ABG Blood gases

An ABG is a test that measures the arterial oxygen tension (PaO_2), carbon dioxide tension ($PaCO_2$), and acidity (pH). In addition, arterial oxyhemoglobin saturation (SaO_2) can be determined.
Many blood gas analyzers will also report concentrations of lactate, hemoglobin, several electrolytes, oxyhemoglobin, carboxyhemoglobin and methemoglobin.

The **stronger and faster driver of pH is the respiratory center**:

Increase the respiration blow off the CO2 decreasing the H+ ion concentration increasing **pH (alkalosis**).

Decrease the respiration and the patient will **increase the amount of CO2 increasing the H+** concentration and decreasing the **pH (acidosis).**

The kidneys also come into to play by controlling the HCO3- in the system and they are slower and not as a strong driver. Increase the bicarbonate in the system and increase the pH(alkalosis) decrease the bicarbonate in the system and decrease the pH(acidosis).

The normal range for pH is 7.35–7.45

pH 7.35 - 7.45
Not a gas, but a measurement of acidity or alkalinity, based on the hydrogen (H+) ions present. The pH of a solution is equal to the negative log of the hydrogen ion concentration in that solution: pH = - log [H+].

PaO2 80 to 100 mm Hg.
The partial pressure of oxygen that is dissolved in arterial blood.
New Born – Acceptable range 40-70 mm Hg.

HCO3 22 to 26 mEq/liter
The calculated value of the amount of bicarbonate in the bloodstream. Not a blood gas but the anion of carbonic acid.

PaCO2 35-45 mm Hg
The amount of carbon dioxide dissolved in arterial blood. Measured. Partial pressure of arterial CO2. (Note: Large A= alveolor CO2). CO2 is called a “volatile acid” because it can combine reversibly with H2O to yield a strongly acidic H+ ion and a weak basic bicarbonate ion (HCO3 -) according to the following equation: CO2 + H2O <--- --> H+ + HCO3

Step by step analysis

Step One

Assessing pH Look at pH and determine if it is acidotic (<7.35), normal (7.35 - 7.45), or alkalotic (> 7.45).

Step Two

Determine respiratory involvement
Review the $PaCO_2$ to assess respiratory involvement. The lungs control the level of carbon dioxide in the arterial blood. The $PaCO_2$ must be evaluated in light of the arterial pH. That is, if the pH is abnormal, we then ask ourselves: would this observed $PaCO_2$, by itself, cause this pH abnormality?
$PaCO_2$:
Normal: 35 - 45 mmHg
Respiratory acidosis: > 45 mmHg
Respiratory alkalosis: <35 mmHg

Step Three

Determine metabolic involvement
Review the plasma [HCO_3^-] or B.E. (Base excess) to determine metabolic involvement (both controlled by non-respiratory factors.) Each of these components must be evaluated based on the current pH. If the pH is abnormal, we ask: would this observed [HCO_3^-] by itself, cause this pH abnormality?
HCO_3^-
Normal: 22 - 26 mEq/L
Metabolic acidosis: <22 mEq/L
Metabolic alkalosis: > 26 mEq/L

Step Four

Assess for compensation
Look at the pH, $PaCO_2$, and B.E. / HCO_3^- to decide whether compensatory mechanisms are at work.

Once the acid-base disorder is identified as respiratory or metabolic, we must look for the degree of compensation that may or may not be occurring. we know that the system not primarily responsible for the acid-base abnormality must assume the responsibility for returning the pH to the normal range. This compensation may be complete (pH is brought into the normal range) or partial (pH is still out of the normal range but is in the process of moving toward the normal range.) In pure respiratory acidosis (high $PaCO_2$, normal [HCO_3^-], and low pH) we would expect an eventual compensatory increase in plasma [HCO_3^-] that would work to restore the pH to normal.

Case 1

Mrs. Puffer is a 35-year-old single mother, just getting off the night shift. She reports to the ED in the early morning with shortness of breath. She has cyanosis of the lips. She has had a productive cough for 2 weeks. Her temperature is 102.2, blood pressure 110/76, heart rate 108, respirations 32, rapid and shallow. Breath sounds are diminished in both bases, with coarse rhonchi in the upper lobes. Chest X-ray indicates bilateral pneumonia.

- ABG results are:
  - pH= 7.44
  - $PaCO_2$= 28
  - HCO_3= 24
  - PaO_2= 54

Problems:

- $PaCO_2$ is low.
- pH is on the high side of normal, therefore **compensated respiratory alkalosis**.
- Also, PaO_2 is low, probably due to mucous displacing air in the alveoli affected by the pneumonia.

Solutions:

- Mrs. Puffer most likely has ARDS along with her pneumonia.
- The alkalosis need not be treated directly. Mrs. Puffer is hyperventilating to increase oxygenation, which is incidentally blowing off CO_2. Improve PaO_2 and a normal respiratory rate should normalize the pH.
- High FiO_2 can help, but if she has interstitial lung fluid, she may need intubation and PEEP, or a BiPAP to raise her PaO_2.
- Expect orders for antibiotics, and possibly steroidal anti-inflammatory agents.
- Chest physiotherapy and vigorous coughing or suctioning will help the patient clear her airways of excess mucous and increase the number of functioning alveoli.

Case 2

Mr. Worried is a 52-year-old widow. He is retired and living alone. He enters the ED complaining of shortness of breath and tingling in fingers. His breathing is shallow and rapid. He denies diabetes; blood sugar is normal. There are no EKG changes. He has no significant respiratory or cardiac history. He takes several antianxiety medications. He says he has had anxiety attacks before. While being worked up for chest pain an ABG is done:

- ABG results are:
  - pH= 7.48
  - PaCO2= 28
  - HCO3= 22
  - PaO2= 85

Problem:

- pH is high,
- PaCO2 is low
- **Respiratory alkalosis.**

Solution:

- If he is hyperventilating from an anxiety attack, the simplest solution is to have him breathe into a paper bag. He will rebreathe some exhaled CO2.This will increase PaCO2 and trigger his normal respiratory drive to take over breathing control.
- * Please note this will **not work** on a person with chronic CO2 retention, such as a COPD patient. These people develop a hypoxic drive, and do not respond to CO2 changes.

Case 3

You are the critical care nurse about to receive Mr. Sweet, a 24-year-old DKA (diabetic ketoacidosis) patient from the ED. The medical diagnosis tells you to expect acidosis. In report you learn that his blood glucose on arrival was 780. He has been started on an insulin drip and has received one amp of bicarb. You will be doing finger stick blood sugars every hour.

- ABG results are:
  - pH= 7.33
  - PaCO2= 25
  - HCO3=12
  - PaO2= 89

Problem:

- The pH is acidotic,
- PaCO2 is 25 (low) which should create alkalosis.
- This is a **respiratory compensation** for the **metabolic acidosis**.
- The underlying problem is, of course, a **metabolic acidosis**.

Solution:

- Insulin, so the body can use the sugar in the blood and stop making ketones, which are an acidic by-product of protein metabolism.
- In the meantime, pH should be maintained near normal so that oxygenation is not compromised

Example Arterial Blood Gas Problems
Normal Values
oxygen 80-100 (on room air)
pH 7.35-7.45 (the lower the number the more acidic)
carbon dioxide 35-45 (elevated=respiratory acidosis; decreased=respiratory alkalosis)
bicarbonate 22-26 (elevated metabolic alkalosis; decreased metabolic acidosis)

1. **Evaluate each parameter and determine acidosis or alkalosis**
2. **If the pH is normal and one of the other two is abnormal, the condition is compensate**
3. **Determine the primary problem**.

Patient situation	Arterial Blood Gases	Interpretation	
A patient with COPD who has come in for a routine exam.	O2 80 pH 7.45 (normal) CO2 50 (acidosis) HCO3 35 (alkalosis)	pH normal (more alkaline) CO2 acidosis HCO3 elevated (buffers the acid, thus makes more alkaline)	compensated metabolic alkalosis
A patient in the emergency is hyperventilating from anxiety over a traffic accident	O2 80 pH 7.50 CO2 30 HCO3 25	both the pH and the CO2 suggest alkalosis. The bicarbonate is normal.	uncompensated respiratory alkalosis
A patient who has vomited a large amount X3	O2 100 pH 7.50 CO2 40 HCO3 28	The patient has lost acid from the stomach and the pH goes up. The kidneys are attempting to compensate by retaining bicarbonate.	uncompensated metabolic alkalosis
A post surgical patient who has rapid, shallow breathing.	O2 76 pH 7.30 CO2 55 HCO3 22	both the pH and CO2 tell us it is respiratory acidosis.	uncompensated respiratory acidosis

Spirometry:

- Most common of the pulmonary function tests (PFTs), measuring lung function, specifically the amount (volume) and/or speed (flow) of air that can be inhaled and exhaled.
- The basic forced volume vital capacity (FVC) test varies slightly depending on the equipment used.
- Generally, the patient is asked to take the deepest breath they can, and then exhale into the sensor as hard as possible, for as long as possible, preferably at least 6 seconds.
- The maneuver is highly dependent on patient cooperation and effort, and is normally repeated at least three times to ensure reproducibility

Parameters of spirometry: Pulmonary function tests (PFTs)

- **Tidal volume (V_T).** This is the amount of air inhaled or exhaled during normal breathing. "***Tides what come in and what goes out"***
- **Minute volume (MV).** This is the total amount of air exhaled per minute.
- **Vital capacity (VC).** This is the total volume of air that can be exhaled after maximum inspiration. ***"In other words what is vital what works"***
- **Functional residual capacity (FRC).**amount of air remaining in lungs after normal expiration. ***"In other words how much trash is left behind"***
- **Total lung capacity.** This is the **total volume of lungs** when maximally inflated. (maximum volume of air present in the lungs) ***"How big are my lungs"***
- **Forced vital capacity (FVC).** This is the amount of air exhaled forcefully and quickly after maximum inspiration measured in liters. ***FVC is the most basic maneuver in spirometry tests.***
- **Forced expiratory volume in 1 second (FEV1)** FEV1 is the volume of air that can ***forcibly be blown out in one second, after full inspiration***
- **FEV1/FVC (FEV1%) In healthy adults 75–80%.** In obstructive diseases (asthma, COPD, chronic bronchitis, emphysema) FEV1 is diminished because of **increased airway resistance to expiratory flow**
- **Forced expiratory flow (FEF).** This is the average rate of flow during the middle half of the FVC test.
- **Peak expiratory flow rate (PEFR).** This is the maximum volume during forced expiration.

Chapman Junior PA Students 6-26-2018

interpretation of arterial blood gas lab results and calculations of alveolar-arterial oxygen gradient

A-a gradient = PAO_2 - PaO_2

in other words, does the oxygen you bring in match the oxygen on the inside

PaO_2 (partial pressure of O2 in the artery) --obtained from the arterial blood gases.
PAO_2 (partial pressure of O_2 in the alveoli)-- obtained from the Alveolar Gas equation.

This factor can vary according to atmospheric conditions and water pressure

Alveolar gas equation:

Factors affecting alveolar pO2

- The Alveolar Gas Equation

$$P_{AO_2} = (P_{atm} - 47)F_{IO_2} - \frac{P_{ACO_2}}{R}$$

Patm = atmospheric pressure, at sea level 760 mm Hg
F_{IO_2} = fractional concentration of oxygen, room air 0.21
P_{ACO_2} = alveolar pressure of carbon dioxide, normally 40 mm Hg

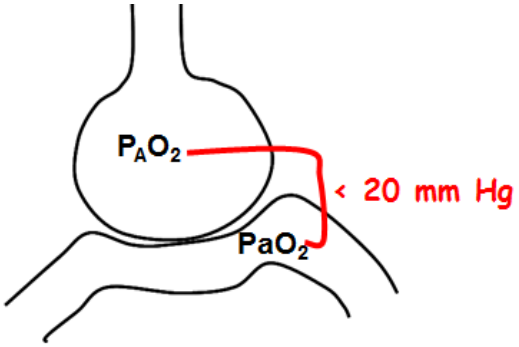
$$R = \text{respiratory exchange ratio} = \frac{CO_2 \text{ produced mL/min}}{O_2 \text{ consumed mL/min}}; \text{ normally } 0.8$$

Patm= Atmosphere Pressure
the higher up you are the less oxygen there is (Mt Everst)
the lower you go the more pressure to move oxygen in (Hyperbaric oxygen)
FiO2= Oxygen concentration (room air is 21%)

Estimating A-a gradient:

Normal A-a gradient = (Age+10) / 4

A-a increases 5 to 7 mmHg for every 10% increase in F_iO_2



Normal $P(A\text{-}a)O_2$ increases with age but is < 20 mm Hg in general

Interpretation: Hypoxemia causes differentiated by A-a Gradient

A. Increased A-a Gradient **(Something is in the way)**
   1. Right to Left Intrapulmonary Shunt (due to fluid filled alveoli)
      - **a. Congestive Heart Failure**
      - b. Adult Respiratory Distress Syndrome (ARDS)
      - c. Lobar Pneumonia
   2. V/Q Mismatch (due to lung dead space)
      - **a. Pulmonary Embolism**
      - b. Atelectasis
      - c. Pneumonia
      - d. Obstructive Lung Disease (e.g. Asthma, COPD)
      - e. Pneumothorax
   3. Alveolar hypoventilation
      - a. Interstitial Lung Disease

B. Normal A-a Gradient **(Nothing is in the way)**
   1. Hypoventilation
      - a. Neuromuscular disorders
      - b. Central nervous system disorder
   2. Low inspired FIO2 (e.g. high altitude)

A-a gradient (Alveolar to arterial gradient): Provides an assessment of alveolar-capillary gas exchange. To calculate you need the alveolar PO2 (PAO2) and arterial pO2 (paO2).
The larger the gradient, the more serious the respiratory compromise.
Indications for ventilatory support:
(1) tachypnea: >35-40/minute.
(2) vital capacity (nml: 65-75). if < 15.
(3) hypoxia. PO2 < 60.
(4) hypercarbia: CO2 > 55 (nml: 35-45).

The buffer systems

The lungs, kidneys, and the buffer system are the primary considerations in the homeostatic process. The lungs can control certain small amounts of carbon dioxide in the blood.

- **Increase respiratory rate blow off CO2 make the situation alkalotic**
- **Decrease respiratory rate retain CO2 make the situation more acidic**

ABG	pH	$PaCO_2$	HCO_3
Respiratory Acidosis	↓	↑	normal
Respiratory Alkalosis	↑	↓	normal
Metabolic Acidosis	↓	normal	↓
Metabolic Alkalosis	↑	normal	↑

- **Kidneys can control HCO3** which is used up to neutralized an acidic environment so the HCO3 is low and has been used up in an acidic environment in the metabolic world (i.e. DKA)
- While **retaining HCO3** would make the situation **alkalotic**

ABG	pH	$PaCO_2$	HCO_3
Respiratory Acidosis	↓	↑	normal
Respiratory Alkalosis	↑	↓	normal
Metabolic Acidosis	↓	normal	↓
Metabolic Alkalosis	↑	normal	↑

-hypoxemia: discuss the 4 principal mechanisms that lead to hypoxemia

The 5 causes of hypoxemia are categorized as

1. hypoventilation (central nervous system depression, obesity hypoventilation)
2. ventilation- perfusion mismatch (eg, obstructive lung disease, interstitial disease)
3. right-to-left shunt (eg, anatomic shunts, physiologic shunts such as atelectasis or pneumonia)
4. diffusion impairment (eg, pulmonary fibrosis, exercise induced)
5. reduced fraction of inspired oxygen (eg, high altitudes).

Common causes of hypercapnic respiratory failure

- include chronic obstructive pulmonary disorder (COPD),
- severe asthma
- neuromuscular diseases such as ALS, myasthenia gravis or traumatic brain injury
- situations of decreased respiratory-motor drive such as CNS infections or malignancy.

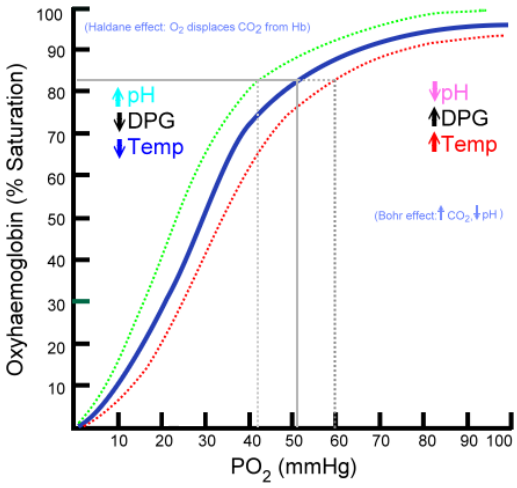
3 major mechanisms of hypoventilation

1. Failure of the CNS (drugs, narcotics, CVA)
2. Failure of the chest wall to expand such as chest wall diseases
   Kyphosis Scoliosis ALS, Diapharm weakness Obesity
3. Obstructions of the airways(asthma COPD)

Oxyhemoglobin dissociation curve

How many 02 passengers can I get on the hemoglobin bus

In its most simple form, the oxyhemoglobin dissociation curve describes the relation between the partial pressure of oxygen (x axis) and the oxygen saturation (y axis).



Physiologic dead space is the sum of anatomic and alveolar dead space.

1. **Anatomic dead space** is the volume of lung that does not exchange gas. This includes the nose, pharynx, trachea, and bronchi. This is about 2 ml/kg in the spontaneously breathing individual and is the majority of physiologic dead space. Endotracheal intubation will decrease the total anatomic dead space.

2. **Alveolar dead space** is the volume of gas that reaches the alveoli but does not take part in gas exchange because the alveoli are not perfused. In healthy patients alveolar dead space is negligible.

- **Hypercapnia** (increase CO2) also known as **hypercarbia** and **CO_2 retention**

- **Hypocapnia (decrease CO2)** usually results from deep or rapid breathing, known as hyperventilation.

PEEP

- PEEP: positive end-expiratory pressure
- A method of ventilation in which airway pressure is maintained above atmospheric pressure at the end of exhalation
- The purpose of PEEP is to increase the volume of gas remaining in the lungs at the end of expiration in order to decrease the shunting of blood through the lungs and improve gas exchange.
- PEEP is done in **ARDS (acute respiratory failure syndrome)** to allow reduction in the level of oxygen being given.