Section VII

ACUTE RESPIRATORY FAILURE

I breathe for my own necessity, for my survival.

Ayn Rand
The Fountainhead
1943

Chapter 20

HYPOXEMIA AND HYPERCAPNIA

> Respiration is thus a process of combustion, in truth very slow, but otherwise exactly like that of charcoal.
>
> Antoine Lavoisier

Antoine Lavoisier was an 18th-century French scientist who was the first to identify oxygen as the essential element for metabolism, and the first to discover that aerobic metabolism is essentially a combustion reaction, where oxygen reacts with an organic fuel and produces carbon dioxide as a by-product. (One of the many tragedies of the French Revolution was the senseless beheading of Antoine Lavoisier in 1794.) Providing the oxygen and removing the carbon dioxide is the responsibility of the lungs, and this chapter describes how the lungs perform this task, and how abnormalities in lung function can lead to deficits in arterial oxygenation (hypoxemia) and accumulation of carbon dioxide (hypercapnia). The last part of the chapter presents a physiological approach to the evaluation of hypoxemia and hypercapnia in individual patients.

PULMONARY GAS EXCHANGE

The efficiency of gas exchange in the lungs is determined by the balance between alveolar ventilation and pulmonary capillary blood flow (1–4). This balance is commonly expressed as the ventilation–perfusion (V/Q) ratio. The influence of V/Q ratios on pulmonary gas exchange can be described using an alveolar–capillary unit, as shown in Figure 20.1. The upper panel shows a perfect match between ventilation and perfusion (V/Q = 1). This is the reference point for defining the abnormal patterns of gas exchange.

Dead Space Ventilation

A V/Q ratio above 1.0 (Fig. 20.1, middle panel) describes the condition where ventilation is excessive relative to pulmonary capillary blood flow. The excess ventilation, known as dead space ventilation, does not participate in gas exchange with the blood. Dead space ventilation includes anatomic dead space, which is the gas in the large conducting airways that does not come in contact with capillary blood (about half of the anatomic dead space is in the pharynx), and physiologic dead space, which is alveolar gas that does not equilibrate fully with capillary blood. In normal subjects, dead space ventilation (V_D) accounts for 20% to 30% of the total ventilation (V_T); i.e., V_D/V_T = 0.2 to 0.3 (1,3).

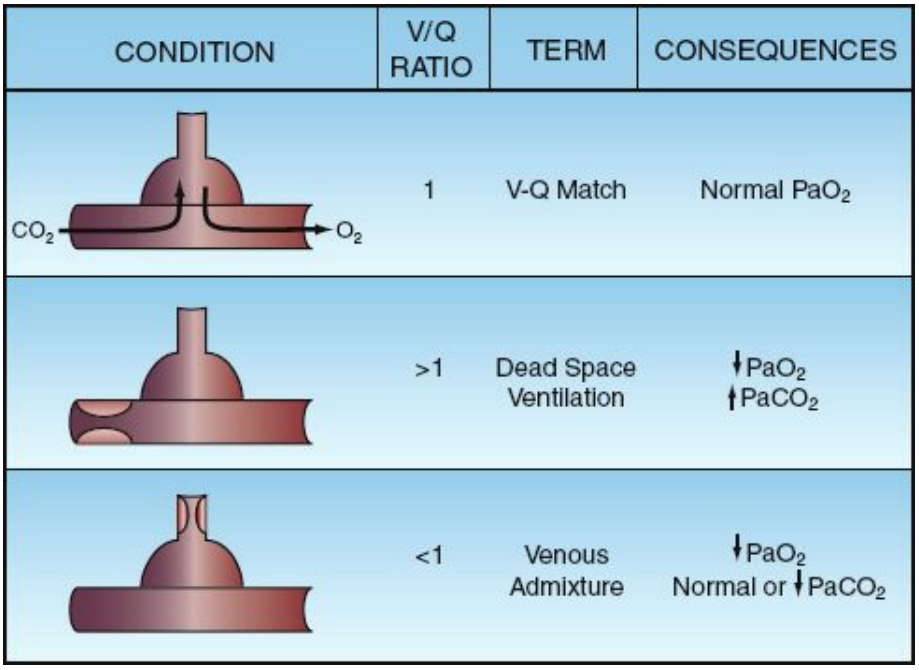

FIGURE 20.1 Ventilation–perfusion (V/Q) relationships and associated blood gas abnormalities.

Pathophysiology

Dead space ventilation increases in the following situations:
1. When the alveolar–capillary interface is destroyed; e.g., emphysema
2. When blood flow is reduced; i.e., low cardiac output
3. When alveoli are overdistended; e.g., during positive-pressure ventilation

ARTERIAL BLOOD GASES: An increase in V_D/V_T above 0.3 results in both hypoxemia (decreased arterial PO_2) and hypercapnia (increased arterial PCO_2), which is analogous to what would happen if you held your breath. The hypercapnia usually appears when the V_D/V_T is above 0.5 (5).

Intrapulmonary Shunt

A V/Q ratio below 1.0 (Fig. 20.1, lower panel) occurs when pulmonary capillary blood flow is excessive relative to ventilation. The excess blood flow, known as intrapulmonary shunt, does not participate in pulmonary gas exchange. There are two types of intrapulmonary shunt. True shunt indicates the total absence of gas exchange between capillary blood and alveolar gas (V/Q = 0), and is equivalent to an anatomic shunt between the right and left sides of the heart. Venous admixture represents the capillary flow that does not equilibrate completely with alveolar gas (0<V/Q <1). As the venous admixture increases, the V/Q ratio decreases until it becomes a true shunt (V/Q = 0).

The fraction of the cardiac output that represents intrapulmonary shunt is known as the shunt fraction. In normal subjects, intrapulmonary shunt flow (Qs) represents less than 10% of the total cardiac output (Qt), so the shunt fraction (Qs/Qt) is less than 10% (1,2,4).

Pathophysiology

Intrapulmonary shunt fraction is increased in the following situations:

1. When the small airways are occluded; e.g., asthma
2. When the alveoli are filled with fluid; e.g., pulmonary edema, pneumonia
3. When the alveoli collapse; e.g., atelectasis
4. When capillary flow is excessive; e.g., in nonembolized regions of the lung in pulmonary embolism

ARTERIAL BLOOD GASES: The influence of shunt fraction on arterial O_2 and CO_2 tensions (PaO_2, $PaCO_2$, respectively) is shown in Figure 20.2. The PaO_2 falls progressively as shunt fraction increases, but the $PaCO_2$ remains constant until the shunt fraction exceeds 50% (4). The $PaCO_2$ is often below normal in patients with increased intrapulmonary shunt as a result of hyperventilation triggered by the disease process or by the accompanying hypoxemia.

Inhaled Oxygen

The shunt fraction also determines the influence of inhaled oxygen on the arterial PO_2. This is shown in Figure 20.3 (4). As intrapulmonary shunt increases from 10 to 50%, an increase in fractional concentration of inspired oxygen (FIO_2) produces less of an increment in the arterial PO_2. When the shunt fraction exceeds 50%, the arterial PO_2 is independent of changes in FIO_2, and the condition behaves like a true (anatomic) shunt. This means that, in conditions associated with a high shunt fraction (e.g., acute respiratory distress syndrome), the FIO_2 can often be lowered to non-toxic levels (FIO_2 below 60%) without further compromising arterial oxygenation. This can be a valuable maneuver for preventing pulmonary oxygen toxicity.

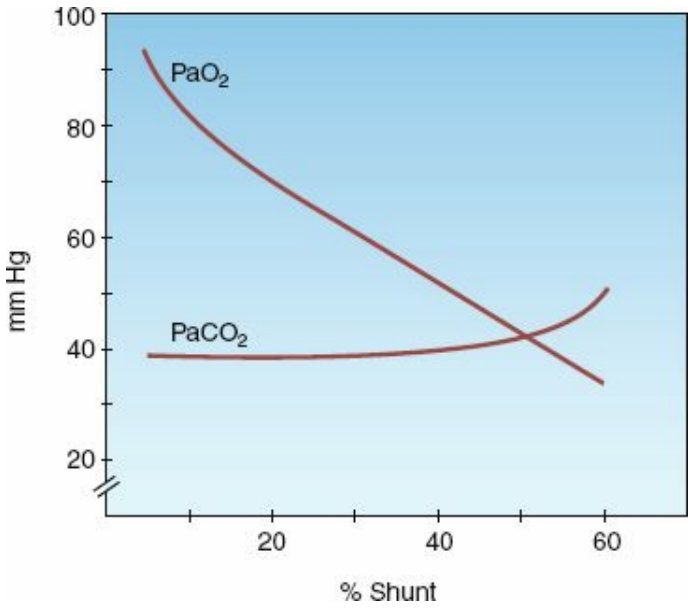

FIGURE 20.2 The influence of shunt fraction on arterial PO_2 (PaO_2) and arterial PCO_2 ($PaCO_2$). From Reference 4.

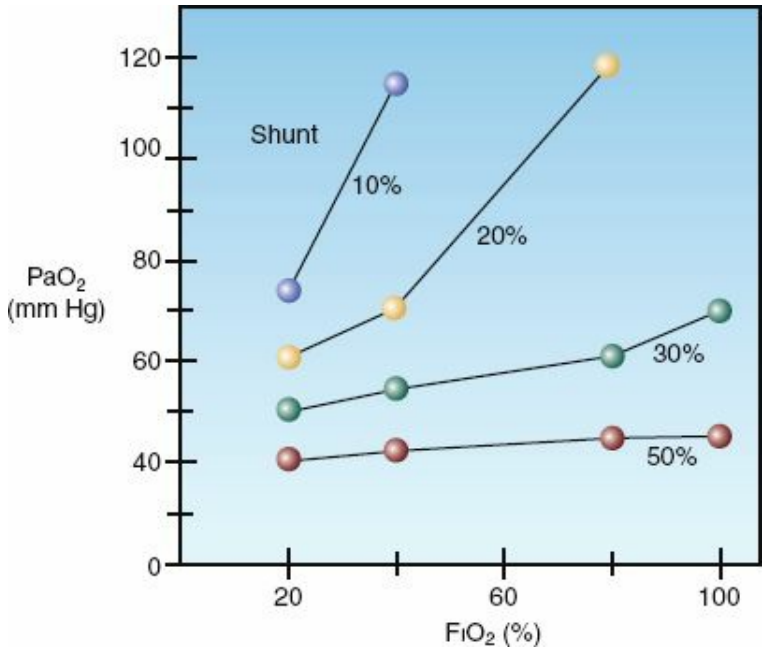

FIGURE 20.3 The influence of shunt fraction on the relationship between the inspired oxygen (FIO_2) and the arterial PO_2 (PaO_2). From Reference 4.

MEASURES OF GAS EXCHANGE

The calculation of dead space ventilation (V_D/V_T) is based on the difference between the PCO_2 in exhaled gas and end-capillary (arterial) blood. In the normal lung, the capillary blood equilibrates fully with alveolar gas, and the exhaled PCO_2 ($PECO_2$) is equivalent to the arterial PCO_2 ($PaCO_2$). As dead space ventilation (V_D/V_T) increases, the $PECO_2$ decreases relative to the $PaCO_2$. The Bohr equation shown below (derived by Christian Bohr, father of Neils Bohr, one of the founders of quantum mechanics) is based on this principle.

$$V_D / V_T = \frac{PaCO_2 - PECO_2}{PaCO_2} \quad \textbf{(20.1)}$$

Thus, when the $PECO_2$ decreases relative to the $PaCO_2$, the calculated V_D/V_T rises. The $PECO_2$ is measured in a random sample of expired gas (mean ex-haled PCO_2), and is not measured at the end of expiration (end-tidal PCO_2).

Intrapulmonary Shunt Fraction

The intrapulmonary shunt fraction (Qs/Qt) is derived by the relationship between the O_2 content in arterial blood (CaO_2), mixed venous blood (CvO_2), and pulmonary capillary blood (CcO_2).

$$Qs/Qt = \frac{CcCO_2 - CaCO_2}{CcCO_2 - CvCO_2} \quad \textbf{(20.2)}$$

The problem with this formula is the inability to measure the pulmonary capillary O_2 content (CcO_2) directly. As a result, pure oxygen breathing (to produce 100% oxyhemoglobin saturation in pulmonary capillary blood) is recommended for the shunt calculation. However in this situation, Qs/Qt measures only true shunt.

The A-a PO_2 Gradient

The PO_2 difference between alveolar gas and arterial blood (PAO_2–PaO_2) is an indirect measure of ventilation–perfusion abnormalities (5–7). The PAO_2–PaO_2 (A-a PO_2) gradient is determined with the alveolar gas equation shown below.

$$P_AO_2 = P_IO_2 - (PaCO_2 / RQ) \quad \textbf{(20.3)}$$

This equation defines the relationship between the PO_2 in alveolar gas (PAO_2), the PO_2 in inhaled gas (PIO_2), the PCO_2 in arterial blood (PaCO2), and the respiratory quotient (RQ). The RQ defines the relative rates of exchange of O_2 and CO_2 across the alveolar–capillary interface: i.e., RQ = VCO_2/VO_2. The PIO_2 is determined using the fractional concentration of inspired oxygen (FIO_2), the barometric pressure (P_B), and the partial pressure of water vapor (P_{H2O}) in humidified gas:

$$P_IO_2 = F_IO_2 (P_B - P_{H_2O}) \quad \textbf{(20.4)}$$

If equations 20.3 and 20.4 are combined (for the alveolar PO_2), the A–a PO_2 gradient can be calculated as follows:

$$\text{A-a } PO_2 = [F_IO2 (P_B - P_{H_2O}) - (PaCO_2/RQ)] - PaO_2 \quad \textbf{(20.5)}$$

In a healthy subject breathing room air at sea level, FIO_2=0.21, P_B=760 mm Hg, P_{H2O}=47 mm Hg, PaO_2=90 mm Hg, $PaCO_2$=40 mm Hg, and RQ=0.8:

$$\text{A-a } PO_2 = [0.21 (760 - 47) - (40/0.8)] - 90 = 10 \text{ mm Hg} \quad \textbf{(20.6)}$$

This represents an idealized rather than normal A-a PO_2 gradient, because the A-a PO_2 gradient varies with age and with the concentration of inspired oxygen.

Influence of Age

As shown in Table 20.1, the normal A-a PO_2 gradient rises steadily with advancing age (6). Assuming that most adult patients in an ICU are 40 years of age or older, the normal A-a PO_2 gradient in an adult ICU patient can be as high as 25 mm Hg when the patient is breathing room air. However, few ICU patients breathe room air, and the A-a PO_2 gradient is increased further when oxygen is added to inhaled gas (see next).

Table 20.1 Normal Arterial Blood Gases

Age (Years)	PaO_2 (mm Hg)	$PaCO_2$ (mm Hg)	A-a PO_2 (mm Hg)
20	84–95	33–47	4–17
30	81–92	34–47	7–21
40	78–90	34–47	10–24
50	75–87	34–47	14–27
60	72–84	34–47	17–31
70	70–81	34–47	21–34
80	67–79	34–47	25–38

All values pertain to room air breathing at sea level.

From the Intermountain Thoracic Society Manual of Uniform Laboratory Procedures. Salt Lake City, 1984:44–45.

Influence of Inspired Oxygen

The influence of inspired oxygen on the A-a PO_2 gradient is shown in Figure 20.4 (7). The A-a PO_2 gradient increases from 15 to 60 mm Hg as the FIO_2 increases from 21% (room air) to 100%. According to this relationship, the normal A-a PO_2 gradient increases 5 to 7 mm Hg for every 10% increase in FIO_2. This effect is presumably caused by the loss of regional hypoxic vasoconstriction in the lungs. Hypoxic vasoconstriction in poorly ventilated lung regions diverts blood to more adequately ventilated regions, and this helps to preserve the normal V/Q balance. Loss of regional hypoxic vasoconstriction during supplemental O_2 breathing maintains blood flow in poorly ventilated lung regions, and this increases intrapulmonary shunt fraction and increases the A-a PO_2 gradient.

The FIO_2 is difficult to estimate accurately when supplemental O_2 is delivered via nasal prongs or "open" face masks (see Chapter 22), and this limits the accuracy of the A-a PO_2 gradient in these situations.

Positive-Pressure Ventilation

Positive-pressure mechanical ventilation elevates the pressure in the airways above the ambient barometric pressure. Therefore, when determining the A-a PO_2 gradient in a ventilator-dependent patient, the mean airway pressure should be added to the barometric pressure (8). In the example presented in equation 20.6, a mean airway pressure of 30 cm H_2O would increase the A-a PO_2 gradient from 10 to 16 mm Hg (a 60% increase). Thus, neglecting the contribution of positive airway pressure during mechanical ventilation will underestimate the degree of abnormal gas exchange.

The a/A PO_2 Ratio

Unlike the A-a PO_2 gradient, the a/A PO_2 ratio is relatively unaffected by the FIO_2. This is demonstrated in Figure 20.4. The independence of the a/A PO_2 ratio in relation to the FIO_2 is explained by the equation below.

$$a/A\ PO_2 = 1 - (A\text{-}a\ PO_2)/P_AO_2 \quad \textbf{(20.7)}$$

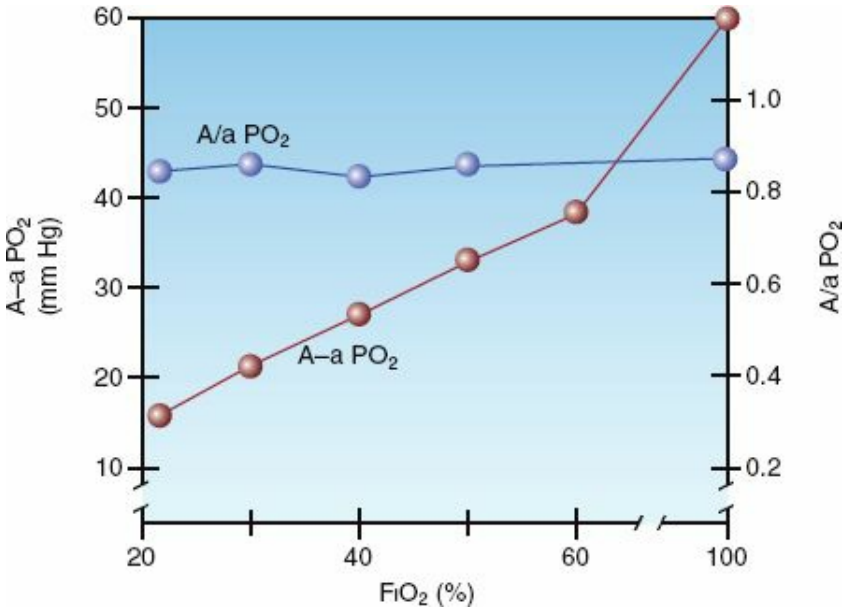

FIGURE 20.4 The influence of FIO_2 on the alveolar-arterial PO_2 gradient (A-a PO_2) and the arterial-alveolar PO_2 ratio (a/A PO_2) in normal subjects. From Reference 7.

Because the alveolar PO_2 is in both the numerator and denominator of the equation, the influence of FIO_2 on the PAO_2 is eliminated. Thus, the a/A PO_2 ratio is a mathematical manipulation that eliminates the influence of FIO_2 on the A-a PO_2 gradient. The normal a/A PO_2 ratio is 0.74 to 0.77 when breathing room air, and 0.80 to 0.82 when breathing 100% oxygen (7).

The PaO_2/FIO_2 Ratio

The PaO_2/FIO_2 ratio is used as an indirect estimate of shunt fraction. The following correlations have been reported (9).

PaO_2/FIO_2	Qs/Qt
<200	>20%
>200	<20%

The major limitation of the PaO_2/FIO_2 ratio is the inability to estimate the FIO_2 accurately when supplemental O_2 is delivered through nasal prongs or "open" face masks (see Chapter 22). (This limitation has also been described for the A-a PO_2 gradient.)

Blood Gas Variability

The arterial PO_2 and PCO_2 can vary spontaneously without a change in the clinical condition of the patient. This is demonstrated in Table 20.2, which shows the spontaneous variation in arterial PO_2 and PCO_2 over a one-hour period in a group of clinically stable trauma victims (10). Note that the arterial PO_2 varied by as much as 36 mm Hg, while the arterial PCO_2 varied by as much as 12 mm Hg. This variability has also been observed in patients in a medical ICU (11). Because of this degree of spontaneous variation, routine monitoring of arterial blood gases can be misleading.

Table 20.2 Spontaneous Blood-Gas Variability

Variation	PaO_2	$PaCO_2$
Mean	13 mm Hg	2.5 mm Hg
95th Percentile	±18 mm Hg	±4 mm Hg
Range	2–37 mm Hg	0–12 mm Hg

Represents variations over a 1-hour period in 26 ventilator-dependent trauma victims who were clinically stable.

From Reference 10.

HYPOXEMIA

Hypoxemia can be defined as an arterial PO_2 below what is expected for a patient's age, as defined in Table 20.1. However, hypoxemia usually doesn't raise red flags until the arterial PO_2 falls below 60 mm Hg (or the arterial O_2 saturation falls below 90%). The causes of hypoxemia can be separated into 3 categories based on the physiological process involved (12,13). Each group of disorders can be distinguished by the A-a PO_2 gradient and/or the mixed venous PO_2, as shown in Table 20.3.

Table 20.3 Sources of Hypoxemia

Source	A-a PO_2	PvO_2
Hypoventilation	Normal	Normal
V/Q mismatch	Increased	Normal
DO_2/VO_2 imbalance	Increased	Decreased

Hypoventilation

Alveolar hypoventilation causes both hypoxemia and hypercapnia, similar to breath-holding. There is no V/Q imbalance in the lungs, so the A-a PO_2 gradient is not elevated. The common causes of alveolar hypoventilation are listed in Table 20.4. Most cases of hypoventilation in the ICU are the result of drug-induced respiratory depression or neuromuscular weakness. Obesity-related hypoventilation (Pickwickian syndrome) is also a consideration, as this condition is present in up to one-third of morbidly obese patients (body mass index >35 kg/m^2) (14).

Table 20.4 Alveolar Hypoventilation in the ICU

Brainstem Respiratory Depression
1. Drugs (e.g., opiates)
2. Obesity-hypoventilation syndrome
Peripheral Neuropathy
1. Critical illness polyneuropathy
2. Guillain-Barré syndrome
Muscle Weakness
1. Critical illness myopathy
2. Hypophosphatemia
3. Myasthenia gravis

Respiratory Muscle Weakness

Most cases of respiratory muscle weakness in the ICU are the result of an idiopathic polyneuropathy and myopathy that is specific to ICU patients, particularly those with sepsis, prolonged mechanical ventilation, and prolonged neuromuscular paralysis (15). The standard method of evaluating respiratory muscle strength is to measure the maximum inspiratory pressure (PI_{max}), which is the maximum pressure recorded during a maximum inspiratory effort against a closed valve. The normal PI_{max} varies with age and gender, but most healthy adults can generate a negative PI_{max} of at least 80 cm H_2O (16). A PI_{max} that does not exceed -25 cm H_2O is considered evidence of respiratory muscle failure (17). (See Chapter 45 for more information on neuromuscular weakness syndromes in the ICU.)

V/Q Mismatch

Most cases of hypoxemia are the result of a V/Q mismatch in the lungs. Virtually any lung disease can be included in this category, but the common ones encountered in the ICU are pneumonia, inflammatory lung injury (acute respiratory distress syndrome), obstructive lung disease, hydrostatic pulmonary edema, and pulmonary embolism. The A-a PO_2 gradient is almost always elevated in these conditions, but the elevation can be minimal in patients with severe airways obstruction (which behaves like hypoventilation).

DO_2/VO_2 Imbalance

As explained in Chapter 10, a decrease in systemic O_2 delivery (DO_2) is usually accompanied by an increase in O_2 extraction from capillary blood, and this serves to maintain a constant rate of O_2 uptake (VO_2) into the tissues. The increased O_2 extraction from capillary blood results in a decrease in the PO_2 of venous blood, and this can have an deleterious effect on arterial oxygenation, as explained below.

Mixed Venous PO_2

The O_2 in arterial blood represents the sum of the O_2 in mixed venous (pulmonary artery) blood and the O_2 added from alveolar gas. When gas exchange is normal, the PO_2 in alveolar gas is the major determinant of the arterial PO_2. However, when gas exchange is impaired, the contribution of the alveolar PO_2 declines and the contribution of the mixed venous PO_2 rises (18). The greater the impairment in gas exchange, the greater the contribution of the mixed venous PO_2 to the arterial PO_2. (If there is no gas exchange in the lungs, the mixed venous PO_2 would be the sole determinant of the arterial PO_2.)

The diagram in Figure 20.5 demonstrates the influence of mixed venous PO_2 on the arterial PO_2 when gas exchange is impaired. The curves in the graph represent the transition from mixed venous PO_2 to arterial PO_2 as blood flows through the lungs. The slope of each curve reflects the efficiency of gas exchange in the lungs. Note that the curve representing the V/Q abnormality results in a lower arterial PO_2 because the slope is decreased (indicating impaired oxygen exchange in the lungs). If this curve begins at a lower mixed venous PO_2, as indicated, the curve shifts downward, resulting in a further decrease in arterial PO_2. This illustrates how a decrease in mixed venous PO_2 can aggravate the hypoxemia caused by a V/Q abnormality. It also indicates that, in the presence of a V/Q abnormality, the mixed venous PO_2 is an important consideration in the evaluation of hypoxemia.

The relationship between O_2 delivery (DO_2), O_2 uptake (VO_2), and the mixed venous PO_2 (PvO_2) can be stated as follows:

$$PvO_2 = k \times (DO_2/VO_2) \qquad \textbf{(20.8)}$$

(k is a proportionality constant.) Thus, any condition that reduces DO_2 (e.g., low cardiac output, anemia) or increases VO_2 (e.g., hypermetabolism) can decrease the PvO_2 and aggravate the hypoxemia caused by abnormal gas exchange in the lungs.

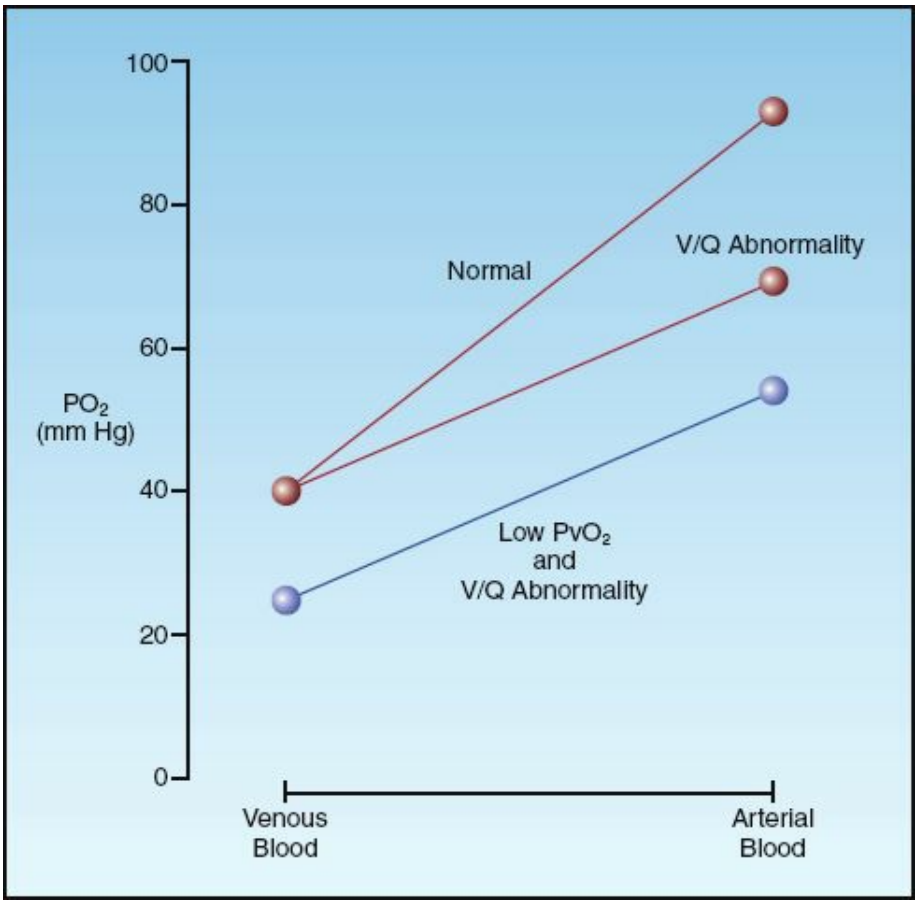

FIGURE 20.5 The influence of a V/Q abnormality on the transition from venous to arterial PO_2, and the added effect of a low mixed venous PO_2 (PvO_2).

Diagnostic Evaluation

The evaluation of hypoxemia can proceed according to the flow diagram in Figure 20.6. This approach uses three measures: A-a PO_2 gradient, mixed venous PO_2, and maximum inspiratory pressure. The PO_2 in superior vena cava blood (central venous PO_2) can be used as the mixed venous PO_2 when there is no indwelling pulmonary artery catheter.

The first step in the approach involves a determination of the A-a PO_2 gradient. After correcting for age and FIO_2, the A-a PO_2 gradient can be interpreted as follows:

1. Normal A-a PO_2 gradient indicates hypoventilation rather than a cardiopulmonary disorder. In this situation, the most likely problems are drug-induced respiratory depression and neuromuscular weakness. The latter condition can be uncovered by measuring the maximum inspiratory pressure (PI_{max}), which is described earlier.
2. Increased A-a PO_2 gradient indicates a V/Q abnormality (cardiopulmonary disorder) and a possible superimposed DO_2/VO_2 imbalance (e.g., a decrease in cardiac output). The mixed venous (or central venous) PO_2 will help to identify a DO_2/VO_2 imbalance.

   a. If the venous PO_2 is 40 mm Hg or higher, the problem is solely a V/Q mismatch in the lungs.

   b. If the venous PO_2 is below 40 mm Hg, there is a DO_2/VO_2 imbalance adding to the hypoxemia created by a V/Q mismatch in the lungs. The source of this imbalance is either a decreased DO_2 (from anemia or a low cardiac output) or an increased VO_2 (from hypermetabolism).

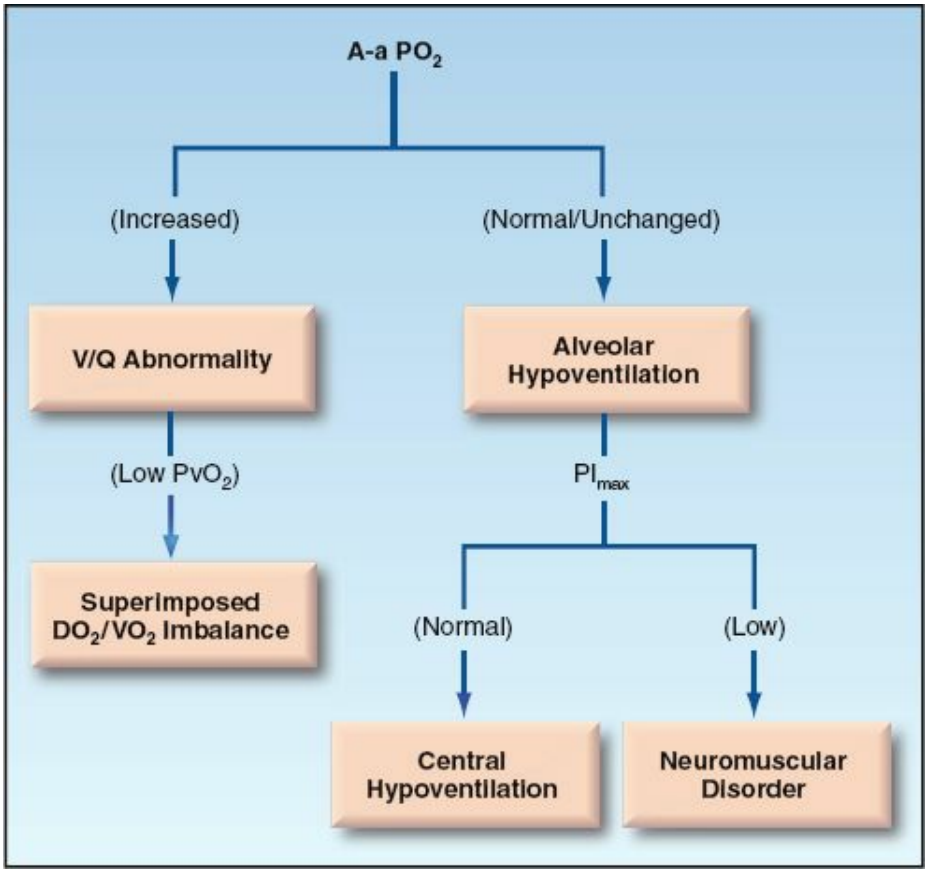

FIGURE 20.6 Flow diagram for the evaluation of hypoxemia.

Spurious Hypoxemia

Spurious hypoxemia is a rarely reported phenomenon that is characterized by hypoxemia in an arterial blood sample without corresponding hypoxemia in circulating blood (as measured by pulse oximetry) (19). This phenomenon seems to occur only in patients with hematologic malignancies who have marked leukocytosis (WBC >100,000) or thrombocytosis (platelet count >1,000,000). The reduced PO_2 in the blood sample has been attributed to O_2 consumption by activated leukocytes in the sample, a phenomenon that has been called leukocyte larceny (20). This does not explain why marked thrombocytosis can also produce spurious hypoxemia because platelets are not oxygen-guzzlers like activated leukocytes. Regardless of the mechanism, there is no accepted method of preventing spurious hypoxemia (rapid cooling of blood samples has had inconsistent results), so you should be aware of the phenomenon and the value of pulse oximetry for validating in vitro PO_2 measurements (pulse oximetry is described in the next chapter).

HYPERCAPNIA

Hypercapnia is defined as an arterial PCO_2 ($PaCO_2$) above 46 mm Hg that does not represent compensation for a metabolic alkalosis (21). The causes of hypercapnia can be identified by considering the determinants of $PaCO_2$ in the following relationship, where VCO_2 is the rate of CO_2 production in the body, V_A is the rate of alveolar ventilation, and k is a proportionality constant (1).

$$PaCO_2 = k \times (VCO_2/V_A) \quad \textbf{(20.9)}$$

Alveolar ventilation is the portion of the total ventilation (V_E) that is not dead space ventilation (V_D/V_T); that is, $V_A = V_E\ (1 - V_D/V_T)$. Therefore, equation 20.9 can be restated as follows:

$$PaCO_2 = k \times [VCO_2/V_E\ (1 - V_D/V_T)] \quad \textbf{(20.10)}$$

This equation identifies three major sources of hypercapnia: (a) increased CO_2 production (VCO_2), (b) hypoventilation ($1/V_E$), and (c) increased dead space ventilation (V_D/V_T).

Hypoventilation

Hypoventilation was discussed briefly in the last section on hypoxemia, and Table 20.4 shows the common causes of hypoventilation. Because hypoxemia is so common in ICU patients, hypercapnia may be the first sign of hypoventilation from neuromuscular weakness or drug-induced respiratory depression. This is also the case in obesity-hypoventilation syndrome, where hypercapnia while awake is often the first evidence of hypoventilation. On the other hand, hypercapnia is a relatively late sign in neuromuscular disorders, and does not appear until the maximum inspiratory pressure or PI_{max} (described earlier) is below 50% of normal (17).

V/Q Abnormality

As mentioned earlier, hypercapnia is not a feature of increased intrapulmonary shunt until late in the process (which is why hypercapnia is not a feature of pulmonary edema or other infiltrative lung processes until they are far advanced). Hypercapnia is more a feature of increased dead space ventilation (such as occurs in advanced emphysema, where there is destruction of the alveolar-capillary interface), and the $PaCO_2$ usually begins to rise when dead space ventilation accounts for more than 50% of total ventilation ($V_D/V_T > 0.5$).

Increased CO_2 Production

An increase in CO_2 production is usually related to oxidative metabolism, but non-metabolic CO_2 production is possible when extracellular acids generate hydrogen ions that combine with bicarbonate ions and generate CO_2. Whatever the source, increased CO_2 production is normally accompanied by an increase in minute ventilation, which eliminates the excess CO_2 and maintains a constant arterial PCO_2. Therefore, excess CO_2 production does not normally cause hypercapnia. However, when CO_2 excretion is impaired, an increase in CO_2 production can lead to an increase in $PaCO_2$. Thus, increased CO_2 production is an important factor in promoting hypercapnia only when the ability to eliminate CO_2 is impaired.

Overfeeding

Overfeeding, or the provision of calories in excess of daily needs, is a recognized cause of hypercapnia in patients with severe lung disease and acute respiratory failure (22). Nutrition-associated hypercapnia occurs predominantly in ventilator-dependent patients, and can delay weaning from mechanical ventilation. Overfeeding with carbohydrates is particularly problematic because the oxidative metabolism of carbohydrates generates more carbon dioxide than the other nutrient substrates (lipids and proteins). This is described in more detail in Chapter 47.

Diagnostic Evaluation

The bedside evaluation of hypercapnia is shown in Figure 20.7. The evaluation of hypercapnia, like hypoxemia, begins with the A-a PO_2 gradient (23). A normal or unchanged A-a PO_2 gradient indicates that the problem is alveolar hypoventilation (the same as described for the evaluation of hypoxemia). An increased A-a PO_2 gradient indicates a V/Q abnormality (an increase in dead space ventilation) that may or may not be accompanied by an increase in CO_2 production.

Measuring CO_2 Production

The rate of CO_2 production (VCO_2) can be measured at the bedside with specialized metabolic carts that are normally used to perform nutritional assessments. These carts are equipped with infrared devices that can measure the CO_2 in expired gas (much like the end-tidal CO_2 monitors described in Chapter 21), and can determine the volume of CO_2 excreted per minute. In steady-state conditions, the rate of CO_2 excretion is equivalent to the VCO_2. The normal VCO_2 is 90 to 130 L/minute/m^2, which is roughly 80% of the VO_2. As mentioned earlier, an increased VCO_2 is evidence for one of the following conditions: generalized hypermetabolism, overfeeding (excess calories), or metabolic acidoses.

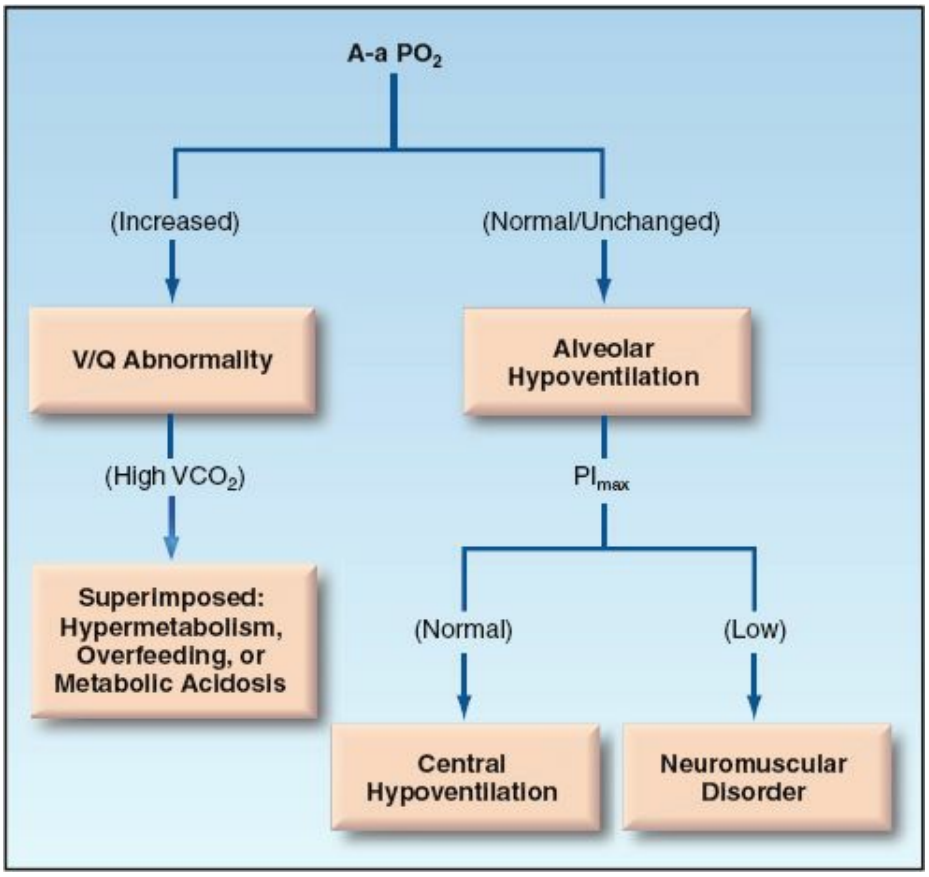

FIGURE 20.7 Flow diagram for the evaluation of hypercapnia.

A FINAL WORD

It is important to remember that the arterial PO_2 is not a useful measure for determining the amount of oxygen in the blood (this requires the hemoglobin concentration in blood and the percent saturation of hemoglobin with oxygen, as shown in Equation 10.6 in Chapter 10). Instead, the PaO_2 (along with the $PaCO_2$) is used to evaluate gas exchange in the lungs, and can be useful in identifying the source of the problem with gas exchange.

An approach to O_2 and CO_2 balance that is superior in many ways to the measurement of arterial blood gases is described in the next chapter.

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