Arterial Blood Gas Interpretation

Lawrence Martin, MD, FACP, FCCP
Associate Professor of Medicine
Case Western Reserve University School of Medicine, Cleveland
larry.martin@adelphia.net

Information in this slide presentation is adapted from All You Really Need to Know to Interpret Arterial Blood Gases (2nd ed.), by Lawrence Martin, MD, Lippincott, Williams, Wilkins
# Normal Arterial Blood Gas Values*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.35 - 7.45</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>35 - 45 mm Hg</td>
</tr>
<tr>
<td>PaO₂</td>
<td>70 - 100 mm Hg **</td>
</tr>
<tr>
<td>SaO₂</td>
<td>93 - 98%</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>22 - 26 mEq/L</td>
</tr>
<tr>
<td>%MetHb</td>
<td>&lt; 2.0%</td>
</tr>
<tr>
<td>%COHb</td>
<td>&lt; 3.0%</td>
</tr>
<tr>
<td>Base excess</td>
<td>-2.0 to 2.0 mEq/L</td>
</tr>
<tr>
<td>CaO₂</td>
<td>16 - 22 ml O₂/dl</td>
</tr>
</tbody>
</table>

* At sea level, breathing ambient air
** Age-dependent
# The Key to Blood Gas Interpretation: Four Equations, Three Physiologic Processes

<table>
<thead>
<tr>
<th>Equation</th>
<th>Physiologic Process</th>
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</thead>
<tbody>
<tr>
<td>1) PaCO₂ equation</td>
<td>Alveolar ventilation</td>
</tr>
<tr>
<td>2) Alveolar gas equation</td>
<td>Oxygenation</td>
</tr>
<tr>
<td>3) Oxygen content equation</td>
<td>Oxygenation</td>
</tr>
<tr>
<td>4) Henderson-Hasselbalch equation</td>
<td>Acid-base balance</td>
</tr>
</tbody>
</table>

These four equations, crucial to understanding and interpreting arterial blood gas data, will provide the structure for this slide presentation.
**PaCO₂ Equation:**  \( \text{PaCO}_2 \) reflects ratio of metabolic CO₂ production to alveolar ventilation

\[
\text{PaCO}_2 = \frac{\text{VCO}_2 \times 0.863}{\text{VA}} \]

- \( \text{VCO}_2 \): CO₂ production
- \( \text{VA} \): Alveolar ventilation
- \( \text{VE} \): Minute (total) ventilation (= resp. rate \times tidal volume)
- \( \text{VD} \): Dead space ventilation (= resp. rate \times dead space volume)
- 0.863 converts VCO₂ and VA units to mm Hg

<table>
<thead>
<tr>
<th>PaCO₂</th>
<th>Condition in blood</th>
<th>State of alveolar ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 45 mm Hg</td>
<td>Hypercapnia</td>
<td>Hypoventilation</td>
</tr>
<tr>
<td>35 - 45 mm Hg</td>
<td>Eucapnia</td>
<td>Normal ventilation</td>
</tr>
<tr>
<td>&lt; 35 mm Hg</td>
<td>Hypocapnia</td>
<td>Hyperventilation</td>
</tr>
</tbody>
</table>
Hypercapnia

\[
\text{PaCO}_2 = \frac{\text{VCO}_2 \times 0.863}{\text{VA}} = \frac{\text{VE} - \text{VD}}{	ext{VA}}
\]

Hypercapnia (elevated PaCO\textsubscript{2}) is a serious respiratory problem. The PaCO\textsubscript{2} equation shows that the only physiologic reason for elevated PaCO\textsubscript{2} is inadequate alveolar ventilation (VA) for the amount of the body’s CO\textsubscript{2} production (VCO\textsubscript{2}). Since alveolar ventilation (VA) equals total or minute ventilation (VE) minus dead space ventilation (VD), hypercapnia can arise from insufficient VE, increased VD, or a combination of both.
Hypercapnia (cont)

\[
\text{PaCO}_2 = \frac{\text{VCO}_2 \times 0.863}{\text{VA}}
\]

\[
\text{VA} = \text{VE} - \text{VD}
\]

Examples of inadequate VE leading to decreased VA and increased \( \text{PaCO}_2 \): sedative drug overdose; respiratory muscle paralysis; central hypoventilation

Examples of increased VD leading to decreased VA and increased \( \text{PaCO}_2 \): chronic obstructive pulmonary disease; severe restrictive lung disease (with shallow, rapid breathing)
Clinical Assessment of Hypercapnia is Unreliable

The PaCO₂ equation shows why PaCO₂ cannot reliably be assessed clinically. Since you never know the patient's VCO₂ or VA, you cannot determine the VCO₂/VA, which is what PaCO₂ provides. (Even if VE is measured [respiratory rate x tidal volume], you cannot determine the amount of air going to dead space, i.e., the dead space ventilation.)

There is no predictable correlation between PaCO₂ and the clinical picture. In a patient with possible respiratory disease, respiratory rate, depth, and effort cannot be reliably used to predict even a directional change in PaCO₂. A patient in respiratory distress can have a high, normal, or low PaCO₂. A patient without respiratory distress can have a high, normal, or low PaCO₂.
Besides indicating a serious derangement in the respiratory system, elevated \( \text{PaCO}_2 \) poses a threat for three reasons:

1) An elevated \( \text{PaCO}_2 \) will lower the \( \text{PAO}_2 \) (see Alveolar gas equation), and as a result will lower the \( \text{PaO}_2 \).

2) An elevated \( \text{PaCO}_2 \) will lower the pH (see Henderson-Hasselbalch equation).

3) The higher the baseline \( \text{PaCO}_2 \), the greater it will rise for a given fall in alveolar ventilation, e.g., a 1 L/min decrease in VA will raise \( \text{PaCO}_2 \) a greater amount when the baseline \( \text{PaCO}_2 \) is 50 mm Hg than when it is 40 mm Hg. (See next slide)
The relationship is shown for metabolic carbon dioxide production rates of 200 ml/min and 300 ml/min (curved lines). A fixed decrease in alveolar ventilation (x-axis) in the hypercapnic patient will result in a greater rise in PaCO₂ (y-axis) than the same VA change when PaCO₂ is low or normal. (This situation is analogous to the progressively steeper rise in BUN as glomerular filtration rate declines.)

This graph also shows that if alveolar ventilation is fixed, an increase in carbon dioxide production will result in an increase in PaCO₂.
PaCO₂ and Alveolar Ventilation: Test Your Understanding

1. What is the PaCO₂ of a patient with respiratory rate 24/min, tidal volume 300 ml, dead space volume 150 ml, CO₂ production 300 ml/min? The patient shows some evidence of respiratory distress.

2. What is the PaCO₂ of a patient with respiratory rate 10/min, tidal volume 600 ml, dead space volume 150 ml, CO₂ production 200 ml/min? The patient shows some evidence of respiratory distress.
1. First, you must calculate the alveolar ventilation. Since minute ventilation is 24 x 300 or 7.2 L/min, and dead space ventilation is 24 x 150 or 3.6 L/min, alveolar ventilation is 3.6 L/min. Then

\[
\frac{300 \text{ ml/min} \times .863}{3.6 \text{ L/min}} = \text{PaCO}_2
\]

\[
\text{PaCO}_2 = 71.9 \text{ mm Hg }
\]

2. \( VA = VE - VD \)

\[
= 10(600) - 10(150) = 6 - 1.5 = 4.5 \text{ L/min}
\]

\[
\frac{200 \text{ ml/min} \times .863}{4.5 \text{ L/min}} = \text{PaCO}_2
\]

\[
\text{PaCO}_2 = 38.4 \text{ mm Hg }
\]
3. A man with severe chronic obstructive pulmonary disease exercises on a treadmill at 3 miles/hr. His rate of CO₂ production increases by 50% but he is unable to augment alveolar ventilation. If his resting PaCO₂ is 40 mm Hg and resting VCO₂ is 200 ml/min, what will be his exercise PaCO₂?
3. Exercise increases metabolic CO\(_2\) production. People with a normal respiratory system are always able to augment alveolar ventilation to meet or exceed the amount of VA necessary to excrete any increase in CO\(_2\) production. As in this example, patients with severe COPD or other forms of chronic lung disease may *not* be able to increase their alveolar ventilation, resulting in an increase in PaCO\(_2\). This patient’s resting alveolar ventilation is

\[
\text{VA} = \frac{200 \text{ ml/min} \times 0.863}{40 \text{ mm Hg}} = 4.32 \text{ L/min}
\]

**Since CO\(_2\) production increased by 50% and alveolar ventilation not at all,** his exercise PaCO\(_2\) is

\[
\text{PaCO}_2 = \frac{300 \text{ ml/min} \times 0.863}{4.32 \text{ L/min}} = 59.9 \text{ mm Hg}
\]
**Alveolar Gas Equation**

\[
PAO_2 = PIO_2 - 1.2 \times (PaCO_2)
\]

Where \( PAO_2 \) is the average alveolar \( PO_2 \), and \( PIO_2 \) is the partial pressure of inspired oxygen in the trachea.

\[
PIO_2 = FIO_2 \times (P_B - 47 \text{ mm Hg})
\]

\( FIO_2 \) is the fraction of inspired oxygen and \( P_B \) is the barometric pressure. 47 mm Hg is the water vapor pressure at normal body temperature.

* Note: This is the “abbreviated version” of the AG equation, suitable for most clinical purposes. In the longer version, the multiplication factor “1.2” declines with increasing \( FIO_2 \), reaching zero when 100% oxygen is inhaled. In these exercises “1.2” is dropped when \( FIO_2 \) is above 60%. 
Alveolar Gas Equation

\[ PAO_2 = PIO_2 - 1.2 \times (PaCO_2) \]

where \( PIO_2 = FIO_2 \times (P_B - 47 \text{ mm Hg}) \)

Except in a temporary unsteady state, alveolar \( PO_2 \) (\( PAO_2 \)) is always higher than arterial \( PO_2 \) (\( PaO_2 \)). As a result, whenever \( PAO_2 \) decreases, \( PaO_2 \) also decreases. Thus, from the AG equation:

- If \( FIO_2 \) and \( P_B \) are constant, then as \( PaCO_2 \) increases both \( PAO_2 \) and \( PaO_2 \) will decrease (hypercapnia causes hypoxemia).
- If \( FIO_2 \) decreases and \( P_B \) and \( PaCO_2 \) are constant, both \( PAO_2 \) and \( PaO_2 \) will decrease (suffocation causes hypoxemia).
- If \( P_B \) decreases (e.g., with altitude), and \( PaCO_2 \) and \( FIO_2 \) are constant, both \( PAO_2 \) and \( PaO_2 \) will decrease (mountain climbing leads to hypoxemia).
Alveolar Gas Equation: Test Your Understanding

1. What is the PAO$_2$ at sea level in the following circumstances? (Barometric pressure = 760 mm Hg)
   a) FIO$_2$ = 1.00, PaCO$_2$ = 30 mm Hg
   b) FIO$_2$ = .21, PaCO$_2$ = 50 mm Hg
   c) FIO$_2$ = .40, PaCO$_2$ = 30 mm Hg

2. What is the PAO$_2$ on the summit of Mt. Everest in the following circumstances? (Barometric pressure = 253 mm Hg)
   a) FIO$_2$ = .21, PaCO$_2$ = 40 mm Hg
   b) FIO$_2$ = 1.00, PaCO$_2$ = 40 mm Hg
   c) FIO$_2$ = .21, PaCO$_2$ = 10 mm Hg
Alveolar Gas Equation: Test Your Understanding - Answers

1. To calculate $PAO_2$ the $PaCO_2$ must be subtracted from the $PIO_2$. Again, the barometric pressure is 760 mm Hg since the values are obtained at sea level. In part a, the $PaCO_2$ of 30 mm Hg is not multiplied by 1.2 since the $FIO_2$ is 1.00. In parts b and c, $PaCO_2$ is multiplied by the factor 1.2.

   a) $PAO_2 = 1.00 (713) - 30 = 683$ mm Hg
   b) $PAO_2 = .21 (713) - 1.2 (50) = 90$ mm Hg
   c) $PAO_2 = .40 (713) - 1.2 (30) = 249$ mm Hg

2. The $PAO_2$ on the summit of Mt. Everest is calculated just as at sea level, using the barometric pressure of 253 mm Hg.

   a) $PAO_2 = .21 (253 - 47) - 1.2 (40) = -5$ mm Hg
   b) $PAO_2 = 1.00 (253 - 47) - 40 = 166$ mm Hg
   c) $PAO_2 = .21 (253 - 47) - 1.2 (10) = 31$ mm Hg
P(A-a)O₂

P(A-a)O₂ is the alveolar-arterial difference in partial pressure of oxygen. It is commonly called the “A-a gradient,” though it does not actually result from an O₂ pressure gradient in the lungs. Instead, it results from gravity-related blood flow changes within the lungs (normal ventilation-perfusion imbalance).

PAO₂ is always calculated based on FIO₂, PaCO₂, and barometric pressure.

PaO₂ is always measured on an arterial blood sample in a “blood gas machine.”

Normal P(A-a)O₂ ranges from @ 5 to 25 mm Hg breathing room air (it increases with age). A higher than normal P(A-a)O₂ means the lungs are not transferring oxygen properly from alveoli into the pulmonary capillaries. Except for right to left cardiac shunts, an elevated P(A-a)O₂ signifies some sort of problem within the lungs.
## Physiologic Causes of Low \( \text{PaO}_2 \)

<table>
<thead>
<tr>
<th>NON-RESPIRATORY</th>
<th>( P(A-a)\text{O}_2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac right-to-left shunt</td>
<td>Increased</td>
</tr>
<tr>
<td>Decreased ( \text{PIO}_2 )</td>
<td>Normal</td>
</tr>
<tr>
<td>Low mixed venous oxygen content*</td>
<td>Increased</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>RESPIRATORY</th>
<th>( P(A-a)\text{O}_2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary right-to-left shunt</td>
<td>Increased</td>
</tr>
<tr>
<td>Ventilation-perfusion imbalance</td>
<td>Increased</td>
</tr>
<tr>
<td>Diffusion barrier</td>
<td>Increased</td>
</tr>
<tr>
<td>Hypoventilation (increased ( \text{PaCO}_2 ))</td>
<td>Normal</td>
</tr>
</tbody>
</table>

* Unlikely to be clinically significant unless there is right-to-left shunting or ventilation-perfusion imbalance
Ventilation-perfusion Imbalance

- A normal amount of ventilation-perfusion (V-Q) imbalance accounts for the normal P(A-a)O$_2$.

- By far the most common cause of low PaO$_2$ is an abnormal degree of ventilation-perfusion imbalance within the hundreds of millions of alveolar-capillary units. Virtually all lung disease lowers PaO$_2$ via V-Q imbalance, e.g., asthma, pneumonia, atelectasis, pulmonary edema, COPD.

- Diffusion barrier is seldom a major cause of low PaO$_2$ (it can lead to a low PaO$_2$ during exercise).
3. For each of the following scenarios, calculate the P(A-a)O$_2$ using the abbreviated alveolar gas equation; assume P$_B$ = 760 mm Hg. Which of these patients is most likely to have lung disease? Do any of the values represent a measurement or recording error?

a) A 35-year-old man with PaCO$_2$ 50 mm Hg, PaO$_2$ 150 mm Hg, FIO$_2$ 0.40.
b) A 44-year-old woman with PaCO$_2$ 75 mm Hg, PaO$_2$ 95 mm Hg, FIO$_2$ 0.28.
c) A young, anxious man with PaO$_2$ 120 mm Hg, PaCO$_2$ 15 mm Hg, FIO$_2$ 0.21.
d) A woman in the intensive care unit with PaO$_2$ 350 mm Hg, PaCO$_2$ 40 mm Hg, FIO$_2$ 0.80.
e) A man with PaO$_2$ 80 mm Hg, PaCO$_2$ 72 mm Hg, FIO$_2$ 0.21.
P(A-a)O₂: Test Your Understanding - Answers to #3

a) \( \text{PAO}_2 = 0.40 \times (760 - 47) - 1.2 \times (50) = 225 \text{ mm Hg}; \ P(A-a)O_2 = 225 - 150 = 75 \text{ mm Hg} \)

The P(A-a)O₂ is elevated but actually within the expected range for supplemental oxygen at 40%, so the patient may or may not have a defect in gas exchange.

b) \( \text{PAO}_2 = 0.28 \times (713) - 1.2 \times (75) = 200 - 90 = 110 \text{ mm Hg}; \ P(A-a)O_2 = 110 - 95 = 15 \text{ mm Hg} \)

Despite severe hypoventilation, there is no evidence here for lung disease. Hypercapnia is most likely a result of disease elsewhere in the respiratory system, either the central nervous system or chest bellows.

c) \( \text{PAO}_2 = 0.21 \times (713) - 1.2 \times (15) = 150 - 18 = 132 \text{ mm Hg}; \ P(A-a)O_2 = 132 - 120 = 12 \text{ mm Hg} \)

Hyperventilation can easily raise PaO₂ above 100 mm Hg when the lungs are normal, as in this case.
d) \[ \text{PAO}_2 = .80 \times (713) - 40 = 530 \text{ mm Hg} \] (Note that the factor 1.2 is dropped since FIO\(_2\) is above 60%)

\[ \text{P(A-a)O}_2 = 530 - 350 = 180 \text{ mm Hg} \]

\( \text{P(A-a)O}_2 \) is increased. Despite a very high \( \text{PaO}_2 \), the lungs are not transferring oxygen normally.

e) \[ \text{PAO}_2 = .21 \times (713) - 1.2 \times (72) = 150 - 86 = 64 \text{ mm Hg}; \text{P(A-a)O}_2 = 64 - 80 = -16 \text{ mm Hg} \]

A negative \( \text{P(A-a)O}_2 \) is incompatible with life (unless it is a transient unsteady state, such as sudden fall in FIO\(_2\) -- not the case here). In this example, negative \( \text{P(A-a)O}_2 \) can be explained by any of the following: incorrect FIO\(_2\), incorrect blood gas measurement, or a reporting or transcription error.
SaO$_2$ and Oxygen Content

Tissues need a requisite amount of oxygen molecules for metabolism. Neither the PaO$_2$ nor the SaO$_2$ tells how much oxygen is in the blood. *How much* is provided by the oxygen content, CaO$_2$ (units = ml O$_2$/dl). CaO$_2$ is calculated as:

$$\text{CaO}_2 = \text{quantity O}_2 \text{ bound to hemoglobin} + \text{quantity O}_2 \text{ dissolved in plasma}$$

$$\text{CaO}_2 = (Hb \times 1.34 \times \text{SaO}_2) + (.003 \times \text{PaO}_2)$$

- Hb = hemoglobin in gm%; 1.34 = ml O$_2$ that can be bound to each gm of Hb; SaO$_2$ is percent saturation of hemoglobin with oxygen; .003 is solubility coefficient of oxygen in plasma: .003 ml dissolved O$_2$/mm Hg PO$_2$. 
Oxygen Dissociation Curve: $\text{SaO}_2$ vs. $\text{PaO}_2$

Also shown are $\text{CaO}_2$ vs. $\text{PaO}_2$ for two different hemoglobin contents: 15 gm% and 10 gm%. $\text{CaO}_2$ units are ml O2/dl.

P50 is the $\text{PaO}_2$ at which $\text{SaO}_2$ is 50%.

Point “X” is discussed on later slide.
You always need to know this when confronted with blood gas data.

SaO$_2$ is measured in a “co-oximeter.” The traditional “blood gas machine” measures only pH, PaCO$_2$, and PaO$_2$, whereas the co-oximeter measures SaO$_2$, carboxyhemoglobin, methemoglobin, and hemoglobin content. Newer “blood gas” consoles incorporate a co-oximeter, and so offer the latter group of measurements as well as pH, PaCO$_2$, and PaO$_2$.

You should always make sure the SaO$_2$ is measured, not calculated. If SaO$_2$ is calculated from PaO$_2$ and the O$_2$-dissociation curve, it provides no new information and could be inaccurate - especially in states of CO intoxication or excess methemoglobin. CO and metHb do not affect PaO$_2$, but do lower the SaO$_2$. 
Carbon Monoxide – An Important Cause of Hypoxemia

- Normal percentage of COHb in the blood is 1-2%, from metabolism and small amount of ambient CO (higher in traffic-congested areas).
- CO is colorless, odorless gas, a product of combustion; all smokers have excess CO in their blood, typically 5-10%.
- CO binds 200x more avidly to hemoglobin than O₂, effectively displacing O₂ from the heme binding sites. CO is a major cause of poisoning deaths world-wide.
- CO has a “double-whammy” effect on oxygenation: 1) decreases SaO₂ by the percentage of COHb present, and 2) shifts the O₂-dissociation curve to the left, retarding unloading of oxygen to the tissues.
- CO does not affect PaO₂, only SaO₂. To detect CO poisoning, SaO₂ and/or COHb must be measured (requires co-oximeter). In the presence of excess CO, SaO₂ (when measured) will be lower than expected from the PaO₂.
CO Does Not Affect PaO₂ – Be Aware!

Review the O₂ dissociation curve shown on a previous slide. “X” represents the 2nd set of blood gases for a patient who presented to the ER with headache and dyspnea.

His first blood gases showed PaO₂ 80 mm Hg, PaCO₂ 38 mm Hg, pH 7.43. SaO₂ on this first set was calculated from the O₂-dissociation curve as 97%, and oxygenation was judged normal.

He was sent out from the ER and returned a few hours later with mental confusion; this time both SaO₂ and COHb were measured (SaO₂ shown by “X”): PaO₂ 79 mm Hg, PaCO₂ 31 mm Hg, pH 7.36, SaO₂ 53%, carboxyhemoglobin 46%.

CO poisoning was missed on the first set of blood gases because SaO₂ was not measured!
Causes of Hypoxia

A General Classification

1. Hypoxemia (= low PaO₂ and/or low CaO₂)
   a. reduced PaO₂ – usually from lung disease (most common physiologic mechanism: V-Q imbalance)
   b. reduced SaO₂ – most commonly from reduced PaO₂; other causes include carbon monoxide poisoning, methemoglobinemia, or rightward shift of the O₂-dissociation curve
   c. reduced hemoglobin content – anemia

2. Reduced oxygen delivery to the tissues
   a. reduced cardiac output – shock, congestive heart failure
   b. left-to-right systemic shunt (as may be seen in septic shock)

3. Decreased tissue oxygen uptake
   a. mitochondrial poisoning (e.g., cyanide poisoning)
   b. left-shifted hemoglobin dissociation curve (e.g., from acute alkalosis, excess CO, or abnormal hemoglobin structure)
How much oxygen is in the blood, and is it adequate for the patient?

PaO₂ vs. SaO₂ vs. CaO₂

The answer must be based on some oxygen value, but which one? Blood gases give us three different oxygen values: PaO₂, SaO₂, and CaO₂ (oxygen content).

Of these three values, PaO₂, or oxygen pressure, is the least helpful to answer the question about oxygen adequacy in the blood. The other two values - SaO₂ and CaO₂ - are more useful for this purpose.
How much oxygen is in the blood?
PaO₂ vs. SaO₂ vs. CaO₂

OXYGEN PRESSURE: PaO₂
- Since PaO₂ reflects only free oxygen molecules dissolved in plasma and not those bound to hemoglobin, PaO₂ cannot tell us “how much” oxygen is in the blood; for that you need to know how much oxygen is also bound to hemoglobin, information given by the SaO₂ and hemoglobin content.

OXYGEN SATURATION: SaO₂
- The percentage of all the available heme binding sites saturated with oxygen is the hemoglobin oxygen saturation (in arterial blood, the SaO₂). Note that SaO₂ alone doesn’t reveal how much oxygen is in the blood; for that we also need to know the hemoglobin content.

OXYGEN CONTENT: CaO₂
- Tissues need a requisite amount of O₂ molecules for metabolism. Neither the PaO₂ nor the SaO₂ provide information on the number of oxygen molecules, i.e., how much oxygen is in the blood. (Neither PaO₂ nor SaO₂ have units that denote any quantity.) Only CaO₂ (units ml O₂/dl) tells us how much oxygen is in the blood; this is because CaO₂ is the only value that incorporates the hemoglobin content. Oxygen content can be measured directly or calculated by the oxygen content equation:

\[
CaO₂ = (Hb \times 1.34 \times SaO₂) + (0.003 \times PaO₂)
\]
SaO₂ and CaO₂: Test Your Understanding

Below are blood gas results from four pairs of patients. For each letter pair, state which patient, (1) or (2), is more hypoxemic. Units for hemoglobin content (Hb) are gm% and for PaO₂ mm Hg.

a) (1) Hb 15, PaO₂ 100, pH 7.40, COHb 20%
(2) Hb 12, PaO₂ 100, pH 7.40, COHb 0

b) (1) Hb 15, PaO₂ 90, pH 7.20, COHb 5%
(2) Hb 15, PaO₂ 50, pH 7.40, COHb 0

c) (1) Hb 5, PaO₂ 60, pH 7.40, COHb 0
(2) Hb 15, PaO₂ 100, pH 7.40, COHb 20%

d) (1) Hb 10, PaO₂ 60, pH 7.30, COHb 10%
(2) Hb 15, PaO₂ 100, pH 7.40, COHb 15%
SaO₂ and CaO₂: Test Your Understanding - Answers

a) (1) \( \text{CaO}_2 = 0.78 \times 15 \times 1.34 = 15.7 \text{ ml O}_2/\text{dl} \)
(2) \( \text{CaO}_2 = 0.98 \times 12 \times 1.34 = 15.8 \text{ ml O}_2/\text{dl} \)

The oxygen contents are almost identical, and therefore neither patient is more hypoxemic. However, patient (1), with 20% CO, is more hypoxic than patient (2) because of the left-shift of the O₂-dissociation curve caused by the excess CO.

b) (1) \( \text{CaO}_2 = 0.87 \times 15 \times 1.34 = 17.5 \text{ ml O}_2/\text{dl} \)
(2) \( \text{CaO}_2 = 0.85 \times 15 \times 1.34 = 17.1 \text{ ml O}_2/\text{dl} \)

A PaO₂ of 90 mm Hg with pH of 7.20 gives an SaO₂ of @ 92%; subtracting 5% COHb from this value gives a true SaO₂ of 87%, used in the CaO₂ calculation of patient (1). A PaO₂ of 50 mm Hg with normal pH gives an SaO₂ of 85%. Thus patient (2) is slightly more hypoxemic.

c) (1) \( \text{CaO}_2 = 0.90 \times 5 \times 1.34 = 6.0 \text{ ml O}_2/\text{dl} \)
(2) \( \text{CaO}_2 = 0.78 \times 15 \times 1.34 = 15.7 \text{ ml O}_2/\text{dl} \)

Patient (1) is more hypoxemic, because of severe anemia.

d) (1) \( \text{CaO}_2 = 0.87 \times 10 \times 1.34 = 11.7 \text{ ml O}_2/\text{dl} \)
(2) \( \text{CaO}_2 = 0.83 \times 15 \times 1.34 = 16.7 \text{ ml O}_2/\text{dl} \)

Patient (1) is more hypoxemic.
Acid-base Balance
Henderson-Hasselbalch Equation

\[
\text{pH} = \text{pK} + \log \frac{[\text{HCO}_3^-]}{0.03 \text{[PaCO}_2\text{]}}
\]

For teaching purposes, the H-H equation can be shortened to its basic relationships:

\[
\text{pH} \sim \frac{\text{HCO}_3^-}{\text{PaCO}_2}
\]
pH is inversely related to \([H^+]\); a pH change of 1.00 represents a 10-fold change in \([H^+]\).  

<table>
<thead>
<tr>
<th>pH</th>
<th>([H^+]) in nanomoles/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.00</td>
<td>100</td>
</tr>
<tr>
<td>7.10</td>
<td>80</td>
</tr>
<tr>
<td>7.30</td>
<td>50</td>
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<tr>
<td>7.40</td>
<td>40</td>
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<tr>
<td>7.52</td>
<td>30</td>
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<tr>
<td>7.70</td>
<td>20</td>
</tr>
<tr>
<td>8.00</td>
<td>10</td>
</tr>
</tbody>
</table>
**Acid-base Terminology**

- **Acidemia**: blood pH < 7.35
- **Acidosis**: a primary physiologic process that, occurring alone, tends to cause acidemia. Examples: metabolic acidosis from decreased perfusion (lactic acidosis); respiratory acidosis from hypoventilation. If the patient also has an alkalosis at the same time, the resulting blood pH may be low, normal, or high.
- **Alkalemia**: blood pH > 7.45
- **Alkalosis**: a primary physiologic process that, occurring alone, tends to cause alkalemia. Examples: metabolic alkalosis from excessive diuretic therapy; respiratory alkalosis from acute hyperventilation. If the patient also has an acidosis at the same time, the resulting blood pH may be high, normal, or low.
Primary acid-base disorder: One of the four acid-base disturbances that is manifested by an initial change in HCO$_3^-$ or PaCO$_2$. They are: *metabolic acidosis* (MAc), *metabolic alkalosis* (MAlk), *respiratory acidosis* (RAc), and *respiratory alkalosis* (RAlk). If HCO$_3^-$ changes first, the disorder is either MAc (reduced HCO$_3^-$ and acidemia) or MAlk (elevated HCO$_3^-$ and alkalemia). If PaCO$_2$ changes first, the problem is either RAlk (reduced PaCO$_2$ and alkalemia) or RAc (elevated PaCO$_2$ and acidemia).

Compensation: The change in HCO$_3^-$ or PaCO$_2$ that results from the primary event. Compensatory changes are *not* classified by the terms used for the four primary acid-base disturbances. For example, a patient who hyperventilates (lowers PaCO$_2$) solely as compensation for MAc does *not* have a RAlk, the latter being a primary disorder that, alone, would lead to alkalemia. In simple, uncomplicated MAc the patient will never develop alkalemia.
**Primary Acid-base Disorders: Respiratory Alkalosis**

Respiratory alkalosis - A primary disorder where the first change is a lowering of PaCO$_2$, resulting in an elevated pH. Compensation (bringing the pH back down toward normal) is a secondary lowering of bicarbonate (HCO$_3^-$) by the kidneys; this reduction in HCO$_3^-$ is not metabolic acidosis, since it is not a primary process.

<table>
<thead>
<tr>
<th>Primary Event</th>
<th>Compensatory Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCO$_3^-$</td>
<td>↓HCO$_3^-$</td>
</tr>
<tr>
<td>↑ pH ~ -------</td>
<td>↑ pH ~</td>
</tr>
<tr>
<td>↓ PaCO$_2$</td>
<td>↓ PaCO$_2$</td>
</tr>
</tbody>
</table>
**Respiratory acidosis** - A primary disorder where the first change is an elevation of PaCO$_2$, resulting in decreased pH. Compensation (bringing pH back up toward normal) is a secondary retention of bicarbonate by the kidneys; this elevation of HCO$_3^-$ is not metabolic alkalosis since it is not a primary process.

<table>
<thead>
<tr>
<th>Primary Event</th>
<th>Compensatory Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCO$_3^-$</td>
<td>↑HCO$_3^-$</td>
</tr>
<tr>
<td>↓pH ~ --------</td>
<td>↓pH ~ --------------</td>
</tr>
<tr>
<td>↑PaCO$_2$</td>
<td>↑PaCO$_2$</td>
</tr>
</tbody>
</table>
**Metabolic acidosis** - A primary acid-base disorder where the first change is a lowering of $\text{HCO}_3^-$, resulting in decreased pH. Compensation (bringing pH back up toward normal) is a secondary hyperventilation; this lowering of PaCO$_2$ is not respiratory alkalosis since it is not a primary process.

<table>
<thead>
<tr>
<th>Primary Event</th>
<th>Compensatory Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ HCO$_3^-$</td>
<td>↓ HCO$_3^-$</td>
</tr>
<tr>
<td>↓ pH ~</td>
<td>↓ pH ~</td>
</tr>
<tr>
<td>PaCO$_2$</td>
<td>↓ PaCO$_2$</td>
</tr>
</tbody>
</table>
**Primary Acid-base Disorders: Metabolic Alkalosis**

Metabolic alkalosis - A primary acid-base disorder where the first change is an elevation of HCO$_3^-$, resulting in increased pH. Compensation is a secondary hypoventilation (increased PaCO$_2$), which is not respiratory acidosis since it is not a primary process. Compensation for metabolic alkalosis (attempting to bring pH back down toward normal) is less predictable than for the other three acid-base disorders.

<table>
<thead>
<tr>
<th>Primary Event</th>
<th>Compensatory Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ HCO$_3^-$</td>
<td>↑ HCO$_3^-$</td>
</tr>
<tr>
<td>↑ pH ~ ------</td>
<td>↑ pH ~ ------</td>
</tr>
<tr>
<td>PaCO$_2$</td>
<td>PaCO$_2$</td>
</tr>
</tbody>
</table>

![Society of Critical Care Medicine - The Intensive Care Professionals](image)
Metabolic acidosis is conveniently divided into elevated and normal anion gap (AG) acidosis. AG is calculated as

\[ AG = \text{Na}^+ - (\text{Cl}^- + \text{CO}_2) \]

Note: \(\text{CO}_2\) in this equation is the “total \(\text{CO}_2\)” measured in the chemistry lab as part of routine serum electrolytes, and consists mostly of bicarbonate. Normal AG is typically 12 ± 4 mEq/L. If AG is calculated using K\(^+\), the normal AG is 16 ± 4 mEq/L. Normal values for AG may vary among labs, so one should always refer to local normal values before making clinical decisions based on the AG.
Metabolic Acid-base Disorders: Some Clinical Causes

**METABOLIC ACIDOSIS**
- Increased anion gap
  - lactic acidosis; ketoacidosis; drug poisonings (e.g., aspirin, ethylene glycol, methanol)
- Normal anion gap
  - diarrhea; some kidney problems (e.g., renal tubular acidosis, interstitial nephritis)

**METABOLIC ALKALOSIS**
- Chloride responsive (responds to NaCl or KCl therapy): contraction alkalosis, diuretics, corticosteroids, gastric suctioning, vomiting
- Chloride resistant: any hyperaldosterone state (e.g., Cushing’s syndrome, Bartter’s syndrome, severe K⁺ depletion)
**Respiratory Acid-base Disorders:** Some Clinical Causes

**Respiratory Acidosis**

$\uparrow$PaCO$_2$ & $\downarrow$ pH

- Central nervous system depression (e.g., drug overdose)
- Chest bellows dysfunction (e.g., Guillain-Barré syndrome, myasthenia gravis)
- Disease of lungs and/or upper airway (e.g., chronic obstructive lung disease, severe asthma attack, severe pulmonary edema)

**Respiratory Alkalosis**

$\downarrow$PaCO$_2$ & $\uparrow$ pH

- Hypoxemia (includes altitude)
- Anxiety
- Sepsis
- Any acute pulmonary insult (e.g., pneumonia, mild asthma attack, early pulmonary edema, pulmonary embolism)
Mixed Acid-base Disorders are Common

- In chronically ill respiratory patients, mixed disorders are probably more common than single disorders, e.g., RAc + MAlk, RAc + Mac, Ralk + MAlk.

- In renal failure (and other conditions) combined MAlk + MAc is also encountered.

- Always be on the lookout for mixed acid-base disorders. They can be missed!
**TIP 1.** Do not interpret any blood gas data for acid-base diagnosis without closely examining the serum electrolytes: Na⁺, K⁺, Cl⁻, and CO₂.

- A serum CO₂ out of the normal range always represents some type of acid-base disorder (barring lab or transcription error).
- High-serum CO₂ indicates metabolic alkalosis &/or bicarbonate retention as compensation for respiratory acidosis.
- Low-serum CO₂ indicates metabolic acidosis &/or bicarbonate excretion as compensation for respiratory alkalosis.
- *Note that serum CO₂ may be normal in the presence of two or more acid-base disorders.*
**TIP 2.** Single acid-base disorders do not lead to normal blood pH. Although pH can end up in the normal range (7.35 - 7.45) with a single mild acid-base disorder, a truly normal pH with distinctly abnormal $\text{HCO}_3^-$ and $\text{PaCO}_2$ invariably suggests two or more primary disorders.

- Example: pH 7.40, $\text{PaCO}_2$ 20 mm Hg, $\text{HCO}_3^-$ 12 mEq/L in a patient with sepsis. Normal pH results from two co-existing and unstable acid-base disorders - acute respiratory alkalosis and metabolic acidosis.
TIP 3. Simplified rules predict the pH and HCO$_3$ for a given change in PaCO$_2$. If the pH or HCO$_3$ is higher or lower than expected for the change in PaCO$_2$, the patient probably has a metabolic acid-base disorder as well.

The next slide shows expected changes in pH and HCO$_3$ (in mEq/L) for a 10-mm Hg change in PaCO$_2$ resulting from either primary hypoventilation (respiratory acidosis) or primary hyperventilation (respiratory alkalosis).
Expected changes in pH and HCO$_3^-$ for a 10-mm Hg change in PaCO$_2$ resulting from either primary hypoventilation (respiratory acidosis) or primary hyperventilation (respiratory alkalosis):

<table>
<thead>
<tr>
<th></th>
<th>ACUTE</th>
<th>CHRONIC</th>
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</thead>
<tbody>
<tr>
<td><strong>Resp Acidosis</strong></td>
<td>pH ↓ by 0.07</td>
<td>pH ↓ by 0.03</td>
</tr>
<tr>
<td></td>
<td>HCO$_3^-$ ↑ by 1*</td>
<td>HCO$_3^-$ ↑ by 3 - 4</td>
</tr>
<tr>
<td><strong>Resp Alkalosis</strong></td>
<td>pH ↑ by 0.08</td>
<td>pH ↑ by 0.03</td>
</tr>
<tr>
<td></td>
<td>HCO$_3^-$ ↓ by 2</td>
<td>HCO$_3^-$ ↓ by 5</td>
</tr>
</tbody>
</table>

* Units for HCO$_3^-$ are mEq/L
Predicted changes in $\text{HCO}_3^-$ for a directional change in $\text{PaCO}_2$ can help uncover mixed acid-base disorders.

a) A normal or slightly low $\text{HCO}_3^-$ in the presence of hypercapnia suggests a concomitant metabolic acidosis, e.g., pH 7.27, $\text{PaCO}_2$ 50 mm Hg, $\text{HCO}_3^-$ 22 mEq/L. Based on the rule for increase in $\text{HCO}_3^-$ with hypercapnia, it should be at least 25 mEq/L in this example; that it is only 22 mEq/L suggests a concomitant metabolic acidosis.

b) A normal or slightly elevated $\text{HCO}_3^-$ in the presence of hypocapnia suggests a concomitant metabolic alkalosis, e.g., pH 7.56, $\text{PaCO}_2$ 30 mm Hg, $\text{HCO}_3^-$ 26 mEq/L. Based on the rule for decrease in $\text{HCO}_3^-$ with hypocapnia, it should be at least 23 mEq/L in this example; that it is 26 mEq/L suggests a concomitant metabolic alkalosis.
TIP 4. In maximally-compensated metabolic acidosis, the numerical value of PaCO₂ should be the same (or close to) as the last two digits of arterial pH. This observation reflects the formula for expected respiratory compensation in metabolic acidosis:

\[
\text{Expected \text{PaCO}_2} = [1.5 \times \text{serum CO}_2] + (8 \pm 2)
\]

In contrast, compensation for metabolic alkalosis (by increase in PaCO₂) is highly variable, and in some cases there may be no or minimal compensation.
Acid-base Disorders: Test Your Understanding

1. A patient’s arterial blood gas shows pH of 7.14, PaCO₂ of 70 mm Hg, and HCO₃⁻ of 23 mEq/L. How would you describe the likely acid-base disorder(s)?

2. A 45-year-old man comes to the hospital complaining of dyspnea for three days. Arterial blood gas reveals pH 7.35, PaCO₂ 60 mm Hg, PaO₂ 57 mm Hg, HCO₃⁻ 31 mEq/L. How would you characterize his acid-base status?
1. Acute elevation of PaCO₂ leads to reduced pH, i.e., an acute respiratory acidosis. However, is the problem only acute respiratory acidosis or is there some additional process? For every 10-mm Hg rise in PaCO₂ (before any renal compensation), pH falls about 0.07 units. Because this patient's pH is down 0.26, or 0.05 more than expected for a 30-mm Hg increase in PaCO₂, there must be an additional metabolic problem. Also note that with acute CO₂ retention of this degree, the HCO₃⁻ should be elevated 3 mEq/L. Thus a low-normal HCO₃⁻ with increased PaCO₂ is another way to uncover an additional metabolic disorder. Decreased perfusion leading to mild lactic acidosis would explain the metabolic component.

2. PaCO₂ and HCO₃⁻ are elevated, but HCO₃⁻ is elevated more than would be expected from acute respiratory acidosis. Since the patient has been dyspneic for several days it is fair to assume a chronic acid-base disorder. Most likely this patient has a chronic or partially compensated respiratory acidosis. Without electrolyte data and more history, you cannot diagnose an accompanying metabolic disorder.
3. State whether each of the following statements is true or false.

a) Metabolic acidosis is always present when the measured serum CO₂ changes acutely from 24 to 21 mEq/L.

b) In acute respiratory acidosis, bicarbonate initially rises because of the reaction of CO₂ with water and the resultant formation of H₂CO₃.

c) If pH and PaCO₂ are both above normal, the calculated bicarbonate must also be above normal.

d) An abnormal serum CO₂ value always indicates an acid-base disorder of some type.

e) The compensation for chronic elevation of PaCO₂ is renal excretion of bicarbonate.

f) A normal pH with abnormal HCO₃⁻ or PaCO₂ suggests the presence of two or more acid-base disorders.

g) A normal serum CO₂ value indicates there is no acid-base disorder.

h) Normal arterial blood gas values rule out the presence of an acid-base disorder.
Acid-base Disorders: Test Your Understanding - Answers

3. a) false
   b) true
   c) true
   d) true
   e) false
   f) true
   g) false
Determine existence of acid-base disorder from arterial blood gas and/or serum electrolyte measurements. Check serum CO₂; if abnormal, there is an acid-base disorder. If the anion gap is significantly increased, there is a metabolic acidosis.

Examine pH, PaCO₂, and HCO₃⁻ for the obvious primary acid-base disorder and for deviations that indicate mixed acid-base disorders (TIPS 2 through 4).
Use a full clinical assessment (history, physical exam, other lab data including previous arterial blood gases and serum electrolytes) to explain each acid-base disorder. Remember that co-existing clinical conditions may lead to opposing acid-base disorders, so that pH can be high when there is an obvious acidosis or low when there is an obvious alkalosis.

Treat the underlying clinical condition(s); this will usually suffice to correct most acid-base disorders. If there is concern that acidemia or alkalemia is life-threatening, aim toward correcting pH into the range of 7.30 - 7.52 ([H+] = 50-30 nM/L).

Clinical judgment should always apply.
Case 1. A 55-year-old man is evaluated in the pulmonary lab for shortness of breath. His regular medications include a diuretic for hypertension and one aspirin a day. He smokes a pack of cigarettes a day.

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<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>FIO₂</td>
<td>.21</td>
<td>HCO₃⁻</td>
<td>30 mEq/L</td>
</tr>
<tr>
<td>pH</td>
<td>7.53</td>
<td>%COHb</td>
<td>7.8%</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>37 mm Hg</td>
<td>Hb</td>
<td>14 gm%</td>
</tr>
<tr>
<td>PaO₂</td>
<td>62 mm Hg</td>
<td>CaO₂</td>
<td>16.5 ml O₂/dl</td>
</tr>
<tr>
<td>SaO₂</td>
<td>87%</td>
<td></td>
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</tbody>
</table>

How would you characterize his state of oxygenation, ventilation, and acid-base balance?
Arterial Blood Gases: Test Your Overall Understanding

Case 1 - Discussion

OXYGENATION: The PaO₂ and SaO₂ are both reduced on room air. Since P(A-a)O₂ is elevated (approximately 43 mm Hg), the low PaO₂ can be attributed to V-Q imbalance, i.e., a pulmonary problem. SaO₂ is reduced, in part from the low PaO₂ but mainly from elevated carboxyhemoglobin, which in turn can be attributed to cigarettes. The arterial oxygen content is adequate.

VENTILATION: Adequate for the patient's level of CO₂ production; the patient is neither hyper- nor hypo-ventilating.

ACID-BASE: Elevated pH and HCO₃⁻ suggest a state of metabolic alkalosis, most likely related to the patient's diuretic; his serum K⁺ should be checked for hypokalemia.
**Case 2.** A 46-year-old man has been in the hospital two days with pneumonia. He was recovering but has just become diaphoretic, dyspneic, and hypotensive. He is breathing oxygen through a nasal cannula at 3 l/min.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.40</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>20 mm Hg</td>
</tr>
<tr>
<td>%COHb</td>
<td>1.0%</td>
</tr>
<tr>
<td>PaO₂</td>
<td>80 mm Hg</td>
</tr>
<tr>
<td>SaO₂</td>
<td>95%</td>
</tr>
<tr>
<td>Hb</td>
<td>13.3 gm%</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>12 mEq/L</td>
</tr>
<tr>
<td>CaO₂</td>
<td>17.2 ml O₂/dl</td>
</tr>
</tbody>
</table>

How would you characterize his state of oxygenation, ventilation, and acid-base balance?
Arterial Blood Gases: Test Your Overall Understanding

Case 2 - Discussion

OXYGENATION: The PaO₂ is lower than expected for someone hyperventilating to this degree and receiving supplemental oxygen, and points to significant V-Q imbalance. The oxygen content is adequate.

VENTILATION: PaCO₂ is half normal and indicates marked hyperventilation.

ACID-BASE: Normal pH with very low bicarbonate and PaCO₂ indicates combined respiratory alkalosis and metabolic acidosis. If these changes are of sudden onset, the diagnosis of sepsis should be strongly considered, especially in someone with a documented infection.
Arterial Blood Gases: Test Your Overall Understanding

Case 3. A 58-year-old woman is being evaluated in the emergency department for acute dyspnea.

- **FIO₂**: 0.21
- **pH**: 7.19
- **PaCO₂**: 65 mm Hg
- **%COHb**: 1.1%
- **PaO₂**: 45 mm Hg
- **SaO₂**: 90%
- **Hb**: 15.1 gm%
- **HCO₃⁻**: 24 mEq/L
- **CaO₂**: 18.3 ml O₂/dl

How would you characterize her state of oxygenation, ventilation, and acid-base balance?
Case 3 - Discussion

**OXYGENATION:** The patient's PaO₂ is reduced for two reasons - hypercapnia and V-Q imbalance - the latter apparent from an elevated P(A-a)O₂ (approximately 27 mm Hg).

**VENTILATION:** The patient is hypoventilating.

**ACID-BASE:** pH and PaCO₂ are suggestive of acute respiratory acidosis plus metabolic acidosis; the calculated HCO₃⁻ is lower than expected from acute respiratory acidosis alone.
Case 4. A 23-year-old man is being evaluated in the emergency room for severe pneumonia. His respiratory rate is 38/min and he is using accessory breathing muscles.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO₂</td>
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</tr>
<tr>
<td>pH</td>
<td>7.29</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>55 mm Hg</td>
</tr>
<tr>
<td>PaO₂</td>
<td>47 mm Hg</td>
</tr>
<tr>
<td>SaO₂</td>
<td>86%</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>23 mEq/L</td>
</tr>
<tr>
<td>%COHb</td>
<td>2.1%</td>
</tr>
<tr>
<td>Hb</td>
<td>13 gm%</td>
</tr>
<tr>
<td>CaO₂</td>
<td>15.8 ml O₂/dl</td>
</tr>
<tr>
<td>Na⁺</td>
<td>154 mEq/L</td>
</tr>
<tr>
<td>K⁺</td>
<td>4.1 mEq/L</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>100 mEq/L</td>
</tr>
<tr>
<td>CO₂</td>
<td>24 mEq/L</td>
</tr>
</tbody>
</table>

How would you characterize his state of oxygenation, ventilation, and acid-base balance?
Case 4 - Discussion

**OXYGENATION:** The PaO₂ and SaO₂ are both markedly reduced on 90% inspired oxygen, indicating severe ventilation-perfusion imbalance.

**VENTILATION:** The patient is hypoventilating despite the presence of tachypnea, indicating significant dead-pace ventilation. This is a dangerous situation that suggests the need for mechanical ventilation.

**ACID-BASE:** The low pH, high PaCO₂, and slightly low calculated HCO₃⁻ all point to combined acute respiratory acidosis and metabolic acidosis. Anion gap is elevated to 30 mEq/L indicating a clinically significant anion gap (AG) acidosis, possibly from lactic acidosis. With an of AG of 30 mEq/L, his serum CO₂ should be much lower, to reflect buffering of the increased acid. However, his serum CO₂ is near normal, indicating a primary process that is increasing it, i.e., a metabolic alkalosis in addition to a metabolic acidosis. The cause of the alkalosis is as yet undetermined. In summary: this patient has respiratory acidosis, metabolic acidosis, and metabolic alkalosis.
Arterial Blood Gas Interpretation

Lawrence Martin, MD, FACP, FCCP

Associate Professor of Medicine
Case Western Reserve University School of Medicine, Cleveland
larry.martin@adelphia.net

The End