Arterial Blood Gases – Interpretation & Sampling

CRC 330 – Cardiorespiratory Assessment Skills
Bill Pruitt, MBA, CPFT, RRT, AE-C, FAARC

Rev 8/24/13
References and supplies

**REQUIRED:**

- Wilkins 7th *Assessment* Ch 8 READ THIS FIRST
  - How to interpret an ABG
- Egan’s 10th Fundamentals– Ch 11, 13, 18
  - Interpretation in depth
- White 5th ed *Competencies* Ch 8
  - How to obtain an ABG from a puncture and an A line
- Chang’s *Respiratory Care Calculations*
- AARC CPG: Blood Gas Analysis and Hemoximetry 2013 Revision & Update
- Supplies: Handout, worksheets, ABG kits
The Arterial Blood Gas Report
“ABGs”

“...the most clinically useful measurement in respiratory care.”

“...the cornerstone in the diagnosis and management of clinical oxygenation and acid-base disturbances.” -- Malley
ABG

- Invasive test (must draw a blood sample)
  - Gives the best information on oxygenation, ventilation, and acid-base status
  - The “Gold standard”

- Supported by other non-invasive tests
  - Pulse oximetry (SpO₂)
  - End-tidal CO₂ (ETCO₂)
  - Transcutaneous O₂ (PtcO₂)
  - Transcutaneous CO₂ (PtcCO₂)
Pulmonary System Fundamentals

- The pulmonary system does two primary things:
  - It gets rid of CO$_2$ and brings in O$_2$
- The cardiovascular system transports these gases from their points of origin (CO$_2$ produced at the tissue level, O$_2$ comes in through the alveoli)
- ABGs measure pulmonary system function directly and the cardiovascular system function indirectly
Good ABGs –
a balance of proper alveolar ventilation…….. and
And proper perfusion.
For delivery to the tissues ... consider Hb, C.O.
Normal Ranges for Blood Gas Values (for adults)

Arterial
- pH ...7.35 to 7.45
- PaO₂ ..80 to 100 mm Hg
- PaCO₂ .35 to 45 mm Hg
- HCO₃⁻ ...22 to 26 mEq/l
- SaO₂.......92-100%
- BE.......-2 to +2 mEq/L

BE = base excess (changes in base/acid levels due to non-respiratory causes)

Mixed Venous
- pH .......7.33 to 7.43
- PvO₂ ....35 to 45 mm Hg
- PvCO₂ ..45 to 50 mm Hg
- HCO₃⁻ ...24 to 28 mEq/l
- SvO₂.....70 to 75%
- BE........0 to +4 mEq/L
“Absolute” Blood Gas Values

In a perfect world with no disease or aging

- **Arterial (ABG)**
  - pH .... 7.40
  - PaO$_2$ ... 100 mm Hg
  - PaCO$_2$ ... 40 mm Hg
  - HCO$_3^-$ .... 24 mEq/l
  - SaO$_2$.......>95%

- **Mixed Venous (VBG)**
  - pH ... 7.35
  - PvO$_2$ ... 40 mm Hg
  - PvCO$_2$ ... 46 mm Hg
  - HCO$_3^-$ ... 26 mEq/l
  - SvO$_2$...75%
You have to know this to go forward

- Memorize the absolute normal values and the normal ranges for arterial and mixed venous blood.
Acid–base interpretation

- Relies on 3 factors (pH, PCO$_2$, and HCO$_3^-$)

  1. pH (normal = 7.35 - 7.45)*
     - If < 7.35 – acidemia
     - If > 7.45 – alkalemia (remember “base”)

  2. PCO$_2$ (normal = 35 – 45 mm Hg)
     - If > 45 mm Hg – acidemia
     - If < 35 mm Hg– alkalemia (remember “base”)

  3. HCO$_3^-$ (normal = 22 – 26 mm Hg)
     - If < 22 mEq/L – acidemia
     - If > 26 mEq/L – alkalemia (remember “base”)

* If pH moves off 7.40 – it is “leaning” toward alkalosis or acidosis
5 KEY Steps for interpreting an ABG

1. Examine the pH and label it based on 7.40. Is it an acidosis or an alkalosis (base)? Label it (A for acid or B for base)
2. Examine both players: CO₂ and HCO₃⁻ using normal ranges… acidosis or alkalosis (base)? Label them A (acid), B (base), or N -normal
3. See which matches up pH and player (A-A or B-B). This tells you the primary cause (respiratory or metabolic) #1 problem
Interpretation (cont)

4. If CO$_2$ or HCO$_3^-$ is contrary to the pH, (example pH leaning acid, and CO$_2$ base)…. this occurs with compensation. Then #2…. the “second player” is working to fix the pH. Evaluate for level of compensation

5. If all 3 are labeled the same (A-A-A or B-B-B) it is a combined acidosis or alkalosis (no compensation at all)
Second player and compensation

- If #2 player is within normal range (WNR) with a bad pH, it is an uncompensated blood gas
- If #2 is out of normal in opposite direction of primary cause but pH is not WNR, it is a partially compensated blood gas
- If moving to oppose the pH problem = trying to compensate. If pH is WNR and #2 is out of normal (opposing the problem), it is a compensated blood gas
## Classification of ABG’s for Acid/base status

<table>
<thead>
<tr>
<th>pH</th>
<th>PCO(_2)</th>
<th>HCO(_3^−)</th>
<th>What is it?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.  7.50</td>
<td>30</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>2.  7.30</td>
<td>50</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>3.  7.47</td>
<td>41</td>
<td>27</td>
<td></td>
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<tr>
<td>4.  7.32</td>
<td>39</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>5.  7.55</td>
<td>25</td>
<td>23</td>
<td></td>
</tr>
<tr>
<td>6.  7.25</td>
<td>48</td>
<td>20</td>
<td></td>
</tr>
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<tbody>
<tr>
<td>1. 7.50b</td>
<td>30b</td>
<td>23n</td>
<td>Uncomp. Resp Alk.</td>
</tr>
<tr>
<td>2. 7.30a</td>
<td>50a</td>
<td>25n</td>
<td>Uncomp. Resp Acidosis</td>
</tr>
<tr>
<td>3. 7.47b</td>
<td>41n</td>
<td>27b</td>
<td>Uncomp. Meta Alk</td>
</tr>
<tr>
<td>4. 7.32a</td>
<td>39n</td>
<td>21a</td>
<td>Uncomp. Meta. Acidosis</td>
</tr>
<tr>
<td>5. 7.55b</td>
<td>25b</td>
<td>23n</td>
<td>Uncomp Resp Alk</td>
</tr>
<tr>
<td>6. 7.25a</td>
<td>48a</td>
<td>20a</td>
<td>Combined Resp&amp;Meta Acid</td>
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<td>27</td>
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<tr>
<td>2.</td>
<td>7.36</td>
<td>64</td>
<td>35</td>
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<tr>
<td>3.</td>
<td>7.57</td>
<td>23</td>
<td>21</td>
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<td>17</td>
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<td>20</td>
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</thead>
<tbody>
<tr>
<td>1. 7.30a</td>
<td>55a</td>
<td>27b</td>
<td>Partially comp. resp acidosis</td>
</tr>
<tr>
<td>2. 7.36a</td>
<td>64a</td>
<td>35b</td>
<td>Compensated resp. acidosis</td>
</tr>
<tr>
<td>3. 7.57b</td>
<td>23b</td>
<td>21a</td>
<td>Part. comp. resp. alkalosis</td>
</tr>
<tr>
<td>4. 7.44b</td>
<td>26b</td>
<td>17a</td>
<td>Comp. resp. alkalosis</td>
</tr>
<tr>
<td>5. 7.30a</td>
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Compensation

**Compensation** – body has correcting process for an acid/base imbalance that has a primary cause (primary player).

- In compensation the pH moves back to normal range by autoregulation of the “second player”.

- Or – if successful, the secondary player has “fixed” the pH problem

- Compensation takes time…. Has 3 stages
3 stages of Compensation

- **Uncompensated** – first stage, early in process

  pH outside normal range due to one or the other abnormal condition … the primary cause. Could be a pulmonary problem or a metabolic problem.

- The other player is still within normal range and no compensation has occurred

  Example pH 7.25, PCO$_2$ - 38 mm Hg, HCO$_3^-$ - 18 mEq/L
**Partially compensated** - abnormal pH – Both pH and primary cause are out of normal limits in one direction and ……

The secondary player is trying to compensate by moving out of normal range in the opposite direction (so both PaCO$_2$ and HCO$_3^-$ are out of normal range)…. but the secondary player hasn’t gone far enough to fix the problem.

Example pH 7.31, PCO$_2$ - 28 mm Hg, HCO$_3^-$ - 18 mEq/L
Compensated - pH within normal range but both PaCO$_2$ and HCO$_3^-$ out of normal range but in opposite directions.

- One is the primary cause - the other is compensating (second player). One “acid”, the other “alkaline” or “basic”
  - pH is usually “leaning” toward acidosis or alkalosis which gives a clue to the primary cause

Example pH 7.36, PCO2 - 22 mm Hg, HCO$_3^-$ - 18 mEq/L
Acid-base balance
No abnormality

$\text{PCO}_2$
40 mmHg

$\text{HCO}_3^-$
24 mEq/L

$\text{pH}$
7.40
Acid-base balance
Respiratory acidosis

\[ \text{PCO}_2 \]
50 mmHg

\[ \text{pH} \]
7.20

\[ \text{HCO}_3^- \]
24 mEq/L
Acid-base balance
Partially compensated respiratory acidosis

PCO$_2$
50 mmHg

pH
7.31

HCO$_3^-$
27 mEq/L
Acid-base balance
Fully compensated respiratory acidosis

\[ \text{PCO}_2 \quad 50 \text{ mmHg} \]

\[ \text{HCO}_3^- \quad 31 \text{ mEq/L} \]

\[ \triangle \quad \text{pH} \quad 7.37 \]
Where is CO₂ produced? Why is O₂ important?

Glucose

Aerobic metabolism (Krebs cycle)

- Oxygen present
- 36 ATP
- 6 CO₂
- 6 H₂O

Anaerobic metabolism (No O₂)

- 2 ATP
- Lactic acid
ABG Values
(measured by a blood gas machine)

- **pH**
  - Measures the level of hydrogen ions (H+) in the blood (acid or alkaline)

- **PaCO\(_2\)**
  - Measures the partial pressure exerted by the CO\(_2\) gas dissolved in the plasma (Uses mmHg or torr for units of measure)

- **PaO\(_2\)**
  - Measures the partial pressure exerted by the O\(_2\) gas dissolved in the plasma (mmHg or torr)

Note: a = arterial blood, v = venous blood
Calculated Values

- **HCO₃⁻ (mEq/L)** - Bicarbonate ions

- **BE (mEq/L)** - Base excess
  - BE = changes in base/acid levels due to non-respiratory causes

- **SaO₂ (percent)** - % of Hb that is saturated with oxygen
Additional Measurements

- **SaO₂ (%)** –
  - To get actual measurement must use a *co-oximeter machine* (Not SpO2)

- **Hematocrit (%)** –
  - Percentage of blood volume that is made up of cells versus plasma

- **Hemoglobin (g/dL)** –
  - The primary carrier for oxygen molecules (found in the RBC)

- **Electrolytes (mEq/L)**
  - Primarily Na⁺, K⁺, Ca⁺, and Cl⁻
Additional Measurements: Abnormal hemoglobin

- **Carboxyhemoglobin (%)** Normal value 0-2%
  - COHb forms when carbon monoxide binds to Hb – stops Hb ability to carry $O_2$
  - Cigarette smoking results in 5–10% COHb.
  - 10% and above can affect delivery of $O_2$ to the tissues (CO poisoning)

- **Methemoglobin (%)** Normal value 0 – 1%
  - Oxidation of the normal ferrous iron contained in the heme part of hemoglobin to ferric iron.
  - Like COHb – MetHb stops the normal delivery of $O_2$ to the tissues by disrupting the Hb ability to carry $O_2$
  - Caused by certain drugs: lidocaine, nitric oxide, nitroprusside, benzocaine spray
Additional Measurements: Abnormal hemoglobin

- Fetal hemoglobin (Hemoglobin F or HbF)
  - High affinity for oxygen
  - Usually converts to adult hemoglobin (HbA) by 6 months after birth
Sickle cell anemia (SCA)

- Inherited trait (not contagious)
- Normal RBC is curved like a sickle, lives 10-20 days (normal RBC -120 days)
- Sickle shape causes pain in a SCA crisis, often pt is hospitalized
  - Most common in Africans and African-Americans. Also found in other ethnic and racial groups, including people from South and Central America, the Caribbean, Mediterranean countries, and India.
  - Affects about 100,000 people in the US.
Additional Measurements

- **Lactate (Lactic acid)**
  - Normal levels (arterial blood) 3–7 mg/dL
  - High levels with strenuous exercise (anaerobic metabolism) or other conditions such as heart failure, a severe infection (sepsis), or shock (decreased blood flow and oxygen delivery throughout the body).
  - Poisons: alcohol (ethanol), wood alcohol (methanol), or antifreeze (ethylene glycol)
  - Liver damage/disease - the liver normally breaks down lactic acid.
Venous Samples - Advantages

- Blood pressures are lower
- Collateral vessels are abundant
- Interruption of venous flow less significant
- More accessible, more sites
- Great for drawing routine labwork
Venous Samples - Problems

- Subject to local influences
- May be difficult to obtain during cardiovascular collapse
- Provide no information regarding pulmonary system gas exchange (oxygen and carbon dioxide levels are measured after exchange at the tissues)
Arterial Samples - Advantages

- Uniform, mixed sample
- Uniformity persists in all arteries under most circumstances
- Provides information regarding how well lungs are working
Arterial Samples – Disadvantages

Hazards/Complications for ABG puncture. Know these

1. Hematoma
2. Arteriospasm
3. Air or clotted-blood emboli
4. Anaphylaxis from local anesthetic
5. Infection in patient or technician
6. Hemorrhage
7. Trauma to the vessel
8. Arterial occlusion
9. Vasovagal response
10. Pain
"Pull out, Betty! Pull out! ... You've hit an artery!"
Indications for ABG

- To evaluate the adequacy of ventilation, the level of oxygenation, the acid-base balance, the oxygen-carrying capacity of the blood, and presence of dyshemoglobins
- To measure the patient’s response to therapy (e.g., oxygen therapy) and/or perform a diagnostic evaluation (e.g., exercise testing)
- To monitor the severity and progression of a known disease
Signs/ Symptoms of Respiratory Abnormality

- Pale or cyanotic appearance
- Abnormal pattern of ventilation
- Tachypnea / dyspnea
- Appears acutely disoriented
- Tachycardia
- Diaphoresis
Pulmonary Diseases

- Pulmonary diseases affect ventilation and interfere with CO\(_2\) elimination (this increases CO\(_2\))

- Pulmonary diseases may also cause problems in oxygenation and affect O\(_2\) coming into the blood (lower O\(_2\) levels)

- Cardiovascular problems affect oxygen delivery and rarely cause problems with CO\(_2\) elimination
Severe COPD – not hospitalized

- Arterial (ABG)
  - pH .... 7.38
  - PaO₂ ...65 mm Hg
  - PaCO₂ ..60 mm Hg
  - HCO₃⁻ ....34 mEq/l
  - SaO₂......89%

"Compensated respiratory acidosis with mild hypoxemia"
ABG’s provide a measure of effectiveness for oxygen or supportive therapy

- Step 1: Obtain a baseline prior to starting oxygen and/or ventilator therapy
- Step 2: Initiate therapy
- Step 3: Assess effects of changes
  - Wait minimum 10 minutes for ventilator patients.
  - For spontaneous breathing wait 20 - 30 + minutes depending on severity of lung pathology … see AARC CPG for the normal wait times)
Supplements to ABG information

- **Pulse oximetry (SpO$_2$)**
  - Obtained with a pulse oximeter

- **Transcutaneous monitoring**
  - Diffusion through skin - PtcO$_2$, PtcCO$_2$. Used mainly with babies.
  - Newer technology has improved use in adults

- **Capnography**
  - Measures expired CO$_2$ - PetCO$_2$. Obtained with a capnograph or end-tidal CO$_2$ monitor
  - Used to monitor post-anesthesia, PCA pumps, Code Blue response (return to spontaneous circulation)
Arterial (ABG)  Non-invasive monitors

- pH
- $\text{PaO}_2$
- $\text{PaCO}_2$
- $\text{HCO}_3^-$
- $\text{SaO}_2$

- $\text{PtcO}_2$
- $\text{ETCO}_2$
- $\text{PtcCO}_2$
- $\text{SpO}_2$
Laws, guidelines, and formulas

- The CO$_2$ formula (Plasma)
- Dalton’s Law of Partial Pressure
- Henry’s Law for gases dissolving in a liquid
- Fick’s Law of Diffusion
- Alveolar Air Equation
- Henderson-Hasselbalch equation
- Arterial oxygen content
Oxygen & Carbon dioxide

- Gases move by diffusion from areas of high concentration to low concentration (high partial pressure to low partial pressure)

- Henry’s Law:
  - More gas dissolves in a liquid across the A-C membrane with higher gas pressure (larger pressure gradient)
Fick’s Law of Diffusion

- More gas dissolves in a liquid across the A-C membrane with a larger surface area.
- Less gas will diffuse across and dissolve with a thicker A-C membrane.
Respiratory System - Removal of CO$_2$

- Primary function: removal of CO$_2$ at same rate as it is produced by cellular metabolism: PaCO$_2$ ~ 40 mm Hg for normal metabolism, healthy lungs

- Alveolar ventilation is *the primary determinant* of PaCO$_2$…. as ventilation increases, CO$_2$ is “blown off” and PaCO$_2$ decreases and vice versa
One of “Basic Blood Gas Formulas”

The CO$_2$ formula

- Carbon dioxide + water (in the presence of carbonic anhydrase) produces carbonic acid which then dissociates into hydrogen ions and bicarbonate ions:

  \[
  H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^- 
  \]

  Carbonic Anhydrase

- A decrease in PaCO$_2$ will pull the reaction to the left, while increases will push to right - so changes in CO$_2$ alters H$^+$. Why is this important?
Acid/Base Terms
Normal pH range 7.35 to 7.45

- Acidemia - acid condition of the blood (described by pH < 7.35)
- Alkalemia - alkaline condition of the blood (described by pH > 7.45)
- Acidosis - process that by itself will lead to an acidemia (increase in H+ ..... decrease in pH)
- Alkalosis - process that by itself will lead to an alkalemia (decrease in H+ ..... increase in pH)
"I see your little, petrified skull ... labeled and resting on a shelf somewhere."
Acid/Base Terms

- **Acid** - any chemical substance that is capable of releasing a H\(^+\) into a solution
- **Base** - any substance that is of combining with or accepting a H\(^+\) in a solution
- **Volatile acids** - can be converted from a liquid form to a gaseous form (example - carbonic acid)
- **Non-volatile acids** are “fixed” - cannot be converted to gas (example - lactic acid)
Respiratory System - $O_2$ Delivery

- Second major function – deliver oxygen to tissues
- Impaired oxygenation leads to anaerobic metabolism and lactic acid production ($C_3H_6O_3$)
- Lactic acid increases $[H^+]$ and results in a metabolic acidosis.
  (Remember the Krebs cycle)
Respiratory impairment has two detrimental effects leading to acidosis

- Changes in respiration can cause acid/base disturbances involving both volatile and non-volatile acids
  - $\uparrow CO_2 \rightarrow \uparrow H^+ = \text{Respiratory acidosis (volatile acid)}$
  - $\downarrow O_2 \rightarrow \uparrow C_3H_6O_3 \rightarrow \uparrow H^+ = \text{Metabolic acidosis (non-volatile, lactic acidosis)}$
Arterial Blood Gas Interpretation

- Tip – memorize 2 sets of numbers:
  - “Absolute” blood gas values
  - Normal ranges for blood gases
    - Be familiar with acceptable therapeutic ranges.

- **Acid-base** status is assessed by pH, PaCO$_2$, and HCO$_3$-

- **Ventilation** status is assessed by the PaCO$_2$ and occurs during the evaluation of the acid-base status

- **Oxygenation** status uses SaO$_2$, PaO$_2$, and should take into account Hb, and C.O. for delivery to tissues.
Acid–base interpretation

- Relies on 3 factors (pH, PCO₂, and HCO₃⁻)
  1. pH (normal = 7.35 - 7.45)
     - If < 7.35 – acidemia
     - If > 7.45 – alkalemia (remember “base”)
  2. PCO₂ (normal = 35 – 45 mm Hg)
     - If > 45 – acidemia
     - If < 35 – alkalemia (remember “base”)
  3. HCO₃⁻ (normal = 22 – 26 mm Hg)
     - If < 22 – acidemia
     - If > 26 – alkalemia (remember “base”)

“Absolute” Blood Gas Values

In a perfect world with no disease or aging

Arterial
- pH .... 7.40
- PaO\textsubscript{2} ... 100 mm Hg
- PaCO\textsubscript{2} .. 40 mm Hg
- HCO\textsubscript{3}⁻ .... 24 mEq/l
- SaO\textsubscript{2} ...... >95%

Mixed Venous
- pH ... 7.35
- PvO\textsubscript{2} ... 40 mm Hg
- PvCO\textsubscript{2} .. 46 mm Hg
- HCO\textsubscript{3}⁻ ... 26 mEq/l
- SvO\textsubscript{2} ... 75%
Normal Ranges for Blood Gas Values (for adults)

**Arterial**
- pH ......7.35 to 7.45
- PaO₂ .....80 to 100 mm Hg
- PaCO₂ ..35 to 45 mm Hg
- HCO₃⁻…22 to 26 mEq/l
- SaO₂.....92 to 100%
- BE.......-2 to +2 mEq/L

**Mixed Venous**
- pH ........7.33 to 7.43
- PvO₂ ......35 to 45 mm Hg
- PvCO₂ ...45 to 50 mm Hg
- HCO₃⁻ .....24 to 28 mEq/l
- SvO₂......70 to 75%
- BE..........0 to +4 mEq/L

BE = base excess (changes in base/acid levels due to non-respiratory causes)
Acceptable Therapeutic Ranges

pH 7.30 - 7.50
PaCO₂ 30 - 50 mm Hg
PaO₂ 60 - 100 mm Hg

- Patients with these values would not usually require supportive measures
  - May be everyday “walk-around” gas values for pulmonary patients
- Patients are often seriously ill and therefore don’t have normal ABG’s
- These do not take place of “normal values”
Hospitals have established “Panic Values” or “Critical Values”
- If measured, you are required to report it to the patient’s nurse and/or the physician and document it.

Documentation is very particular and precise per hospital policy
- Insure timely and correct care is given
- Avoid liability
Critical Values at Mobile Infirmary (don’t memorize these…. FYI)

- pH < 7.25 or > 7.58
- PaCO$_2$ > 70 mmHg
- PaO$_2$ < 50 mm Hg
- SaO$_2$ < 85%
- COHb > 15%
- MetHb > 10%
- Hb < 8 or > 20 g/dl
  - Normals for men 13.5 - 16.5 g/dl
  - Normal for women 12.0 – 15.0 g/dl
Critical Values at USA Medical Center (FYI)

- pH $< 7.20$ or $> 7.55$
- $\text{PaCO}_2$ $< 20$ or $> 60$ mm Hg
- $\text{PaO}_2$ $< 50$ mm Hg
CO₂ - The Respiratory Parameter

- \( \text{PaCO}_2 \) - pressure (tension) of the dissolved CO₂ gas in arterial blood. Units = mm Hg or torr

- **Normal \( \text{PaCO}_2 \) (35 – 45 mm Hg) = normal ventilation**
CO₂ out of normal range often means trouble with ventilation

- **High PaCO₂** (>45 mm Hg) - respiratory acidemia described as hypoventilation, ventilatory failure, hypercarbia, hypercapnia

- **Low PaCO₂** (<35 mm Hg) - respiratory alkalemia described as hyperventilation, hypocarbia, hypocapnia

**KNOW ALL** these descriptive terms for high and low PaCO₂!
CO₂ Elimination

CO₂: waste product of metabolism...eliminated from the body in two ways: (converted + kidneys and through the lungs)

1. Conversion of CO₂ to H₂CO₃ which yields H⁺ and HCO₃⁻. H⁺ is excreted through kidneys

\[
\text{H}_2\text{O} + \text{CO}_2 \leftrightarrow \text{H}_2\text{CO}_3^- \leftrightarrow \text{H}^+ + \text{HCO}_3^- \\
\text{Carbonic Anhydrase}
\]

2. **Most important** is CO₂ elimination through the lungs!
Hyperventilation

- Minute volume = tidal volume x frequency
  - $V_E = V_T \times f$
  - $5.0 \text{ L} = 500 \text{ ml} \times 10$

- Hyperventilation will blow off CO$_2$ (reduction in the blood level of CO$_2$ – below 35 mm Hg)

- Hyperventilation occurs with an increased tidal volume and/or frequency

Hypoventilation- the opposite of all of this

CO$_2$ – above 45 mm Hg
Good ABGs –
a balance of proper alveolar ventilation........ and
And proper perfusion.
For delivery to the tissues .... consider Hb, C.O.
Causes of Respiratory Acidosis
(alveolar hypoventilation – high CO₂)

- Obstructive lung disease (COPD)
  - Most often V/Q mismatch
    - (V = alveolar ventilation, Q = alveolar perfusion)
  - Normal V/Q is 0.8
  - V reduced, Q normal… low V/Q

- Acute asthma (late phases)
  - V/Q mismatch, low V/Q

- CNS depression:
  - over-sedation, reduced function of the respiratory center (head trauma), narcotics (drug OD), lesions
    - V/Q mismatch, low V/Q

- Ventilatory failure, alveolar hypoventilation
Causes of Respiratory Acidosis
(alveolar hypoventilation – high CO₂)

- Neuromuscular diseases:
  - myasthenia gravis, Guillain-Barre syndrome, muscular dystrophy, polio, amyotrophic lateral sclerosis
  - V/Q mismatch, low V/Q

- Restrictive disorders:
  - Pickwickian syndrome (aka - OSA), kyphoscoliosis, morbid obesity
  - V/Q mismatch, low V/Q

- Respiratory acidosis also called ventilatory failure, alveolar hypoventilation
Causes of Respiratory Alkalosis
(alveolar hyperventilation – low CO₂)

- Hypoxemia
- Emotional disorders: nervousness and anxiety, pain, fear
- Pulmonary emboli, fibrosis, etc
- Pregnancy
- Brain injury
- Salicylates (adults)

- Fever
- Gram negative septicemia
- Hepatic insufficiency
- Congestive heart failure
- Asthma (*early or mild attack)
- Severe anemia
The real reason dinosaurs became extinct
HCO$_3^-$ : the Metabolic Parameter

HCO$_3^-$ - bicarbonate ion
(normal range 22 to 26, absolute 24)

With everything else being normal:
If HCO$_3^-$ < 22 mEq/L = Metabolic acidosis
If HCO$_3^-$ > 26 mEq/L = Metabolic alkalosis

HCO$_3^-$ levels are regulated by the kidney
Reminder: Acid/Base Definitions

- **Acid** - substance that increases hydrogen ion concentration (proton donor)
- **Base** - substance that decreases hydrogen ion concentration (proton acceptor)
- **Strong acids** - dissociate completely or nearly so
- **Buffers** - weak acids/weak bases that accept or donate hydrogen ions based on availability
What is pH?

- pH is the negative expression of the logarithmic measure of hydrogen ion (H+) concentration in the blood:
  \[ \text{pH} = -\log[H+] \]

- Exponential expressions:
  - \(1,000 = 10^3\)
  - \(1/1,000 = 10^{-3}\)
  - \(1/100,000 = 10^{-5}\)

- Logarithmic expressions:
  - \(10^3 = 3\)
  - \(10^{-3} = -3\)
  - \(10^{-5} = -5\)
  (What would the negative log be?)

- 1 L of water contains 0.0000001 moles of H+ (or \(10^{-7}\)) H+ so…….

- pH of water = 7.0 (water is a neutral solution)
Ionization Constant of Water
(Equilibrium Constant)

\[ H_2O \leftrightarrow H^+ + OH^- \] (Water dissociates into \( H^+ \) + \( OH^- \))

\[ K = \frac{[H^+][OH^-]}{[H_2O]} \]

(K is the equilibrium constant used for measuring the extent of dissociation)

\[ K_w = [H^+][OH^-] \]

\[ K_w = [1 \times 10^{-7}][1 \times 10^{-7}] = 1 \times 10^{-14} \]

K for water = 7
pH = 7.0 for pure water
Now – what about blood and hydrogen ions?
**CO₂ and Acid/Base balance**

**Carbonic Anhydrase**

\[
\text{H₂O} + \text{CO}_2 \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^-
\]

2 forms of the Henderson/Hasselbalch Equation:

1. \[\text{pH} = \text{pK} + \log\left(\frac{[\text{HCO}_3^-]}{[\text{H}_2\text{CO}_3]}\right)\]

   \(\text{pK} = \) the ionization constant for blood and is equal to 6.1

   \(\text{H}_2\text{CO}_3\) is directly related to \(\text{PCO}_2\) using 0.03 so we can substitute

2. \[\text{pH} = 6.1 + \log\left(\frac{[\text{HCO}_3^-]}{0.03 \times \text{P}_{\text{CO}_2}}\right)\]

   0.03 is used to convert mmHg to mEq
Usefulness of the Henderson-Hasselbalch Equation

- Defines a relationship between pH, CO$_2$, and HCO$_3^-$
  - Provides an equation to show the effect of changes in either [HCO$_3^-$] or [CO$_2$] on the body’s pH

- pH is not only affected by changes in CO$_2$, but also changes in ratio of [HCO$_3^-$] to [CO$_2$]

- 20:1 ratio between [HCO$_3^-$] and [CO$_2$] yields normal pH of 7.40
Buffering and the H-H equation

- See Egan page 295 Figure 13-1
  - Bicarb and non-bicarb buffer for H⁺
- Read through page 296 Mini-Clini
  - applying the H-H equation clinically

*KNOW BOTH FORMS OF THE H-H EQUATION.*

Don’t get the details mixed up – practice writing these out with the right brackets

1. \[ \text{pH} = \text{pK} + \log([\text{HCO}_3^-]/[\text{H}_2\text{CO}_3]) \]
2. \[ \text{pH} = 6.1 + \log[\text{HCO}_3^-]/0.03 \times \text{P} \text{CO}_2 \]
Acid/Base Regulation

Normal pH determined by:

- Buffering action in the blood
- Removal of CO$_2$ (remember carbonic acid - volatile acid) through respiration (lungs)
- Generation of HCO$_3^-$ by removal of fixed acids in the urine (kidney)
- Remember – Lungs (ventilation) affects CO$_2$..... Kidney alter HCO$_3^-$
Consequences of abnormal pH

7.80 (low H⁺)........Death

. Convulsions
. (alkalosis) Arrhythmias
. Irritability

7.40 __ __ __ __ Normal __ __ __ __ __ __ __ __

. Drowsiness
. (acidosis) Lethargy
. Coma

6.80 (high H⁺)......Death
"Auntie Em, Auntie Em! ... There's no place like home! ... There's no place like home."
Buffering of Volatile Acid

**Cell:**  \[ C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_20 \]  
(Glucose)  

**Interstitial Fluid:**  
\[ CO_2 \]

**RBC:**  
\[ CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^- \]  
\[ H^+ + Hb \leftrightarrow HHb \]

**Plasma:**  
\[ \uparrow Cl^- \]

*Chloride shift or Hamburger phenomenon (See Egan p. 252) [or 265 in 10th ed]*

(Also, see Krebs cycle…. Cellular metabolism)
Transported in three basic forms:
- dissolved CO$_2$ - plasma (about 7%)
- with Hb to form carbamino-hemoglobin (in the RBC (about 12%)
- converted to HCO$_3^-$ (about 80%) by our old faithful formula (you should know this by heart) to transport to the lungs then process reverses and CO$_2$ is moves across A-C membrane to be exhaled
  (The H$^+$ released is buffered by combining with Hb)
- see Fig. 11-13 in Egan pg 265
Spinning the Henderson-Hasselbalch Equation

$$\text{Base} = \frac{\text{Kidney}}{\text{Acid}} = \frac{\text{HCO}_3^-}{\text{Lung}} = \frac{\text{Combined CO}_2}{\text{Dissolved CO}_2}$$

\[
\begin{align*}
24 \text{ mEq/L} &= 24 \text{ mEq/L} \\
40 \text{ torr } x \ 0.03 &= 1.2 \text{ mEq/L} \\
20 &= 1
\end{align*}
\]
Metabolic Parameter

\[ \text{HCO}_3^- \text{ - bicarbonate ion} \]
(normal range 22 to 26, absolute 24 mEq/L)

\[ \text{HCO}_3^- < 22 \text{ mEq/L} \text{ - Metabolic acidosis} \]

\[ \text{HCO}_3^- > 26 \text{ mEq/L} \text{ - Metabolic alkalosis} \]
Causes of Metabolic Alkalosis (high HCO$_3^-$)

- Upper GI problems (vomiting removes hydrochloric acid)
- Excessive ingestion of licorice, sodium bicarbonate
- KIDNEY excretion or reabsorption
  - Diuretic treatment (causes severe K$^+$ depletion)
  - Cushing’s disease (pituitary tumor – causes body to increase steroid production)
  - Administration of corticosteroids (steroids)
  - Hyeraldosteronism
Causes of Metabolic Acidosis (low HCO$_3^-$)

- Ketoacidosis (diabetic, starvation, alcoholic)
- Poisonings (salicylate [children], ethylene glycol, methyl alcohol, paraldehyde)
- Lactic acidosis (heavy exercise, heart failure, sepsis, shock $\rightarrow$ anaerobic metabolism) liver damage
- Renal failure
- Lower GI problems: Loss of bicarbonate due to diarrhea
Kidneys problems cause metabolic alkalosis as the body tries to keep ionic charges in balance

- Normal function
  - $\text{Na}^+ \text{ & Cl}^- $ pass from the blood into the glomerulus
  - $\text{Na}^+$ must be recovered in the renal tubule cells
    - 80% is recovered with an anion (usually $\text{Cl}^-$)
    - 20% exchange for cation (usually $\text{H}^+$ or $\text{K}^+$) which is then excreted in urine

- Kidney disease
  - If $\text{Cl}^-$ decreases, the body must $\uparrow$ excretion of $\text{H}^+$ & $\text{K}^+$. This results in a hypochloremic alkalosis
  - If $\text{K}^+$ decreases, the body must $\uparrow$ $\text{H}^+$ excretion. This results in a hypokalemic alkalosis
Review: Normal ranges for blood gas values

- **Arterial**
  - pH .......7.35 to 7.45
  - PaO₂ ....80 to100 mm Hg
  - PaCO₂ 35 to45 mm Hg
  - HCO₃⁻ ...22 to 26 mEq/l
  - SaO₂.....92 to 100%
  - BE.........-2 to +2 mEq/L

- **Venous**
  - pH .......7.33 to 7.43
  - PvO₂ .....35 to 45 mm Hg
  - PvCO₂ 35 to45 mm Hg
  - HCO₃⁻ ...24 to 28 mEq/l
  - SvO₂.....70 to 75%
  - BE.........0 to +4 mEq/L
Combined or Mixed problems

- **Combined acidosis:** pH is low and both $\text{HCO}_3^-$ and $\text{PaCO}_2$ are moving into acid status together (low $\text{HCO}_3^-$ and high $\text{PaCO}_2$)
  
  Example: $\text{pH} = 7.10$, $\text{PaCO}_2 = 50$, $\text{HCO}_3^- = 17$

- **Combined alkalosis:** pH high, high $\text{HCO}_3^-$ and low $\text{PaCO}_2$
  
  Example: $\text{pH} = 7.55$, $\text{PaCO}_2 = 31$, $\text{HCO}_3^- = 29$

- What about compensation?
Rules of Thumb

- $\text{HCO}_3^-$ will $\uparrow$ 1 mEq/L for every 10 mm Hg $\uparrow$ in $\text{PaCO}_2$ for *acute respiratory acidosis*
- $\text{HCO}_3^-$ will $\uparrow$ 5 mEq/L for every 10 mm Hg $\uparrow$ in $\text{PaCO}_2$ for *chronic respiratory acidosis*
- $\text{HCO}_3^-$ will $\downarrow$ 1 mEq/L for every 5 mm Hg $\downarrow$ in $\text{PaCO}_2$ for *acute respiratory alkalosis*
- Kidneys will never fully compensate a respiratory acidosis (pH will stay a little acidic)
But what about this?

- The patient: 55 yr old smoker for 40 yrs ... 2 ppd.... 80 pack years history
- 5’10”  152 lbs (What does this guy look like?)
- ABG
  - pH           7.42
  - PaCO$_2$     55 torr
  - HCO$_3^-$    33 mEq/L
- Interpretation?
Hey man…. It’s not my job…..
Practice time

- Do worksheet for acid-base balance – 8 sample blood gases to interpret for acid-base and oxygenation (in your handout materials)

- Practice this - you must get ABG interpretation 100% right every time once you get into the hospital

- Practice, practice, practice
# Evaluation of Hypoxemia

<table>
<thead>
<tr>
<th>Hypoxemia Type</th>
<th>PaO$_2$ Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>80 - 100 torr</td>
</tr>
<tr>
<td>Mild hypoxemia</td>
<td>60 - 79 torr</td>
</tr>
<tr>
<td>Moderate hypoxemia</td>
<td>40 – 59 mm Hg</td>
</tr>
<tr>
<td>Severe hypoxemia</td>
<td>&lt;40 torr</td>
</tr>
</tbody>
</table>

(Note: ↑ in PaCO$_2$ will cause ↓ in PaO$_2$ based on the alveolar air equation)
Alveolar Air Equation

\[ \text{PAO}_2 = \text{FIO}_2 \times (\text{Pb} - \text{PH}_2\text{O}) - (\text{PaCO}_2 \times 1.25) \]

- **FIO\(_2\)** - fractional concentration of inspired oxygen %
- **Pb** - barometric pressure (normal 760 mmHg)
- **PH\(_2\text{O}\)** - water vapor pressure (normal 47 mmHg)

Note: \( \text{Pb} = \text{PO}_2 + \text{PCO}_2 + \text{PN}_2 \) and each gas is a fraction of the whole

- **1.25** - conversion factor (may also divide here by \(0.8\))
- Units of measure for all the pressures = mmHg

**or**

\[ \text{PAO}_2 = \text{FIO}_2 \times (\text{Pb} - \text{PH}_2\text{O}) - \text{PaCO}_2 \times (\text{FIO}_2 + [1-\text{FIO}_2]) / \text{R}) \]

See Chang’s *Resp Care Calculations* pg 14
Alveolar Air Equation

- What is the expected PAO\(_2\) for room air?
  - \[ \text{PAO}_2 = 0.21 \times (760 \text{ mm Hg} - 47 \text{ mm Hg}) - (40 \text{ mm Hg} \times 1.25) \]
  - \[ \text{PAO}_2 = 0.21 \times (713 \text{ mm Hg}) - 50 \text{ mm Hg} \]
  - \[ \text{PAO}_2 = 149.73 \text{ mm Hg} - 50 \text{ mm Hg} \]
  - \[ \text{PAO}_2 = 100 \text{ mm Hg} \]

- What if you are in Denver, CO…. what changes?
  - Hyperbaric chamber?
  - Henry’s Law of solubility of gases in liquids - See Egan pg 108

- A-a gradient \(\rightarrow\) \[ \text{PAO}_2 - \text{PaO}_2 \]
  - Normally < 10 mmHg at room air
  - < 65 mmHg on 100% oxygen

See Chang’s Resp Care Calculations pg 11
## Gas pressures at various altitudes

<table>
<thead>
<tr>
<th>Location</th>
<th>Alt. (ft)</th>
<th>$P_B$</th>
<th>FIO$_2$</th>
<th>PIO$_2$</th>
<th>PaCO$_2$</th>
<th>PAO$_2$</th>
<th>PaO$_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mobile, AL</td>
<td>0</td>
<td>760</td>
<td>.21</td>
<td>150</td>
<td>40</td>
<td>102</td>
<td>95</td>
</tr>
<tr>
<td>Cleveland, Oh</td>
<td>500</td>
<td>747</td>
<td>.21</td>
<td>147</td>
<td>40</td>
<td>99</td>
<td>92</td>
</tr>
<tr>
<td>Denver</td>
<td>5280</td>
<td>640</td>
<td>.21</td>
<td>125</td>
<td>34</td>
<td>84</td>
<td>77</td>
</tr>
<tr>
<td>Pike’s Peak</td>
<td>14,114</td>
<td>450</td>
<td>.21</td>
<td>85</td>
<td>30</td>
<td>62</td>
<td>55</td>
</tr>
<tr>
<td>Mt. Everest</td>
<td>29,028</td>
<td>253</td>
<td>.21</td>
<td>43</td>
<td>7.5</td>
<td>35</td>
<td>28</td>
</tr>
</tbody>
</table>

Note the A-a gradient
Hypoxia vs Hypoxemia

- Hypoxemia - low PaO$_2$ in the blood (hypoxemic)
  - We can measure this fairly easily

- Hypoxia - inadequate oxygen delivery to tissues (hypoxic)
  - We have a difficult time measuring this but we often see the outcome of hypoxia in declining function of systems
4 Types of Hypoxia

1. **Hypoxemic hypoxia** - low PaO$_2$ (blood) due to several possibilities:
   - low FIO$_2$ and/or low Pb
   - V/Q mismatch (normally .8 - see Egan and Wilkins)
   - shunt (perfusion without ventilation)
   - diffusion defect

**NOTE:** This type has good response to oxygen therapy except in cases of shunting
Hypoxia - 4 types

2. **Anemic hypoxia** - hemoglobin missing or malfunction
   - COHb - carbon monoxide poisoning
   - MetHb - methemoglobin (nitrite poisoning)
   - Anemia - low hemoglobin, bleeding, Fe deficiency
   - SCA - sickle cell anemia
Hypoxia - 4 types

3. Stagnant hypoxia - inadequate cardiac output
   - shock
   - regional vasoconstriction
   - excessive bleeding
Hypoxia - 4 types

- **4. Histotoxic hypoxia** - inability to utilize $O_2$ (cyanide poisoning)
  - Toxic = poisoned
  - Cyanide disrupts cellular utilization of oxygen (stops aerobic metabolism / Krebs cycle)
Correction of Hypoxemia

- Uncorrected Hypoxemia - PaO$_2$ below normal limits despite oxygen therapy
- Corrected Hypoxemia - PaO$_2$ in acceptable range with oxygen therapy
- Excessively Corrected Hypoxemia - PaO$_2$ above acceptable range with oxygen therapy, but patient would be hypoxemic without supplemental oxygen

Example - PaO$_2$ 125 mm Hg on 40% oxygen delivery, drops to 76 mm Hg on room air.
4 parts to assessing oxygenation

1. **PaO₂** (mm Hg) - partial pressure exerted by oxygen dissolved in blood

2. **SaO₂** - oxygen saturation of hemoglobin; the amount of oxygen combined with hemoglobin expressed as a percent of total oxygen carrying capacity:

   \[
   \text{SaO}_2 = \frac{\text{amount of } O_2 \text{ on Hb}}{\text{O}_2 \text{ carrying capacity of Hb}} \times 100
   \]
4 parts to assessing oxygenation

3. **CaO₂** (ml O₂/100 ml blood) - Oxygen content (units of measure called volume %)

4. **Circulation** - primarily C.O. and local perfusion
   You may have adequate oxygen content in the blood but due to inadequate circulation or blocked circulation, tissue hypoxia exists

- Oxygen delivery depends on adequate circulation
- \[ \text{DO₂} = \text{C.O.} \times \text{CaO₂} \times 10 \]
  Multiply by 10 to change the CaO₂ from vol % (deciliters to liters)
Oxygen Content \((\text{CaO}_2)\)

- Oxygen is carried in the blood two ways - combined with Hb and dissolved (reflected in \(\text{PaO}_2\))
- Each gram of Hb can carry a maximum of 1.34 ml of \(\text{O}_2\) if 100% saturated (Some texts use 1.39 ml)
- For each mm Hg in \(\text{PaO}_2\), 0.003 ml of oxygen will dissolve (per 100 ml blood)
- \(\text{CaO}_2\) is calculated from the \(\text{PaO}_2\), the \(\text{SaO}_2\), and the Hb
Oxygen Content \((\text{CaO}_2)\)

- \(\text{CaO}_2 = 1.34 \times \text{SaO}_2 \times \text{Hb} + 0.003 \times \text{PaO}_2\)

- Know this formula

- Know that the units of measure are: 
  
  \(\text{ml } \text{O}_2/100\text{ml blood} \) (called “volumes percent”)

Read Ch 11 “Gas Exchange” in Egan
“Normal” PaO$_2$ changes with age

- Up to 60 years old; normal > 80 mmHg
  - Normal drops by 1 mmHg for each year above 60
- 70 years old; normal > 70 mmHg
- 80 years old; normal > 60 mmHg
- 90 years old; normal > 50 mmHg

Why? - Normal aging process causes alveolar degeneration and decreasing lung function (“natural” emphysema)
Read Egan Chapter 11

“Gas Exchange”
Calculation of CaO$_2$

- Given the following data:
  - PaO$_2$ = 90 torr
  - SaO$_2$ = .97
  - Hb = 15 g/100 ml blood (gram %)

- What is the CaO$_2$?

(Note: If all you have is a Hct, Hb can be estimated by Hb = 1/3 Hct)
Calculation of CaO$_2$

CaO$_2$ = O$_2$ combined with Hb + O$_2$ dissolved
= 1.34 x Sat x Hb + 0.003 x PaO$_2$
= 1.34 x .97 x 15 + 0.003 x 90
= 19.5 ml/100 ml + 0.3 ml/100 ml
= 19.8 ml/100 ml

Note: O$_2$ dissolved in plasma accounts for only 1.5% of total O$_2$
Practice time

- Do worksheet on CaO$_2$ and PAO$_2$

- Normal CaO$_2$ is about 18 to 21 vol%
Respiratory Jeopardy

- $\text{CaO}_2 = \text{arterial oxygen content}$
- What is $\text{CvO}_2$?
- What would the formula be for $\text{CvO}_2$?
- What about $\text{C(a-v)O}_2$?
- How does the body handle a very low hemoglobin? What could fix it (or improve the situation)? What can we do?
- What is $V/Q$ mismatch?

See Chang’s Resp Care Calculations pg 20
Oxyhemoglobin Dissociation Curve
Significance of $P_{50}$

- $P_{50}$ is the point on the oxyhemoglobin dissociation curve where the hemoglobin is 50% saturated with oxygen molecules. $Pa \, O_2$ is the measured value.
  - Normal $P_{50}$ is 27 mmHg
  - $P_{50} > 27$ mm Hg: means the curve has shifted to right
  - $P_{50} < 27$ mm Hg: curve has shifted to the left
Significance of $P_{50}$

- Shift to left: greater affinity of Hb for O$_2$ (naturally found in the area of the alveoli)
- Shift to right: less affinity of Hb for O$_2$, but O$_2$ is more easily released to tissues (naturally found at the tissue level)
  - Some blood gas machines will give you the $P_{50}$ in the results
Oxyhemoglobin, % saturation

$P_{O_2}$, mm Hg

Left shift
- Decreased temp
- Decreased 2-3 DPG
- Decreased $[H+]$
- CO

Right shift
- (reduced affinity)
- Increased temp
- Increased 2-3 DPG
- Increased $[H+]$
Causes of Oxy-Hb Curve Shift to Left

- Alkalemia \( \downarrow [H^+] \) (\( \uparrow \) pH)
- Hypothermia \( \downarrow \) temperature
- Hypocarbia \( \downarrow \) CO\(_2\)
- \( \downarrow \) 2,3 diphosphoglycerate \( \downarrow \) 2,3 DPG

(Remember - shift left gives greater affinity - occurs naturally in the lung . Why?)
Causes of Oxy-Hb Curve Shift to Right

- Acidemia $\uparrow [H^+]$ ( $\downarrow$ pH)
- Hyperthermia $\uparrow$ temp
- Hypercarbia $\uparrow$ CO$_2$
- $\uparrow$ 2,3 DPG $\uparrow$ 2,3 DPG

Right shift means less affinity. Naturally occurs at the tissue level [WHY?] but may also be due to a problem.
Good Sputum Bowl Questions:

- Bohr effect – refers to the phenomenon that adding CO$_2$ to the blood enhances the release of O$_2$ from hemoglobin Hb affinity? Curve shifts which way? Occurs where?

- Haldane effect – refers to the phenomenon that adding O$_2$ to the blood enhances the release of CO$_2$ from the hemoglobin
  
  - (See Egan 265 in 10$^{th}$]

- Remember the Hamburger phenomenon?
Co-oximeter measurements

- Uses spectrophotometry to measure and the percentage of hemoglobin, and the percent of saturation of oxyhemoglobin, carboxyhemoglobin, and methemoglobin from a blood sample.

- Spectrophotometry uses light absorption patterns to identify and measure Hb, HbO$_2$, COHb, and metHb
CO-oximeter vs pulse oximeter measurements

- SaO₂ is from the co-ox, SpO₂ is from the pulse ox.
- Pulse ox reads %HbO₂ (referred to as SpO₂) only and can be fooled by abnormalities such as COHb (falsely high SpO₂) and MetHb (falsely low SpO₂), dark nail polish (falsely high SpO₂) movement, etc.
- Most pulse oximeters don’t read anything more than %HbO₂ (SpO₂)
ABG machines will give a calculated \( \text{SaO}_2 \) that is based on the pH and \( \text{PaO}_2 \). Problems such as abnormal hemoglobin (such as methemoglobin) and CO poisoning are not reflected in the calculated \( \text{SaO}_2 \) (nor in a pulse oximeter measurement - \( \text{SpO}_2 \)).

So.....Many hospitals use a co-oximeter with all ABG’s.
Link to a practice site for interpreting ABGs

- Select the primary problem, the compensatory mechanism, and the level of compensation

  [http://www.vectors.cx/med/apps/abg.cgi](http://www.vectors.cx/med/apps/abg.cgi)
First pants, THEN your shoes
pH (Sanz) Electrode

- Composed of two half cells, a reference and a sample side separated by a pH sensitive glass membrane. The reference side holds a solution that has a constant pH.
- A solution (blood) in the sample side causes a potential difference, or voltage, due to the H⁺ ion concentration from one side to the other.
- A voltmeter, calibrated in pH units, reads the voltage.

(Read Egan page 394 - see Fig 18-6)
pH (Sanz) Electrode

- The reference electrode is composed of either a silver/silver-chloride (Ag/Ag-Cl) or mercury/mercurous chloride (calomel) electrode.
- The sample electrode is a silver/silver-chloride (Ag/Ag-Cl) electrode.
- Potassium chloride (KCl) is used as a medium to provide a bridge between the 2 electrodes (this completes the electrical circuit).
- This is a *potentiometric* electrode (it measures voltage).
pH (Sanz) Electrode

- Maintenance is needed to clean off build-up of blood proteins (Using a bleach solution)
- Electrode temperature needs to be held at body temperature (37°C)
- The electrode should always be “wet” (saline rinse - not water)
- Must be checked daily for calibration and quality control on a shift-by-shift schedule
Measuring pH (Sanz electrode)
Sanz pH Electrode

**Fig 4-2.**—Schematic illustration of the modern, ultramicro pH electrode (see text).
**PO₂ (Clark) Electrode**

- Composed of 2 electrodes in a potassium chloride electrolyte solution: 1 - anode, 1 - cathode
- Silver anode oxidizes chloride ions to silver chloride and releases electrons, thus generating a current: \[4 \text{Ag} \rightarrow 4\text{Ag}^+ + 4e^-\]
- Platinum cathode reduces \(\text{O}_2\) to form \(\text{OH}^-\) ions which consumes the electrons
  - \[\text{O}_2 + 2\text{H}_2\text{O} + 4e^- \rightarrow 4\text{OH}^-\]
- This sets up a flow of current proportional to the amount of oxygen present in the sample
PO$_2$ (Clark) Electrode

- This is *polarographic* electrode (uses $\pm$ poles to measure the gas) and is also termed an *amperometric* measurement because it measures current (amps)

- As the platinum electrode becomes degraded, it must be polished periodically

- Must be checked daily for calibration and quality control on a shift-by-shift schedule
Measuring PO$_2$ (Clark electrode)
Clark PO$_2$ Electrode

**Chemistry and Physics**

PO$_2$ ELECTRODE:

- Platinum wire cathode
- Polypropylene membrane
- Silver anode
- Cathode tip
- PO$_2$ electrolyte

*Fig 4-6. — Schematic illustration of the Clark electrode (see text).*
PCO₂ (Severinghaus) electrode

- Uses a modified pH electrode to measure PCO₂ by the equation:
  \[ \text{H}_2\text{O} + \text{CO}_2 \leftrightarrow \text{H}_2\text{CO}_3 \leftrightarrow \text{H}^+ + \text{HCO}_3^- \]
- CO₂ from the blood crosses a permeable membrane and reacts with a HCO₃⁻ solution
- The resulting H⁺ produces a potential difference across the pH sensitive glass which is measured and converted to a measured PCO₂
- This is also a potentiometric electrode (it measures voltage)
Severinghaus PCO₂ Electrode

**Fig 4-4.**—Schematic illustration of the modern Pco₂ electrode (see text). The space between the silicon membrane and the nylon spacer is greatly enlarged for clarity.
Key points for ABG Electrodes

- **Sanz pH electrode** measures potential difference across a pH sensitive membrane (potentiometric)
- **Severinghaus PCO₂ electrode** allows CO₂ to react with HCO₃⁻ to produce H⁺ then measures the H⁺ (potentiometric)
- **Clark PO₂ electrode** measures O₂ by electron consumption (amperometric)
Calibration

- Checks the accuracy of the electrodes before analyzing blood samples
- pH calibration is done using 2 buffering solutions with known pH values (6.840 and 7.384)
- PCO$_2$ calibration is done using 2 gas mixtures with known CO$_2$ values (usually 5% and 10% CO$_2$)
- PO$_2$ calibration is also done with 2 gas mixtures with known O$_2$ values (usually 0% and 12 or 20%)
- Cal gas #1 - O$_2$ 0%, PCO$_2$ 5%, balance N$_2$
- Cal gas #2 - O$_2$ 12%, PCO$_2$ 10%, balance N$_2$
ABG Quality Control - QC

- 3 levels of controls are used for each electrode (acid, normal, and alkaline). Run all 3 controls each shift (usually Q8, sometimes Q12).
- Plotted by hand or computerized tracking for warning or out of control based on \( \pm 2 \) standard deviations.
- Regular maintenance is required to keep machines in calibration.
Quality Assurance (QA)  
Quality Control (QC)

- **QA** - Systematic process to monitor, document, and regulate the accuracy and reliability of the measurement
  - Errors can occur before, during, or after the blood analysis is done

- **QC** – periodic check on equipment performance to assure calibration, stability, and reliability
  - Uses statistical methods to evaluate accuracy and precision
Quality Assurance - QA

- Errors before analysis
  - Wrong patient
  - Wrong information (time drawn, settings, etc.)
- Errors after analysis
  - Problems with transmitted information – ie transposed numbers (7.14 given to clerk as 7.41)
- Errors during analysis
  - Improper maintenance, calibration checks, or quality control (QC)
ABG Quality Control

“Drift” and “out of control” situations require troubleshooting, maintenance, and/or electrode replacement (Read Ruppel pg. 309 - 314 for cal & QC)

Also – see Egan pg 397 Fig 18-9
Shewart/Levy-Jennings chart
Tonometry

- Gases are used as QC and for calibration for $O_2$ and $CO_2$ electrodes
- Comparing calibration with gases versus measuring $O_2$ and $CO_2$ in blood, the electrodes behaved differently
- To fix this, a tonometer is used to “bubble” the gases through a liquid for about 20 minutes to condition the gases (temperature and saturation) and reduce error.
  - Becoming an obsolete issue but....
  - Used to be the “Gold Standard”
pH – checked with buffering solutions

- Buffering solutions have a known pH for calibration checks
  - Aqueous buffers – no proteins, may behave differently than blood
  - Whole blood – infection control issues
  - Commercially prepared controls – eliminate tonometry. Precise for pH and CO\textsubscript{2} but not as good with O\textsubscript{2} (very temperature sensitive)
Circulatory status

Tissue oxygenation

cLactate(P)

Metabolic status

Oxygen uptake

- $pO_2$ 
- $FO_2$(I) ▼
- $F$Shunt ▲
- $pCO_2$ ▲
- Ambient pressure▼

Pulmonary disease
Cardiac right to left shunt
Low alveolar ventilation

Oxygen transport

- $ctHb$ ▼
- $ctO_2$▼
- $LO_2$ ▼
- $sO_2$▼
- $pO_2$ (see above)▼
- $p50$ (see above) ▲
- $FO_2Hb$▼
- $FCOHb$ ▲
- $FMethHb$▲

Smoke or gas poisoning
Toxic effects

Oxygen release

- $pH$ ▲
- $pCO_2$▼
- Temp▼
- c2,3-DPG▼
- $FCOHb$ ▲
- $FHbF$ ▲

Metaboloc alkalosis
Respiratory alkalosis
Hyperventilation
Hypophosphatemia
Smoke or gas poisoning
Neonates, hematological disorders

▲ higher than reference range
▼ lower than reference range
Systems overview for $O_2$ and $CO_2$ transport
RapidLab 1200 - USA C&W NICU and PICU
**STAT-1**  **THE POC Device**

**Only** POC system that:

- Places caregiver at Patient's side
- Ensures accurate results
  - Eliminates user-induced errors
- QC internal for caregiver
  - NO Wet QC to run
- Always ready for testing
- 2 drops, 2 minutes
  - Patient Care is initiated
- Labeling, transporting, and *waiting* is eliminated
- Meets CAP, CLIA, JCAHO
i-STAT1 Test Menu

- **Blood Gases:** pH, PCO$_2$, PO$_2$
- **Electrolytes:** Na, K, Cl, Ca
- **Chemistries:** Glu, creatinine, BUN
- **Hematocrit**
- **Calculated Values:** bicarbonate, TCO$_2$, HCO$_3^-$, base excess, sO$_2$, anion gap, hemoglobin
- **Coagulation:** ACTc, PT and ACT$_k$
- **Cardiac troponin levels** - cTnI
### i-STAT Cartridge combinations

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ABG’s give us a snapshot of ventilation, acid/base balance, and oxygenation. They are used every day by RCP’s, MD’s, RN’s and other professionals. Critical patients may have 4 - 8 ABG’s in a day.

ABG interpretation involves understanding many different systems and processes - so practice, question, study, and practice more
Summary

- Review Key Points in Egan (at end of ch 11, 13, and 18)

- Do Review Questions in Wilkins (end of ch 8)
References and supplies

REQUIRED:

- Egan 10th ed Fundamentals Ch 18
- White 5th ed Competencies Ch 8
- Wilkins 7th ed Assessment Ch 8
- Supplies: Handout, ABG arm, ABG kits
- Youtube.com….. Dalhousie Univ (8’18”) and Medical Education UK (6’14”)
  - Supplemental: Malley Blood Gases
Arterial Sampling - Hazards

- High pressure system - more potential for bleeding
- Clotting or severing the artery can cause loss of circulation distal to the puncture
- Infection hazards to patient and therapist
- Painful to patient - hyperventilation can alter baseline values
ABG sample sites

- Most common sites used in adults = radial, brachial, and femoral arteries
- Most common infant sites = radial and scalp (temporal) arteries
- Other sites include the axillary, ulnar, dorsal is pedis, and superficial temporal arteries (also the umbilical arteries in first 24 - 48 hours after birth)
- Axillary
- Ulnar
- Dorsalis pedis
- Umbilical artery

Diagram showing major arteries including:
- Carotid artery
- Pulmonary artery
- Brachial artery
- Ulnar artery
- Radial artery
- Femoral artery
- Tibial artery
- Dorsalis pedis artery
ABG Samples

- Blood gases may be drawn from a capillary stick (infants)
- Indwelling arterial line (which also gives continuous blood pressure monitoring capability)
- Pulmonary artery catheter (Swan-Ganz)
  - mixed venous sample (catheter also provides valuable hemodynamic measurements)
Supplies needed to obtain an ABG

- **Heparinized syringe**
  - Sodium heparin (liquid) can dilute and drop PaCO$_2$
  - Dry, crystalline (or lithium) heparine is preferred, must be mixed well by gently rolling or shaking the sample

- **Needle** (adults = 20 to 22 gauge, infants = 25 gauge, 5/8” to 1½ “ long)
Supplies needed to obtain an ABG

- Gloves
- Alcohol wipes
- Gauze
- Band aid or tape
- Needle disposal system,
- Label

Optional - syringe with 25 gauge needle and 1 mL of 0.5% lidocaine for local anesthetic
Procedure for obtaining the sample - radial artery

1. Gather supplies, check order, verify patient identity, wash hands, apply gloves

2. Explain procedure to ease patient anxiety and fear

3. Perform modified Allen’s test to check for collateral circulation (positive test = 10 - 15 second flush)
2. Using your fingers, apply occlusive pressure to both the ulnar and radial arteries.
4. Patient should be seated or lying down with arm extended. Rolled towel under wrist may help.
5. If auto-fill, set syringe for 1 to 3 ml (adult sample)
6. Palpitate artery with non-dominant hand, swab area thoroughly with alcohol wipe
6. Palpitate artery again, then using dominant hand to hold syringe, gently insert the needle through skin and into artery using a 45 degree angle to enter the artery. Be sure the bevel of the needle is up.
3. The syringe should be held at a 45° angle or less in your opposite hand, much like you would hold a pencil or a dart. This near-parallel insertion of the needle will minimize trauma to the artery and allow the smooth muscle fibers to seal the puncture hole after you withdraw the needle.
7. Look for **flash** of blood in the hub. Auto-fill syringes will self-fill. Otherwise, gently aspirate to obtain adequate sample size.

8. Once the syringe is full, remove needle from arm and quickly apply pressure using sterile gauze. Hold for 5 minutes. (anticoagulant therapy – hold longer)
9. Properly dispose of needle. Cap syringe, roll to mix heparin, label the sample, *(place in ice slurry)* – no longer recommended.

10. Verify no oozing from the site, cover with gauze and tape or band aid.

11. Clean up waste etc. Transport sample to blood gas measuring area.

Problems to avoid - air bubbles

- Air bubbles in the sample can change the measurement - $PO_2$ may increase or decrease, $PCO_2$ may decrease (moves toward room air values). Why?

- What happens if the patient is receiving 100% $O_2$?

- Inspect for bubbles, tap syringe to move them to the top (needle end) Eject the air and cap syringe tightly
Problems to avoid - delay in running the ABG

- Delays allow the blood cells to continue metabolism: \( O_2 \) is used up, \( CO_2 \) and acids are generated. pH, \( PaO_2 \), and \( PaCO_2 \) will be altered. Measured values will look worse than reality.
  - Old thinking: Put in ice slurry to slow metabolism. See White, *Basic Clinical Lab Competencies*, pg 144 (old thinking)

- Now – using a plastic syringe: keep at room temperature and run within 30 minutes. Otherwise (if delay is longer than 30 minutes) use glass syringe to obtain and put in ice slurry
Problems to avoid – arterial versus venous, or…..

- Venous blood samples should not be reported as arterial.
  - If possible, re-stick or else report results as venous.
- When the artery is entered, blood will cause flash and pulsation.
  - Venous sample will usually not flash and will ooze into syringe.
- Arterial samples are generally bright red, venous are dull to purple.
Other problems/issues

- Watch for (rare) vasovagal response during the stick (distress, anxiety, nausea, sense of impending doom).
  - If this occurs, stop the procedure, place patient head down, place cool wash cloth on forehead – report to M.D. and RN.

- Going through the artery (flash and stop)
  - Try drawing the needle straight back

- Changing the angle and re-inserting
Considerations

- Anticoagulant therapy delays normal clotting mechanisms and increases chances for hematoma formation.
- Three common drugs that interfere with normal coagulation: heparin, coumadin (warfarin), and aspirin.
- Patient will often know, so ask – “Are you on any “blood thinners like coumadin or warfarin?”
Temperature correction:
- Controversial but may be required, depending on the hospital.
- ABGs corrected for increased body temperature (hyperthermia) will increase results in PaO₂, PaCO₂, and will decrease the result for pH.

Reverse is true for decreased temperature (hypothermia)…
- correction will decrease PaO₂, PaCO₂, and increase pH
Samples drawn from an indwelling catheter

- No needles are used. System involves a 3-way stopcock and waste syringe or venous arterial blood management protection system (VAMP)
- Arterial line (A-line) usually placed in radial, femoral artery
- Umbilical artery catheter (UAC) for newborns
- Swan-Ganz or pulmonary artery (PA) catheter
Three-way stop cock
with luer-lock connection
Procedure for drawing from an A-line/stopcock

1. Gather supplies: waste syringe, heparanized ABG syringe, alcohol prep, gauze, clean cap for sample port, label, ice slurry. Check order and put on gloves.
2. Check for good waveform on monitor.
3. Stopcock should be off (closed) to sample port.
4. Remove sample port cap, wipe with alcohol prep, and connect waste syringe to sample port (this is a Luer-lock connection).
Sampling from A-line

5. Turn stopcock off (closed) to flush solution and draw waste into syringe until line is primed with fresh blood. (3 – 5 cc waste)

6. Turn stopcock $\frac{1}{2}$ closed between sample port and the flush solution.

7. Remove waste syringe and attach blood gas syringe to sample port.
Sampling from A-line

8. Turn stopcock off (closed) to the flush solution (open to sample port). Draw arterial sample into syringe.

9. Turn stopcock $\frac{1}{2}$ closed between sample port and flush solution. Remove blood gas sample syringe.
Sampling from A-line

10. Cover sample port with sterile gauze, turn stopcock off (closed) to patient and activate flush solution valve to flush the sample port into the gauze. Place clean cap on sample port after flushing.

11. Turn stopcock off (closed) to sample port. Activate flush solution valve to flush the line back to the patient. Check that the line is clear all the way to the insertion site at the skin.
12. Check for good waveform on the monitor. Remove any bubbles from blood gas syringe then cap the sample.

13. Roll to mix heparin, label the sample, (ice slurry – no longer done).

14. Clean up waste etc. - Properly dispose of waste syringe, gauze used for flushing. Transport sample to blood gas measuring area. Analyze within 30 minutes.

15. Document procedure and ABG values. Report any panic values to physician
Lab Assignment

- See White’s *Lab Competencies*
- Read all of Chapter 8 & perform the following:
  - Do self-test on page 173
  - Palpate radial and brachial artery on 2 classmates
  - Perform Allen’s test on 2 classmates
  - Practice technique for radial artery puncture on arm simulator
  - Practice arterial line stopcock manipulation for drawing a sample
http://www.youtube.com/watch?v=stxntv0KkB
E

http://www.youtube.com/watch?v=0Rr6vpFM
KPE