the tissue curve which is supposed to be always 20 mm pO$_2$ lower than the venous value, this being the assumed minimum gradient which will suffice for adequate supply of oxygen to the tissues. The slope of the dotted lines representing this diffusion gradient is such that it would suffice for the diffusion of 5 vols. of oxygen to 4 vols. of CO$_2$ and it depends upon the molecular weight of the molecules and the solubilities of the gases in the tissues.
O₂ TRANSPORT & CARDIAC OUTPUT AT ALTITUDE

FIG. 11

FIG. 12

FIG. 13
On account of the high solubility of the CO$_2$ compared to that of oxygen the lines appear almost horizontal and all of the same slope. From these curves it can be seen that without increase in the cardiac output or excessive hyperventilation (i.e., greater than normal amount) the tissue pO$_2$ will fall to zero and symptoms of severe anoxia may be expected at altitudes higher than about 18,000 feet. If more than 20 mm oxygen gradient is required, anoxia would occur at still lower altitudes.

In conclusion these considerations show that so far as the transport of oxygen is concerned some additional hyperventilation at altitude is likely to be beneficial. It is not to be feared because of the fallacious argument that the decrease in pCO$_2$ prevents the unloading of oxygen in the tissues. This does not prove by itself that a further lowering of the alveolar pCO$_2$ will improve the performance or the maintenance of consciousness at altitude. One of the compensations for low O$_2$ is an increase in cardiac output and cerebral flow and a lowered pCO$_2$ antagonizes this compensation. It is difficult to predict, therefore, with certainty the net effect of further increase in ventilation. Performance tests indicate, however, that anoxia and acapnia appear to be additive in their effects and that the optimum pCO$_2$ is ordinarily lower than the value attained by the average person at altitude. If, therefore, in a given individual at altitude it is found that the performance seems to improve with an increase in alveolar pCO$_2$ and a corresponding decrease in pO$_2$, it probably means that (1) his ventilation was previously increased above the optimum, or (2) that the cardiac output was low and was markedly improved by the increased carbon dioxide.

m.) Gas Transport at Ground Level Breathing Air

It is furthermore of interest to focus upon the gas transport at ground level, particularly where the ventilation or the cardiac output may become insufficient. This situation is met in first aid resuscitation, prolonged resuscitation in respirator cases and during anesthesia in the operating room. Figure 14 illustrates the O$_2$ and CO$_2$ transport when air is breathed at a barometric pressure of 760 mm Hg. The inspired oxygen is 149 mm. It is assumed that (1) the metabolic rate remains constant, (2) the R.Q. is .8, and (3) there is no alveolar-arterial gas gradient. The alveolar gas tensions must lie somewhere on the R = .8 line. A CO$_2$ of 35 mm has been arbitrarily chosen as a normal value produced by a normal alveolar ventilation designated as 100%. The gas tensions produced by 1/2 and 2 times the normal ventilation (controlled by a respirator or manually by an anesthetist) are indicated by solid squares. Other values will fall on the R line according to the alveolar ventilation equation. The alveolar values are here assumed to equal the arterial values. One may now, as outlined previously, plot all the possible venous gas tensions corresponding to the various arterial tensions existing on the R = .8 line. If we choose an A-V O$_2$ difference of 5 vols. % as a normal cardiac output, the mixed venous gas tensions will have values indicated by the dotted line designated as 100% cardiac output. The values for 1/2 and 2 times the normal cardiac output are likewise indicated.
Consideration of Figure 14 indicates that for a given cardiac output the highest venous oxygen tensions are found when the ventilation and alveolar pCO₂ are normal. Even hyperventilation like hypoventilation causes then a decrease in the venous oxygen tension.

Further it is evident that at groundlevel the venous pO₂ is largely controlled by the cardiac output and is relatively independent of the ventilation while the venous CO₂ tensions and pH are largely uninfluenced by the cardiac output but vary widely with changes in ventilation. Thus, at normal cardiac output change in ventilation from 50 to 200% decreases the venous pCO₂ 56 mm but varies the venous pO₂ only ± 4 mm. Conversely, at normal ventilation rate a change in cardiac output from 50 to 200% increases the venous pO₂ 27 mm and decreases the venous pCO₂ only 7 mm. At high altitudes when the inspired oxygen tension becomes a limiting factor, the changes in venous pO₂ due to ventilation become relatively more important. It is, of course, largely the shape of the oxygen dissociation curve which maintains the venous pO₂ relatively constant in spite of wide variations of ventilation. Thus, at normal cardiac output change in ventilation from 50 to 200% may change the arterial pO₂ 60 mm, while the venous pO₂ changes only 6 mm. The CO₂ dissociation curve does not have this property.

These examples are obviously idealized conditions where the R.Q. remains the same. Actually with sudden depressions in ventilation rather marked changes occur which lower the R values and therefore shift the alveolar and arterial values to the left on this diagram. Such conditions are illustrated in the following chapter. Likewise, it might be pointed out that this analysis considers the whole body as a single capillary bed, whereas each organ has its own venous gas tensions and blood flows as illustrated in Figure 2, and the distribution of the blood between these different circulations may be altered so as to permit one organ, such as the brain, to prosper at the expense of other less essential tissues. All predictions based on this type of analysis are necessarily subject to modification by such special circumstance.

n.) Hypoventilation

Alveolar hypoventilation can be observed (1) by an increase in the external dead space, (2) by an increase in the resistance to breathing, and (3) by depression of the respiratory center (anesthesia). In either case hypoventilation involves a displacement of the alveolar or arterial point to a lower iso-ventilation line. Acute reduction in alveolar ventilation increases the pCO₂ and lowers the pO₂. Since the storage capacities in blood and tissues for the CO₂ are so much larger than that for O₂, the CO₂ tension will initially rise very much less than the O₂ will drop with a resultant large change in R until the steady state is reattained (10,11). Figure 15 illustrates the respiratory depressant action of a single standard dose, i.v., of sodium pentobarbital in a dog on the oxygen and CO₂ transport (12). The large circles indicate the alveolar, arterial and mixed venous blood gas tension before the anesthetic was given. The solid points indicate the changes during the following three hours at half hour intervals. Placing the R chart
FIG. 14 GAS TRANSPORT AT GROUND LEVEL

FIG. 15 GAS TRANSPORT DURING ANESTHEISA
on this diagram one will see the initial depression of R with anesthesia and finally a recovery with relatively high R values when the anesthetic wears off. The arterial tensions follow the alveolar values but with a large A-a O₂ gradient typical for dogs. The HbO₂ saturation is reduced to 85% 30 minutes after the induction. It will be noted that the mixed venous tensions do not change very much over the whole period because the cardiac output remains unaltered and the slope of the oxygen dissociation curve is relatively steep in this region. In anesthesia as in hyperventilation (Figure 6) the alveolar point moves in clockwise direction along an alveolar air loop on the pCO₂ pO₂ diagram. The two loops however are located in opposite directions from the normal point.

6) Breathing Air in Submarines or Other Closed Spaces

When a man is enclosed in an air-tight space, the oxygen percentage gradually diminishes as the carbon dioxide percentage rises until at a certain point death occurs. The changing composition of the air can be represented on a PCO₂-pO₂ diagram by a straight line comparable to an R.Q. diagonal. In this case when the space is strictly closed and the volume is large compared to the man the negative slope of the line is equal to the R.Q. of the man. If, on the other hand, the space is open to the outside through a pipe so that air is admitted or expelled to maintain constant pressure, then the equation must be modified slightly to cover this dilution. If further the volume becomes smaller and approaches the volume of the man, then the slope becomes less because of the CO₂ stored in the body.

Quantitative aspects of this situation are illustrated in Figure 16. When there are 10,000 or more liters of volume surrounding the man then the pO₂ will fall from the initial 149 mm to 100 mm in 30 or more hours and the pCO₂ at an R.Q. of 0.8 will rise to 39 mm = 0.8 (149-100). If, however, there are only 100 liters of gas surrounding the man, the pO₂ will fall as before to 100 mm in only 0.3 hours at the same basal rate of oxygen consumption while the pCO₂ may rise only to 19.5 mm or only half as much as before. This will be the case if half of the CO₂ produced is stored in the tissues. If the body can store as much CO₂ with a rise of pCO₂ of 1 mm as would be required to raise the pCO₂ of 100 liters 1 mm, then we say that the man's "equivalent volume for CO₂" is 100 liters. This seems to be a reasonable value to assume for short term experiments. For more prolonged experiments a considerably larger value could be assumed because of the more slowly reacting storage capacity of the bones (13).

In a submerged submarine the habitability of the closed space can be improved by constant removal of CO₂ by soda lime or other absorbant and the simultaneous addition of oxygen. The effect of the continuous absorption of CO₂ at a given rate without the addition of oxygen can be illustrated quantitatively by Figure 17. Here the rate of removal of CO₂ is expressed as a clearance or the number of liters of air cleared of CO₂ per minute per man. At a clearance of 12 liters per minute per man and a given rate of gas exchange as indicated on the chart the pCO₂ will rise along the lower curve but will never become much greater than 15 mm. At about this level the rate of production of CO₂ becomes just equal.
Composition of Submarine Air

Equivalent Volume of Man for CO₂ assumed = 100 L.

R = 0.8

O₂ per min = 300 cc.

FIG. 16

Air in Submarine at different rates of CO₂ clearance

\[ \frac{V}{V_{O₂}} (21 - \frac{P_{O₂}}{B}) = \log_e \frac{V_{CO₂}}{V_{CO₂}} - \frac{V}{B} \frac{P_{CO₂}}{B} \]

\[ V_{O₂} = 0.3 \text{ L/min} \]

\[ V_{CO₂} = 0.24 \text{ L/min} \]

FIG. 17
to the rate of production. The time required to reach this point depends upon the
volume of the submarine and the number of men. Time values are not indicated on
this chart and the equation of the curves given in Figure 11 does not contain time as
one of the parameters. The shape of the curves, in other words, is independent of
the time. It is only the rate of progress along the curve which depends upon time.
At lower rates of clearance the pCO₂ rises higher. When the clearance is zero, the
composition of the air changes according to the 0.8 R.Q. diagonal, as indicated
previously.

A general summary of possible changes in the composition of the air in a
submarine or other closed space is diagrammed in Figure 18. The 0.8 diagonal
indicates the changing composition of the air as a result of respiration by the
occupants of the ship. The arrow indicates the composition after 3.1 hours if there
are 1000 liters per man and each man consumes 0.3 liters of oxygen per minute
with an R.Q. of 0.8. There will then be 4% CO₂ in the air and the alveolar ventila-
tion will be twice normal (VR = 2). With this point for inspired air the simultaneous
composition of the alveolar air can also be indicated for this ventilation rate. It
will lie on the dotted line of slightly steeper slope. On this line the pO₂ for a given
pCO₂ is slightly greater because fresh air containing more oxygen is continuously
added in amounts equal to the difference between the volume of oxygen consumed
and the volume of CO₂ given off.

If now the submarine is continuously ventilated with fresh air at a rate of 6
liters per man per minute, then the composition of the air will be maintained
indefinitely at the 4% CO₂ point. If this ventilation is further increased, the point
representing the composition of the submarine air will move down along the 0.8
diagonal. If the ventilation is increased to 12 liters per man per minute, the
composition of the submarine air will be stabilized at 2% CO₂. Every time the
ventilation is doubled the CO₂ content of the air will be halved. At infinite
ventilation rate the submarine air remains of the same composition as "pure"
outdoor air. If at the 4% CO₂ point an effort is made to improve conditions by the
addition of pure oxygen (without the escape of any contaminated air — i.e., at
constant volume) the composition will change along a straight line drawn from the
4% point to the pure O₂ point at 0% CO₂ and 760 mm pO₂. Mixtures on this chart
always lie along the straight line connecting the two subject points.

Again starting at the 4% CO₂ point the habitability of the submarine can be
improved by mere absorption of CO₂. If this is done in a strictly closed vessel,
the pO₂ will not change, but if fresh air is allowed to enter in proportion to the
loss of CO₂ the composition changes along the arrow, as indicated, with a slight
increase in the pO₂. With a decrease of pressure in the submarine (such as that
produced by the activities of the snorkel pump when the inlet valve closes) the
partial pressures of both CO₂ and O₂ decrease along a line drawn from the 4%-
point to the origin where partial pressures of both O₂ and CO₂ are zero.

Finally it would be possible to improve conditions in this submarine by
pumping the air through a CO₂ absorber at a rate of say 12 liters per minute. In
this case the CO₂ would fall along the curved line stabilizing at 15 mm pCO₂ in 2 hours at the point where the CO₂ production just equals the CO₂ absorption. Unless oxygen is supplied, however, the pO₂ will continually fall until consciousness is lost. If this same clearance rate is maintained from the time breathing in this closed space is first begun, the composition changes along the curved dotted line as indicated previously in Figure 17.

If breathing in the closed space continues as indicated in Figure 16 without any ventilation or absorption of CO₂, the pO₂ may be expected to reach a dangerously low level of 50 mm when the pCO₂ reaches 75 mm or about 10% CO₂. Since 10% CO₂ produces narcosis by itself, it is difficult to say whether death will eventually result from hypercapnia or anoxia. Since these two hazards are additive rather than antagonistic in their effects, it is probable that both contribute to the eventually lethal effects.

p.) The N₂-CO₂ Diagram

In section (e) it was shown that the alveolar N₂ is a function not only of the exchange ratio but also of the concentration of CO₂ found in the alveolar gas. This relationship is given by Equation 5 and is plotted as the N₂-CO₂ diagram in Figure 19. The R lines radiate from the inspired nitrogen, PₑN₂. The R = 1.0 line is perpendicular indicating that PₑN₂ remains unchanged in the alveolar air at any pCO₂. R values less than 1 will yield PₑN₂ values greater than PₑN₂, (+), and vice versa (-). In addition the iso-ventilation lines are superimposed on Figure 19 similar to the ventilation lines of the transparent chart (Diagram IX). The alveolar ventilation equation (Equation 5) is expressed in terms of PₑN₂, R and oxygen consumption.

Figure 19 shows the typical pathway observed when the alveolar ventilation is reduced by increasing the apparatus dead space by 1300 cc (upper curve) (10). The solid dot indicates the normal resting alveolar gas tension. The solid line shows the alveolar pathway during the next 10 minutes which returns to an R of .8 and a pCO₂ of 46. Upon recovery (dotted line) the retained CO₂ is blown off, R increases temporarily to 1.2 and finally returns to the original starting values. The maximal N₂ concentration difference between dead space breathing and recovery is 28 mm or approximately 4% N₂ in the alveolar air. Even greater differences in PₑN₂ can be observed with hyperventilation followed by recovery. The lower loop shows the effects of 10 minutes of controlled breathing at a rate of slightly over 6 L of alveolar ventilation per 100 cc of O₂ uptake (10). The R values initially approach 1.8 and the alveolar values parallel the iso-ventilation line slowly approaching the new steady state (R = .8) as the CO₂ reserves are blown off. With recovery (dotted lines) the ventilation is reduced followed by very low R values and tremendous rise in PₑN₂.

The equations (No. 5) have been derived on the assumption that no N₂ is taken up or given off by the blood. On the other hand it is quite apparent from the pathways in Figure 8 that such a transfer of N₂ must take place and invalidate to
Uncontrolled Change
R = 0.8
Alveolar R = 0.8

Submarine Air
14.5% O₂ VR = 2
4.0% CO₂

Snorkel pressure decrease
Clearance of CO₂
12 L/min

Absorb CO₂

2 Hr
1 Hr

FIG. 18

FIG. 19
THE N₂ - CO₂ DIAGRAM
some extent these equations during the unsteady state. However, the transfer is quite small and amounts to 1.0 cc N₂ per minute per 10 mm Δ PN₂ for a cardiac output of 6 liters. Therefore, this transfer is inconsequential in altering the pN₂ calculated for the static N₂ state.

q.) CO₂ Inhalation

So long as the inspired air contains no CO₂ and the subject breathes air the ventilation and R diagrams, Nos. VIII and IX, are applicable and no further calculations are necessary. When the O₂ fraction of the inspired air is not 0.209 and the CO₂ fraction is not zero, the R diagonals and the iso-ventilation lines must be calculated by Equations 6 and 7, respectively.

In one special case, however, it is possible to use the transparent charts for R.Q. and ventilation lines provided herewith. This is the case where the CO₂ is increased by merely adding CO₂ to air as if a CO₂ tank were leaking into the inspired air. In this case the decrease in pO₂ is always 0.209 of the increase in pCO₂ and the slopes of the R lines remain unchanged irrespective of the amount of CO₂ added. Likewise the iso-ventilation lines (for constant O₂ intake) remain identical. This being so Diagram IX can be used for ventilation and Diagram VIII can be used for R. For the latter the origin of the R diagonals on the transparency placed over the proper pCO₂ and pO₂ values of the inspired air and the correct R can then be determined for any alveolar point.

This is illustrated in Figure 20. The original gas mixture (air) is at point H, the alveolar point at A on the 0.8 R diagonal. When CO₂ is added to the inspired air, this mixture changes along the line HGFE. Four identical sets of R.Q. diagonals are drawn with origins at H, G, F, and E. These could be traced from the transparent Diagram VIII if it were placed successively at these four points.

On the same chart is drawn a set of iso-ventilation lines as in the transparent Diagram IX. In this special case these lines apply to all the inspired air points along the line HGFE and can therefore be used without change for all 4 sets of R diagonals. From these lines it appears that the ventilation is 1.73 lit/min for each 100 cc of O₂ consumed per minute if the alveolar point is at A and the inspired air at H and the R.Q. = 0.8. To maintain the same alveolar pCO₂ at point K in the second set of R.Q. diagonals with the inspired air point at G, the ventilation would have to be 3.46 lit/min or twice as large. Actually the respiratory centers cannot entirely compensate for the 2.8% of CO₂ which is inspired and the ventilation increases only to 3.1 lit/min with the alveolar point at B. This is calculated from Gray's equation V̇AR = 0.4 pCO₂ - 15 which gives the alveolar ventilation ratio V̇AR in terms of the alveolar pCO₂. Similarly if the inspired air point is at E the ventilation would have to be infinite to keep the alveolar pCO₂ = 40. Actually the alveolar point will be at C on the observed line ABCD with a ventilation of about 8 lit/min per 100 cc O₂ consumed. Finally when the inspired CO₂ = 8.4% at E, the alveolar point after complete equilibration will be at D with 8.4% CO₂ and a ventilation rate of about 18 lit/min per 100 cc O₂ intake.
FIG. 20  CO₂ IN THE INSPIRED AIR
Thus as CO₂ slowly accumulates in the inspired air the alveolar point moves along this line ABCD, each point on that line (in the steady state) being on the 0.8 diagonal of a set of R.Q. diagonals which is to be thought of as moving up the inspired air line HGFE. Further as the inspired CO₂ increases, the ventilation increases and the alveolar and inspired air points move closer together.

Similarly, of course, any other set of R.Q. diagonals for an inspired oxygen fraction different from that in air could be used along the corresponding HGFE line for CO₂ dilution. If, however, CO₂ is added to air in exchange for the O₂ or for the N₂, or in any way which does not dilute both N₂ and O₂ equally, a new set of R.Q. diagonals will have to be calculated by Equation 6 and a new set of iso-ventilation lines by Equation 7.
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