CARBON DIOXIDE RETENTION DURING HYPOVENTILATION IN EXPERIMENTAL ANIMALS

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Hypercapnea and respiratory acidosis have been observed frequently during anesthesia in the past fifteen years. Beecher and Murphy (1), Ellison, Ellison, and Hamilton (2), Gibbon et al. (3), and Dripps and Severinghaus (4) have described various aspects of this problem. The basic cause of the hypercapnea, in most instances, has been hypoventilation, the resulting hypoxia having been corrected by administration of increased concentrations of inspired oxygen.

That severe hypercapnea has a deleterious effect is without dispute. Carbon dioxide has a narcotic action when its tension in arterial blood exceeds 60–70 mm. of mercury (5), and may act synergistically with anoxia, morphine, and the barbiturates in producing respiratory depression (6). Higher levels alone will produce complete anesthesia, and eventually death (7).

Hopkins, Anzola, and Clowes (8) have shown that experimental animals may survive periods of hypercapnea severe enough to abolish the brain waves, but if this depression is maintained for over 20 minutes the animals subsequently die of normovolemic shock.

A precipitous fall in blood pressure (9) or even ventricular fibrillation (10) may occur following rapid correction of severe hypercapnea by hyperventilation.

Some years ago we observed that oxygen rich anesthetic mixtures were sometimes required in order to maintain normal oxygen saturation, as measured by a recording earpiece oximeter, in patients undergoing surgery. This suggested the presence of hypoventilation, and with it, hypercapnea. A study was therefore undertaken, first to determine quantitatively the degree of hypercapnea and respiratory acidosis which develop in experimental animals following reduction of effective alveolar ventilation, adequate oxygenation being maintained; second, to determine how much the concentration of inspired oxygen need be increased in order to maintain a relatively constant arterial oxygen saturation (near 85 per cent), as the alveolar ventilation is reduced; and third, to evaluate the reliability and usefulness of the “alveolar equations” in predicting such changes.

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Methods

Mongrel dogs ranging in weight from 8 to 15 kg., given premedication of atropine sulfate, 0.05 to 0.1 mg./kg., were anesthetized with pentobarbital sodium, 30 mg./kg., intravenously. A Wood cuvette oximeter, arranged for continuous recording (11), was connected between a femoral artery and vein for determining arterial oxygen saturation. Heparin sodium, containing 10 mg./ml., was given to prevent clotting within the cuvette, 0.2 to 0.3 ml./kg. being given intravenously, and 0.2 ml. intramuscularly. An additional 0.1 ml./kg. was administered intravenously each hour. Auffed endotracheal tube or tracheal cannula was inserted, and dimethyl tubocurarine iodide was slowly administered by vein in sufficient dosage (ranging from 0.15 to 0.33 mg./kg.) to eliminate autonomous respiratory movements. At hourly intervals thereafter, an additional 0.10 mg./kg. was administered intravenously. Artificial respiration was maintained by a Starling-type variable stroke, variable speed respirator connected to the endotracheal tube or tracheal cannula. Tidal volume and respiratory rate were recorded by means of a Tissot spirometer equipped with kymograph connected to the inlet of the Starling respirator. Arterial blood pressure (3 animals) was measured by strain gauge connected to a femoral artery, and lead II electrocardiograms were recorded on a 4-channel ink writing oscillograph.* End-tidal carbon dioxide tensions were determined in 2 animals using a modified Rahn sampler connected to a recording infrared carbon dioxide analyzer.†

The experimental procedure was then started. Ventilation was regulated with the animal breathing air so that arterial pH was close to the normal value of 7.4. After 20 to 30 minutes equilibration, simultaneous arterial samples for total plasma carbon dioxide and pH were drawn, with tidal volume and respiratory rate being recorded. Alveolar ventilation was then reduced in stepwise fashion by decreasing the tidal volume, respiratory frequency being kept constant at 4 to 9/minute. With each reduction in ventilation, inspired oxygen tension, monitored by Pauling-type oxygen analyzer,‡ was increased just enough to maintain arterial oxygen saturation above 85 per cent.

Serial arterial pH determinations were performed until there was less than 0.02 pH unit change for a given concentration of inspired oxygen and tidal volume, at which time simultaneous samples of arterial blood were taken for pH and total carbon dioxide determinations. A Cambridge meter, § with 0.4 ml. glass electrode kept at 37 C. by means of a water jacket, was used for measuring, within five minutes of withdrawal, the pH of the blood samples. Total carbon dioxide was determined by the method of Van Slyke and Neill, blood samples being

* Grass Instrument Co., Quincy, Massachusetts.
‡ Arnold O. Beckman, Inc., South Pasadena, California.
kept in oiled plastic syringes in an ice bath prior to centrifugation. Bicarbonate concentration and partial pressure of carbon dioxide in plasma were then calculated using the Henderson-Hasselbalch equation, since the data were beyond the limits of the scales of available nomograms for blood.

**RESULTS**

Table 1 contains average data from 9 experiments, arranged in order of decreasing alveolar ventilation (VA), which was calculated from the expression: \( VA = (V_T - V_D) f \), where \( V_T \) and \( V_D \) refer to tidal and dead space volumes, and \( f \) to respiratory frequency, in breaths per minute. Attempts to calculate \( V_D \) by the Bohr dead space equation were abandoned because of the small minute volumes and high values for arterial PCO\(_2\) obtained during hypoventilation. \( V_D \) was then as-

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<td><strong>AVERAGE DATA OBTAINED DURING STEP-WISE REDUCTION OF TOTAL VENTILATION (( \dot{V}_T )) AND EFFECTIVE ALVEOLAR VENTILATION (( \dot{V}_A )) IN ANESTHETIZED, CURARIZED DOGS</strong></td>
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<td>Stages of Hypoventilation</td>
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Moist inspired oxygen tension (\( P\dot{O}_2 \)) was increased at each step just enough to keep arterial oxygen saturation (SaO\(_2\)) near 85 per cent. Predicted values for arterial CO\(_2\) tension (\( PaCO_2 \)) were calculated from the “ventilation-metabolism” equation \( \dot{V}_A = \frac{\dot{V}_T \cdot R \cdot 0.863}{\frac{PaCO_2}{PACO_2}} \), which specifies a reciprocal relation between \( PaCO_2 \) and \( \dot{V}_A \). Gas volumes are expressed as BTPS (body temperature and pressure, saturated).

\( \dot{V}_A \) was set to equal 50 ml and to be constant with decreasing tidal volumes, both of which are first approximations based on the data of Severinghaus and Stupfel (12) and of others quoted by them. (As stated below, subsequent work has indicated that \( V_D \) does vary directly with \( V_T \).)

Values obtained during the control period suggest the presence of slight hyperventilation (alveolar PCO\(_2\) = 37.6 mm.) with mild metabolic acidosis (pH = 7.34, [HCO\(_3^-\)] = 19.44 mM/L), possibly related to a 24 hour fast prior to experimentation. As could be anticipated, when ventilation was reduced, alveolar PCO\(_2\) increased, pH fell, and higher tensions of inspired oxygen were required to maintain arterial saturation near 85 per cent.

**Degree of Hypercapniae Following Reduction in Alveolar Ventilation.**—We can predict the shape of the curve relating alveolar ventila-
tion ($V_A$) to alveolar carbon dioxide tension ($PACO_2$) by means of the so-called “ventilation-metabolism” equation:

$$\dot{V}_A = \frac{\dot{V}O_2 \cdot R \cdot 0.863}{PACO_2}. \quad (1)$$

Briefly, this expression indicates a reciprocal relation between the amount of gas eliminated from the alveoli, and the tension of carbon dioxide in that gas.

Assuming that $\dot{V}O_2$ (oxygen consumption per minute) and $R$ (respiratory exchange ratio, equal to the respiratory quotient except in unsteady states of respiratory exchange, such as early in hyperventilation) are relatively constant throughout the experimental procedure, we can substitute $K_1$ for the numerator, and:

$$\dot{V}_A = \frac{K_1}{PACO_2}. \quad (1a)$$

Thus the curve should be of the form of a rectangular hyperbola.

In our experiments, arterial carbon dioxide tension ($PaCO_2$) was calculated rather than alveolar $PCO_2$. Because of the extremely small diffusion gradient for carbon dioxide across the lung, $PaCO_2$ may be considered (except in a few pathological conditions) to be equal to $PACO_2$. For this reason, these two terms will be used interchangeably henceforth in this paper.

Figure 1 shows the inverse relation between alveolar ventilation in liters per minute (abscissa) and arterial $PCO_2$ in mm. of mercury. Values for $PaCO_2$ on the experimental curve, while close to predicted values with moderate reduction in alveolar ventilation, become in-

![Fig. 1. Relation between “effective alveolar ventilation” ($V_A$) and arterial carbon dioxide tension ($PaCO_2$) showing how, with each reduction in ventilation, there is a proportionate rise in $PaCO_2$. The discrepancy between predicted and experimental values at low levels of alveolar ventilation is probably due to falsely low calculated values for the latter, as explained in text.](image-url)
creasingly less than the latter with severe reduction. The discrepancy is explained by the recent work of Williams and Rayford (13) and Severinghaus and Stupfel (14), both pairs of investigators finding a direct relation between physiological dead space and tidal volume in dogs. With the marked reduction in tidal volumes occurring during hypventilation in our experiments, \( \bar{V}_D \) would be less than the assumed value of 50 ml, hence true "effective alveolar ventilation" would be greater than the figure calculated from the expression: \( \bar{V}_A = (\bar{V}_T - \bar{V}_D) f \).

A further reason for the lower experimental values for \( PCO_2 \) could be the failure to reach a steady state, with carbon dioxide continuing to flow to tissues, there to be buffered. That this is probably a minor effect, however, is suggested by the fact that experimental points were not determined until blood pH reached essentially constant values (± 0.05 pH units), a process requiring over an hour when ventilation was severely reduced.

Relation of Inspired Oxygen Tension to Alveolar Ventilation.—Considering next the relation between inspired oxygen tension necessary to maintain a constant arterial oxygen saturation, and values for alveolar ventilation, we may again predict the shape of the curve. Starting with a simplified version of the "alveolar gas equation" (5):

\[
PAO_2 = PIO_2 - 1.14 PACO_2,
\]

(2)

where \( PIO_2 \) is the oxygen tension of moist inspired gas, and solving for \( PACO_2 \), we obtain:

\[
PACO_2 = \frac{PIO_2 - PAO_2}{1.14}.
\]

(2a)

Now, \( PAO_2 \) may be considered, as a first approximation, to be a constant, here \( K_2 \), if the arterial oxygen saturation is kept relatively constant, near 85 per cent. This is only an approximation because of the Bohr effect. Data for the latter are not available for tensions exceeding 90 mm. \( PCO_2 \), but assuming as a rough extrapolation that \( PO_2 \) had to be increased as much as 70 mm. to maintain saturation at 85 per cent with the extreme hypercapnea obtained, the error is rather small relative to values of inspired oxygen used (700 mm.).

Therefore:

\[
PACO_2 = \frac{PIO_2 - K_2}{1.14}.
\]

(2b)

Finally, by substituting this modified alveolar gas equation into the "ventilation metabolism" equation (1a), it is evident that the relation of alveolar ventilation to inspired oxygen is of the form of a rectangular hyperbola:

\[
\bar{V}_A = \frac{K_1 \cdot 1.14}{PIO_2 - K_2}.
\]

(3)
The experimental curve (fig. 2) turns out to be approximately hyperbolic in shape. No attempt was made to construct a curve for predicted values because of lack of data for the Bohr effect.

![Graph showing relation of inspired oxygen tension to arterial oxygen saturation](image)

**Fig. 2.** Relation of inspired oxygen tension needed to maintain arterial oxygen saturation near 85 per cent, to effective alveolar ventilation. The curve resembles a rectangular hyperbola, indicating an inverse relation between \( P_A \) and \( P_{O_2} \), as predicted by substitution in the alveolar equations (see text). In other words, hypoxia may be prevented, in the presence of reduced ventilation, only by a proportionate increase in inspired oxygen.

*Relation of Alveolar PCO₂, Hydrogen Ion Concentration, to Inspired Oxygen Tension.*—Turning next to the relation between hydrogen ion concentration and the inspired oxygen tension needed to maintain adequate oxygenation, we find that prediction of the shape of this curve necessitates use of an equation described by Gray (5) relating hydrogen ion concentration to arterial PCO₂:

\[
H^+ = 0.652 \cdot PaCO_2^{* + 13.5}.
\]

Substituting the previously modified alveolar gas equation (2b) in equation (4), one obtains:

\[
H^+ = 0.652 \left( \frac{P_{O_2} - K_z}{1.14} \right) + 13.5.
\]

This indicates that there should be a linear relation between hydrogen ion concentration and inspired oxygen tension under the conditions of the experiment. Whether this relation will apply at very high carbon dioxide tensions is, however, uncertain, since equation (4) is only known to apply for values of PaCO₂ up to 90 mm. The relation between alveolar PCO₂ and inspired oxygen tension should also be linear as shown by equation (2b) above.

The curve relating hydrogen ion concentration and inspired oxygen tension (fig. 3) proved in our experiments to be linear for oxygen tensions up to 400 mm. with a decreasing slope at higher tensions. The same is true of the curve relating arterial PCO₂ to inspired oxygen.
tension. The nonlinearity of portions of the curves is probably chiefly due to falsely low values for effective alveolar ventilation, as described on page 157. Here, too, no predicted curve was constructed, first because of lack of data for the Bohr effect, and second, because of the need for considerable extrapolation beyond the measured values for equation (4).

![Graph showing the relation between PaCO₂ (triangles) and hydrogen ion concentration in billions of a mole per liter (circles), to the inspired oxygen tension needed to maintain arterial oxygen saturation near 85 per cent as ventilation is reduced. Both curves appear to be linear for values of inspired oxygen tension up to 400 mm., as predicted from the equations given in text. These data indicate the inevitable consequences, that is, hypercapnea and acidosis, of hypventilation, even though adequate oxygenation is maintained by increasing concentrations of inspired oxygen.](image)

**Fig. 3.** Relation of arterial PCO₂ (triangles) and hydrogen ion concentration in billions of a mole per liter (circles), to the inspired oxygen tension needed to maintain arterial oxygen saturation near 85 per cent as ventilation is reduced. Both curves appear to be linear for values of inspired oxygen tension up to 400 mm., as predicted from the equations given in text. These data indicate the inevitable consequences, that is, hypercapnea and acidosis, of hypventilation, even though adequate oxygenation is maintained by increasing concentrations of inspired oxygen.

**Acid-Base Pathway.**—The general acid-base pathway followed during the experiment is shown on a pH-bicarbonate diagram (fig. 4). The data of Hastings and Steinhaus (15) indicate the normal buffer curve of dog blood, with a buffer value (Δ[HCO₃⁻]/ΔpH) of 25. Our experimental curve has a much flatter slope, equal to 14 in its linear portion, with further flattening at very low pH values, suggesting the development of a significant metabolic, as well as respiratory acidosis. In those of our experiments in which the hypercapnea was corrected by restoring normal ventilation, the plasma bicarbonate returned to near its control values. This is quite different from the situation existing when fixed acid is added to the blood, which leads to a deficit in bicarbonate. Lack of such a deficit is explained by the studies of Brown (16), who attributes the metabolic acidosis found in hypercapneic dogs at least partially to a reversible liberation of phosphate ions from the red blood cells.

**Cardiovascular Changes Observed During Hypercapnea.**—The blood pressure (average values from 3 experiments), rose from the con-
control value of 130/90 to 170/115 when alveolar $PCO_2 = 225$ mm. and $pH = 6.65$. As previously reported by Dripps (9), we found in preliminary experiments that if the animals were suddenly hyperventilated at this juncture, a precipitous, but reversible, fall in blood pressure occurred, reaching 50/0 within one minute. In the present experiments, in which the hypercapnea became progressively more severe, there was a gradual fall in blood pressure to 60/20, or lower, as $pH$ fell below 6.60 and alveolar $PCO_2$ rose above 300 mm.

![Graph showing pH-bicarbonate diagram](image)

Fig. 4. $pH$-bicarbonate diagram showing acid-base pathway followed during the increasingly severe hypercapnea produced by reducing effective alveolar ventilation, arterial oxygen saturation being maintained near 85% by increasing inspired oxygen tension. Data of Hastings and Steinhaus represent the normal buffer curve for dog blood. Flatness of the experimental curve suggests metabolic as well as respiratory acidosis.

Electrocardiograms (3 experiments) showed progressive depression of the S-T segment and lowering of the T wave, leading to T wave inversion. Such marked changes only occurred late in the procedure, after blood pressure had fallen to 60/20, or below. They are presumably due to stagnant anoxia, since hyperventilation at this time caused a prompt rise in blood pressure and reversion of the electrocardiogram to normal.

No electrocardiographic evidence of arrhythmias was present, but one animal died of cardiac arrest as a result of the hypoventilation procedure, even though arterial oxygenation was near 85 per cent.

*End-Tidal versus Arterial $PCO_2*—When alveolar ventilation was normal, values of $PCO_2$ in end-tidal gas samples were close to those calculated for arterial blood. With the reduction of ventilation, however, an ever increasing discrepancy was noted. End-tidal $PCO_2$ reached a maximum tension of 62 mm. (40 mm. lower than simultaneous arterial carbon dioxide) when alveolar ventilation had been reduced to one third of its control value. Upon decreasing ventilation to one tenth of its normal value, end-tidal $PCO_2$ gradually fell to 12 mm., as arterial $PCO_2$ rose to 246 mm. Manual compression of the animal’s chest at this time produced an end-tidal sample with a carbon dioxide tension greater than 125 mm.
DISCUSSION

In this study, the maximum arterial carbon dioxide tension obtainable through the retention of endogenous carbon dioxide during hypoventilation, with adequate arterial oxygen saturation being maintained by means of pure oxygen, was roughly 400 mm. Similarly, Brown (16) found that it was not possible to exceed this tension and maintain adequate oxygenation in dogs breathing mixtures of carbon dioxide and oxygen.

The pH consistently fell in our experiments from a control value near 7.4 to 6.5, but no lower, a figure close to that of 6.4 found by Brown to be the lowest value compatible with life in dogs breathing carbon dioxide in oxygen (16).

Certain logical consequences of the data presented here may have practical application in the field of anesthesiology, as follows: First, it is obvious, as predicted by the “ventilation-metabolism” equation, and as verified by the data plotted in figure 1, that a decrease in effective alveolar ventilation will produce a proportional increase in arterial carbon dioxide tension, provided oxygen consumption is relatively constant. Second, if it becomes necessary to increase the concentration of oxygen in the inspired air in order to maintain adequate oxygenation (assuming relatively normal cardiopulmonary function), then one has evidence of a proportional decrease in effective alveolar ventilation, as predicted by combination of the “ventilation-metabolism” equation with the “alveolar gas” equation, and as determined experimentally by the graph shown in figure 2. Third, in the situation where adequate oxygenation is maintained by a given increase in oxygen tension, then, as predicted first by the alveolar gas equation, and second by an equation relating PCO₂ to hydrogen ion concentration, there will be a roughly proportional increase in both factors, as confirmed by the experimental data shown in figure 3.

Thus, when hypoxia due to hypoventilation is corrected by adding oxygen, the inevitable consequence is retention of carbon dioxide and respiratory acidosis.

The data obtained with the carbon dioxide analyzer indicate that with increasingly severe hypoventilation, the discrepancy between PCO₂ of end-tidal samples and that of arterial blood increases to the point where end-tidal samples give absolutely no information regarding the PCO₂ of arterial blood. Unfortunately, there is no colorimetric method for measuring the PCO₂ of arterial blood, as there is in the case of the photoelectric oximeter for measuring oxygen saturation.

However, the data given above suggest that it might be possible to use the earpiece oximeter to prevent hypercapnea. The reasoning is as follows: If the oxygen saturation falls with the patient breathing air, then one has evidence of hypoventilation, most frequently due to depression of the respiratory centers by the anesthetic agent. Conversely, if the saturation can be brought back to near normal by means
of assistance to respiration, with the patient still breathing air, then ventilation is at least adequate. Thus, by keeping the concentration of oxygen in the anesthetic mixture relatively low, not over 21 to 25 per cent in patients with normal cardiopulmonary function, the oximeter might be used to help regulate assistance to respiration so as to prevent not only hypoxia, but also hypoventilation and hypercapnea.

In patients with abnormal cardiopulmonary function, whether due to disease, to the effects of opening the chest during surgery, or both, maintenance of adequate oxygenation will of course require addition of oxygen to the inspired air, even if ventilation is normal. But if oxygen saturation is to act as a sensitive measure of hypoventilation, only the minimal concentration necessary to maintain adequate saturation, between 90 and 95 per cent, should be used. Otherwise, the arterial oxygen tension will be high enough to lie on a flat part of the oxygen dissociation curve, and a considerable decrease in ventilation, capable of producing hypercapnea, will produce only a slight fall in saturation.

We have found in unanesthetized patients with severe emphysema that an oxygen saturation of between 90 and 95 per cent can usually be obtained with 30 to 50 per cent oxygen. Somewhat higher concentrations would be required with the chest open during surgery. A fall of 5 per cent or greater during the course of the operation should make one suspect hypoventilation.

With right-left anatomical shunts, whether due to congenital heart disease or A-V fistulas in the lung, administration of pure oxygen during surgery serves the useful purpose of raising oxygen saturation 10 to 12 per cent. But if the saturation drops, say 5 per cent or more from this higher value, hypoventilation may again be the cause.

Alternatively, a fall in saturation during open chest surgery may be due to an increased "shunt-like" effect resulting from circulation of blood through portions of lung poorly ventilated as a result of the varying degrees of collapse necessitated by the surgical procedure.

In order to distinguish whether a given fall in saturation was due to hypoventilation, or to increased "shunt-like" effect, the anesthesiologist need only increase assistance to respiration for a few minutes. A marked rise in saturation would indicate hypoventilation, whereas little or no rise would indicate increased shunt-like effect, necessitating higher concentrations of oxygen, even up to 100 per cent.

Although we have tested this proposed method for regulating assistance to respiration by means of the earpiece oximeter in only a small number of cases, we believe the rationale is sound, and that the application should be further evaluated in clinical practice. In the hands of one experienced in its use, it is our opinion that the Wood absolute reading ear oximeter is accurate enough for such purposes, especially if set before induction of anesthesia to read 100 per cent when the patient is breathing pure oxygen (except with right-left anatomical shunts) and if recalibrated every 15 to 30 minutes.
It should be mentioned here that when a fast acting carbon dioxide analyzer is used during anesthesia its usefulness is enhanced by assistance to respiration. Thus, with ample tidal volumes, the dead space gas of each breath is expelled, and the carbon dioxide tension of end-tidal samples then approaches that of true alveolar gas and of arterial blood. Although in severe emphysema the \( PCO_2 \) of such samples may be lower by 10 mm. or more than that of arterial blood, the analyzer provides an approximate measure of the \( PCO_2 \) of arterial blood.

By contrast, during hypoventilation in our experiments, with adequate oxygenation being maintained, values of \( PCO_2 \) of end-tidal samples were one-tenth or less of those for arterial blood, and were therefore both meaningless and misleading.

**Summary**

Alveolar ventilation was reduced in step-wise fashion, in dogs, and inspired oxygen was simultaneously increased just enough to keep arterial saturation, measured by cuvette oximeter, near 85 per cent. Under these conditions:

- There is an approximately reciprocal relation between effective alveolar ventilation and arterial \( PCO_2 \).
- A similar reciprocal relationship also exists between alveolar ventilation and the inspired oxygen tension (minimal \( PIO_2 \)) required to maintain oxygen saturation near 85 per cent.
- There is an approximately linear relation between minimal \( PIO_2 \) and both arterial \( PCO_2 \) and hydrogen ion concentration.

With extreme reduction in ventilation requiring 100 per cent oxygen to maintain adequate oxygenation, arterial \( PCO_2 \) reached nearly 400 mm., and \( pH \) 6.5, the acidosis having both respiratory and metabolic components.

The possibility is raised of using an earpiece oximeter as an aid in regulating assistance to respiration to prevent hypoventilation, adequate oxygenation being maintained in the majority of patients on considerably less than 100 per cent inspired oxygen.

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**References**