PCO_2 and alveolar ventilation

VD/VT AND THE BOHR DEAD SPACE EQUATION

The normal ratio of dead space to tidal volume (VD/VT or VD/VE, which is the same thing) is approximately 150 ml VD/500 ml VT, or 0.3. Normal VD/VT ranges from approximately 0.28 to 0.33.

As pointed out previously, VD/VT can be elevated from either a reduction of VT or an actual increase in VD. Either cause of increased VD/VT can cause a decrease in alveolar volume (VA) and hence in alveolar ventilation (VA).

When the cause of increased VD/VT is lung disease, ventilatory adaptations will try to keep VA and $PaCO_2$ normal (Table 41). These adjustments of course require an intact central nervous system and intact chest bellows. When ventilatory adaptations fail, VA will fall and $PaCO_2$ will rise.

On occasion it is useful to quantitate the VD/VT. This can be done using the Bohr dead space equation:

$$\frac{VD}{VT} = \frac{PaCO_2 - PeCO_2}{PaCO_2} \quad \textbf{(Eqn 4-11)}$$

where $PeCO_2$ is the mean expired carbon dioxide pressure, which is obtained from an expired air sample that is collected over a few minutes time. Normal $PeCO_2$ is approximately 28 mm Hg. Thus 40--28/40 = 0.30.

Clinical problem 11
During an attempt to wean a patient from the use of artificial ventilation, her $PaCO_2$ and PeCO2 are measured with the following results: $PaCO_2$, 56 mm Hg; $PeCO_2$, 26 mm Hg. What is the VD/VT? Apart from any other factors, does this ratio indicate the patient can be removed from the ventilator?

DANGERS OF HYPERCAPNIA

The dangers from an elevated $PaCO_2$ are usually not from the excess carbon dioxide per se. In fact carbon dioxide is a respiratory stimulant until very high $PaCO_2$ values are reached (90 mm Hg or greater), at which time carbon dioxide may depress breathing. In addition to indicating a state of respiratory failure, there are three distinct dangers associated with an elevated $PaCO_2$.

Low PaO_2 from high $PaCO_2$. For a constant fraction of inspired oxygen (FIO_2), as the $PaCO_2$ rises, the alveolar oxygen pressure (PAO_2) falls, roughly on a mm Hgformm Hg basis. This change can be appreciated from the relationship of $PaCO_2$ to PAO_2 in the alveolar air equation (discussed extensively in Chapter 5); in addition a fall in PAO_2, results in a corresponding drop in PaO_2. Although this cause of hypoxemia can often be corrected by judicious use of supplemental oxygen, there are situations in which a further rise in $PaCO_2$ will cause PaO_2 to fall to a dangerous level.

Low pH from high $PaCO_2$. A rise in $PaCO_2$ will lead to a fall in pH; this can be seen in the relationship of $PaCO_2$ to pH (Henderson-Hasselbalch equation; see Fig. 45 and Chapter 7). Acidemia is a potential trigger of cardiac arrhythmias. Although the critical level for hydrogen ion concentration varies in each situation, an arterial pH below 7.30 that is not improving should be considered potentially lifethreatening.

Decreased ventilatory reserve. Finally, a high PaCO2 represents a precarious situation in terms of ventilatory reserve. Small changes in alveolar ventilation (VA) that would be inconsequential in a healthy individual can be disastrous in someone retaining carbon dioxide. The reason why can be seen in the hyperbolic relationship when VA is plotted against $PaCO_2$ (Fig. 43). For example, when carbon dioxide production (VCO_2) is 200 ml/ min, a 500 ml/min decrease in VA (which may occur during central nervous system depression) will increase $PaCO_2$ only 5 mm Hg, from a normal baseline $PaCO_2$ of 40 mm Hg (VA = 4.3 L/min) to 45 mm Hg (VA = 3.8 L/min). When the baseline $PaCO_2$ is 60 mm Hg (VA = 2.9 L/min), the same 500 ml/min decrease will elevate $PaCO_2$ by 12.5 mm Hg to 72.5 mm Hg (VA = 2.4 L/min). The resultant changes in PaO_2 and pH will also be amplified.

Clinical problem 12
For the following two patients, an initial VA and PaCO2 are given. Assuming constant VCO_2 of 200 ml/min, what is the new $PaCO_2$, after the described change in VA? Patient A: VA = 6 $PaCO_2$ = 29 VA decreases by 1 L/min L/min mm Hg because of administra tion of anesthesia Patient B: VA = 3 $PaCO_2$ = 57.5 VA decreases by 1 L/min L/min mm Hg because of pulmonary edema

$PaCO_2$ AND NEED FOR VENTILATORY ASSISTANCE

Because hyperventilation represents an excess of alveolar ventilation (VA), intubation to increase VA is never needed if the $PaCO_2$ is low. (Patients with low $PaCO_2$ may need artificial ventilation to help correct other problems, e.g. hypoxemia or severe alkalosis.)

Ventilatory assistance is used only for patients with elevated PaCO2 or in those unusual situations where $PaCO_2$ is in the normal range but the patient is in imminent danger of lifethreatening hypoventilation. Ventilatory assistance usually requires tracheal intubation and connection to an artificial respirator. Alternate methods of directly augmenting VA, such as by using a tightfitting face mask and intermittent positive pressure breathing, are almost never satisfactory in adults needing ventilatory assistance.

Figure 4-3

Fig. 43. $PaCO_2$ vs. alveolar ventilation (VA). The relationship is shown for carbon dioxide production rates of 200 ml/min and 300 ml/min. A decrease in alveolar ventilation (VA) in the hypercapnic patient will result in a greater rise in $PaCO_2$ than will the same VA change when $PaCO_2$ is low or normal. Also, an increase in carbon dioxide production when VA is fixed will result in an increase in $PaCO_2$.

Given an elevated $PaCO_2$, a good clinical rule is *never* intubate for hypercapnia alone. Different durations of hypercapnia, different buffering capacities, oxygen levels, and a host of other variables influence the need for ventilatory assistance. In the presence of hypercapnia, intubation and artificial ventilation are indicated only if one or more of the following are also present and judged to be life threatening:

1. Decreased mental status, not improving and potentially worsening
2. Increased fatigue, not improving and potentially worsening
3. Low pH (usually less than 7.30), not improving and potentially worsening
4. Low arterial oxygen pressure (PaO_2) that cannot otherwise be improved except by lowering the PaCO2
5. Secretions or mucus that is threatening upper airways patency

Obviously, a great deal of clinical judgment must enter into the decision to intubate a patient. Once a patient is intubated, $PaCO_2$ can only be followed by specific measurement for the reasons given previously. Even though the tidal volume and the respiratory rate are set by the ventilator and minute ventilation is therefore known, VA and carbon dioxide production remain unknown. Furthermore, in the nonstable patient, one cannot assume a constant dead space ventilation (VD) since changing tidal volume, respiratory rate, and the time course of acute parenchymal disease (changing V/Q relationships) may influence VD throughout a patient's course.

Weaning a patient from the ventilator also involves careful observation of his arterial blood gases and, on occasion, measurements of lung mechanics (see Chapter 10). It is impossible to give specific guidelines about how often to obtain blood gas analysis; this decision has to be individualized for each patient's course. A single blood gas measurement following intubation may not reflect a stable $PaCO_2$; generally at least two or more blood gas measurements should be obtained in the first few hours after intubation to help assure that the patient has reached a ventilatory steady state.

NON INVASIVE MEASUREMENT OF PCO_2

Because carbon dioxide is never diffusion limited, alveolar carbon dioxide pressure ($PACO_2$) is assumed equal to arterial carbon

dioxide pressure ($PaCO_2$). In theory, measurement of $PACO_2$ could substitute for $PaCO_2$, although in practice this is not always the case.

Figure 44A shows a normal tracing of partial pressure of carbon dioxide (PCO_2) measured during a single expired tidal volume with an infrared carbon dioxide analyzer. The first part of

the expired air is the same as the last part that was *inspired* on the previous breath; (it is deadspace air from the upper airways and will contain almost no carbon dioxide). Gradually, air from some of the alveoli begins to join this deadspace air, and the PCO_2 rises. By the very end of exhalation all the deadspace air has left the lungs, and the last few milliliters of air are from the alveoli only. This tracing shows that the endtidal PCO_2 ($PetCO_2$) is approximately 38 mm Hg, which indicates a normal $PaCO_2$.

Figure 4-4

Fig. 44. **A**, Carbon dioxide measurement during a single expired breath. In this example from a healthy patient, the endtidal point reflects alveolar, and hence arterial, partial pressure of carbon dioxide. **B,** Continuous monitoring of endtidal carbon dioxide ($PetCO_2$). This patient has severe chronic obstructive pulmonary disease. Some variation is seen during quiet breathing, but average $PetCO_2$ is approximately 50 mm/Hg. $PaCO_2$ measured at the same time was 74 mm Hg.

$PetCO_2$ can be measured on a continuous basis (Fig. 44, *B),* but the measurement has limitations. One has to assure that the carbon dioxide cannula, which delivers the expired air to the carbon dioxide analyzer, is not contaminated with room air. This is not so much of a problem with intubated patients for whom the cannula is inserted in the ventilator's expiratory circuit as it is in other patients.

Perhaps the major pitfall is the difficulty of obtaining true $PACO_2$ in patients with severe lung disease. In such cases $PetCO_2$ may not reflect alveolar and arterial PCO_2 because of severe ventilation/perfusion imbalance and a resulting large increase in physiologic dead space (see Chapter 5). In the example shown in Fig. 44. *B,* from a patient with severe chronic obstructive pulmonary disease, the $PetCO_2$ averaged approximately 50 mm Hg, but $PaCO_2$ was 74 mm Hg, resulting in a $PaCO_2PetCO_2$ difference of 24 mm Hg. In this situation the diseased alveoli do not empty evenly, and the end tidal sample still reflects considerable dead space air.

A $PaCO_2PetCO_2$ difference does not obviate the value of the endtidal measurement for physiologic monitoring; a rise in $PetCO_2$ still suggests a rise in $PaCO_2$, but one cannot equate the measured $PetCO_2$ with $PaCO_2$. For physiologic monitoring of critically ill patients, one or two comparisons should be made of $PetCO_2$ with $PaCO_2$ before following the $PetCO_2$ trend.

The absolute value of the $PaCO_2PetCO_2$ difference has also been advocated for diagnostic purposes, especially in acute pulmonary embolism where the value is often much higher than in chronic lung conditions. The pulmonary embolus creates extra dead space by blocking perfusion to a group of alveoli. However, because of a lack of specificity, this measurement is not widely used in clinical practice.

$PaCO_2$ - ITS RELATIONSHIP TO OXYGENATION AND ACIDBASE BALANCE

Any discussion of gas exchange should begin with $PaCO_2$ since it is the only blood gas value that provides information on ventilation, oxygenation, and acidbase balance. Fig. 45 shows the relationship of $PaCO_2$ to alveolar ventilation (the $PaCO_2$ equation), alveolar partial pressure of oxygen (the alveolarair equation; see Chapter 5), and pH (HendersonHasselbalch equation; see Chapter 7).

Figure 4-5

Fig. 45. Arterial carbon dioxide pressure ($PaCO_2$) in ventilation, oxygenation, and acidbase equations. A rise in $PaCO_2$ indicates diminished VA in relation to VCO_2, and will result in a fall in PaO_2 and pH. See Chapters 5 and 7.

SUMMARY

For gas exchange to occur fresh air must be brought into the alveoli. Alveolar ventilation (VA) is defined as the amount of fresh air that enters the alveoli *and* takes part in gas exchange; it is the difference between total or minute ventilation (VE) and the amount of air that does not take part in gas exchange--the deadspace ventilation (VD).

VA is inversely related to the partial pressure of carbon dioxide in arterial blood ($PaCO_2$) and is directly related to metabolic carbon dioxide production (VCO_2). When VA rises proportionately higher than VCO_2, $PaCO_2$ is reduced, a condition known as hyperventilation; conversely, a level of VA proportionately lower than normal will raise $PaCO_2$ (hypoventilation).

By employing a constant (0.863) to equate the different units for PaCO2, VA, and VCO_2, the three variables can be related thus: $PaCO_2 = VCO_2 \times 0.863/VA$. Normally, VA will rise to match any increase in VCO_2. During mild to moderate exercise, both VA and VCO_2 increase proportionately, so that $PaCO_2$ stays the same; the exercising person neither hyperventilates nor hypoventilates.

Based on this equation, it follows that hypercapnia is always caused by a level of VA that is inadequate for VCO_2. Furthermore, since VA = VE VD, all cases of hypercapnia can be seen as caused by a reduced or inadequate VE, or an elevated VD (or a combination of the two). Hypercapnia caused by drug overdose, for example, can be explained by a reduction in VE. Hypercapnia in chronic obstructive lung disease can be explained by an elevation of VD.

The most common cause of elevated VD is ventilation perfusion imbalance. Deadspace ventilation can also be elevated in states of rapid shallow breathing, in which a largerthannormal proportion of each tidal volume goes to satisfy anatomic dead space.

$PaCO_2$ is a key blood gas measurement. Not only does it help assess adequacy of VA, but it is also a component of the alveolar air equation and the HendersonHasselbalch equation.

REVIEW QUESTIONS

State whether each of the following are true or false.

1. To estimate $PaCO_2$ at the bedside, one can start with a measurement of 40 mm Hg, then subtract 2 mm Hg for every breath above 10/min.

2. $PaCO_2$ is inversely related to alveolar ventilation.

3. $PaCO_2$ is directly related to level of carbon dioxide production.

4. $PaCO_2$ is always low if the alveolar ventilation is twice the resting level.

5. Normally, one can voluntarily hyperventilate to lower $PaCO_2$ more than 10 mmHg.

6. Normally, one can voluntarily hypoventilate to raise $PaCO_2$ more than 10 mmHg.

7. Deadspace ventilation can rise solely from a change in the pattern of breathing, i.e., without a change in the lung architecture.

8. Most of the blood carbon dioxide is carried in the form of bicarbonate.

9. To calculate the ratio of dead space to tidal volume using the Bohr equation, one need measure only tidal volume and $PaCO_2$.

10. As $PaCO_2$ goes up, alveolar PO_2 goes down.

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