# ABG...EASY AS...1,2,3

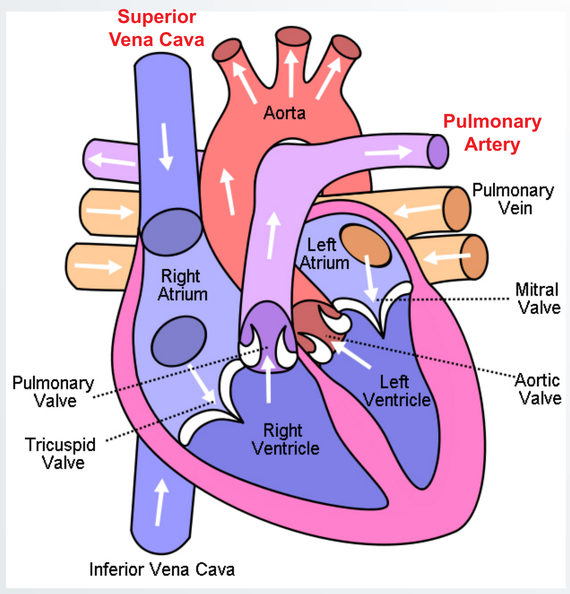
## NORMAL VALUES & DEFINITIONS

| NAME | DEFINITION | VALUE |
|---|---|---|
| pH | Refers to hydrogen ion (H+) levels, hence the 'H' in pH. H+ levels are important because a lack of (deficit) or too much (excess) will tell you if the patient is acidotic or alkolotic. One confusing point about pH is that it is an INVERSE ratio, which means that the more H+ present, the lower the pH and vice versa. | 7.35 - 7.45 |
| Acid | Can give away a H+ or can separate (dissociate) hydrogen from its ion, so the hydrogen is not positive and therefore no longer an acid.<br>Acids are end products of metabolism and must be buffered or excreted to achieve a normal pH | 20 parts base /<br>1 part acid |
| Base | Unlike Acids, bases can accept a H+ and bond with hydrogen. They are all negative and like to 'buffer' body acids. | |
| Base excess / Base deficit | Represents an increase or decrease in the amount of base compared with the amount of acids present | -2 to +2mmol/L |
| $HCO3^-$ | Concentration of hydrogen carbonate in blood. Used to determine along with pH and $CO_2$ source of acid base imbalance. | 22-26mmol/L |
| *p*$CO_2$ | Carbon dioxide partial pressure (tension). Reflects alveolar ventilation as it diffuses across the alveolar capillary membrane and "blown off". | 35-45 mmhg |
| *p*$aO_2$ | Arterial oxygen tension. In other words how well the lungs are able to pick up oxygen, i.e. supply, but not demand (this is shown in a mixed venous gas, discussed later). | 75-100mmhg |
| Lactate (Lactic Acid) | When cells no longer have enough $O_2$ for 'normal' aerobic metabolism (cell hypoxia) Anaerobic metabolism takes over resulting in lactate production, leading to lactic acidosis | 0.5 - 2.0mmol/L |
| Hb (Haemoglobin) | Amount of haemoglobin in blood possibly capable of carrying oxygen. | 135 - 180g/L 7 |

## MIXED VENOUS BLOOD GAS VALUES

| NAME | VALUE |
|---|---|
| pH | 7.33 - 7.44 |
| $HCO3^-$ | 24-28mmol/L |
| *p*$CO_2$ | 41-57 mmhg |
| *p*$aO_2$ | 35-40mmhg |
| *s*$O_2$ | 70 - 75% |

Mixed venous gases measures oxygen left in the blood as it returns to the heart (right side) after it has been pumped around the body supplying cells with oxygen.
The body normally extracts 25% of available oxygen and leaves 75% in reserve in times of stress or illness.[4]




*The heart above shows where our subclavian central lines sits - in the superior vena cava, it is here we can take venous blood gases. Samples from the pulmonary artery are however, more accurate and can only be performed if the patient has a PA catheter.*

## 'OTHER' VALUES OFTEN OVERLOOKED

Anion gap - This value is used in metablic acidosis to find the cause. It reflects unmeasured anions (negatively charged ions) such as proteins, sulfates, phosphates etc. The equation to find the anion gap is (Na + K+) - (cl + [$HCO3^-$])
a high value can be caused by keto or lactic acidosis, or renal failure. Low values may indicate hyponatremia or decreased plasma protiens.
10-20mmol/L

OXYGEN STATUS
p50a - oxygen tension at 50% saturation on ODC. This is used to reflect affinity of Hb for oxygen.
25-29mmhg
*F*MetHb - This is the fraction of methaemoglobin. Think of methaemoglobin like haemoglobin, but we carry less than 1% of it in our blood. As it is unable to combine with oxygen and it also decreases the oxygen carrying capacity of blood. Exposure to certain drugs and chemicals can dangerously elevate levels.
0.2-0.6%
*F*COHb - Fraction of carboxyhaemoglobin. Much similar to FMetHb in its actions. Affinity of Hb for carbon monoxide is 200 times greater than that of oxygen and impairs oxygen transport and release (ODC shift to left, alkolosis). This level can be high in heavy smokers.
0.0-8.0%
*F*shunte - Relative physiological shunt. Basically the amount of venous (de-oxygenated) blood that did not receive oxygen whilst travelling through the lungs. This can be caused by atelectasis, a pulmonary embolism (PE), mucous plugs and pulmonary oedema, all of which reduces oxygen transport into the blood.
1.0-10%
*F*$O_2$Hb - fraction of oxyhaemoglobin. This measures just how much blood (Hb) is carrying oxygen compared to the total amount of Hb capable of carrying. Similar to your s$O_2$, however *F*COHb and *F*MetHb are not included as they are not capable of carrying oxygen.
94-98%

Hct - Hematocrit. This shows us how much red blood cells there are in a sample of blood - i.e. how watered down (or not) blood is. 36-44%7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***Author: Amy O'Donnell Date: 9th February, 2008***


## 3 STEPS TO ABG INTERPRETATION

1. What is the pH? Is it alkolotic (↑7.45) or acidotic (↓7.35)?

2. What's happening with the respiratory system ($CO_2$) and the metabolic systems ($HCO3^-$)?
- If the problem is in the **lungs (respiratory)** the $CO_2$ will be heading in the opposite direction of the pH.

For example: respiratory acidosis: The pH will be low - ↓pH 7.22 and the $CO_2$ will be high - ↑$CO_2$ 55mmhg
- If the problem is **metabolic** the HCO3 will head in the same direction as the pH

For example: metabolic alkalosis: The pH will be high - ↑7.55 and the $HCO3^-$ will also be high $HCO3^-$ ↑35mmol/L.
Note: an easy way to remember for a "M"etabolic problem, think "M" as in the pH will head in the sa"M"e direction as the $HCO3^-$. For respiratory the pH will head in the "O"pposite direction as the C"O"2.

3. Is there any (if any) compensation occurring?
- No compensation:

pH remains abnormal, and the 'other' value (where the problem isn't occurring, i.e. $CO_2$ or $HCO3^-$) will remain normal or has made no attempt to help normalise the pH.
For example: in uncompensated metabolic acidosis:
pH ↓7.23, $HCO3^-$ ↓ 15mmol/L, and the $CO_2$ will be normal at 40mmHg.
- Partial compensation

pH is still abnormal, and the 'other' value is abnormal in an attempt to help normalise the pH.
For example: in partially compensated respiratory alkolosis:
pH ↑7.62, $CO_2$ ↓27 and the $HCO3^-$ will be abnormal at ↓17mmol/L
- Full compensation

The pH is normal, as the 'other' value is abnormal and has been successful in normalising the pH.
For example: Fully compensated metabolic acidosis
pH 7.38, $HCO3^-$ ↓15mmol/L and the $CO_2$ ↓30mmHg [6]

## WHAT CAUSES THESE CHANGES?

METABOLIC ACIDOSIS
Can be caused by either an increase in circulating acids and or a loss of base ($HCO3^-$). These include:
- Renal failure (unable to excrete acids or H+)
- Lactic acidosis (increase in circulating acids)
- Keto - acidosis (increase in circulating acids)
- Diarrhoea ($HCO3^-$ loss)

METABOLIC ALKOLOSIS
Can be caused by an increase in $HCO3^-$ or loss of metabolic acids. These include:
- Prolonged vomiting (acid loss)
- GI suctioning (acid loss)
- Hypokalaemia (H+ (an acid) excreted to maintain electrolyte balance)

RESPIRATORY ACIDOSIS
Caused by increased $CO_2$ levels which is then converted to an acid (H+) as the body tries compensate by excreting acids via the kidneys. These include:
- Hypoventilation:
  - sedatives/sedation/opiates
- Depression of respiratory centre in brain stem via trauma
- Pneumonia
- Pulmonary oedema
- Asthma

RESPIRATORY ALKALOSIS
Caused by a hyperventilation, the body getting rid of too much $CO_2$, for example:
- Anxiety
- Hypoxaemia (caused by heart failure)

## TREATMENT GOALS/GUIDELINES

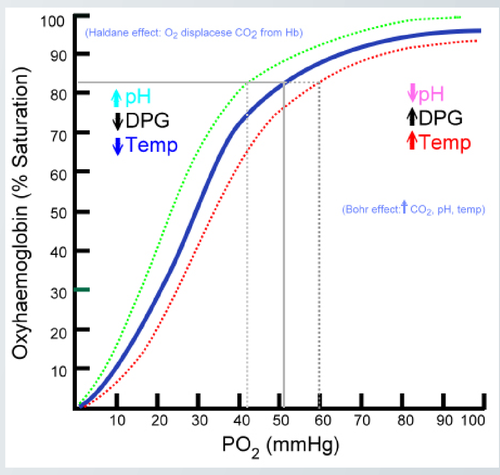
As with both acidosis and alkolosis, there needs to be a diagnosis of why the patients acid base status is such. Treatment is then aimed at stabilising the initial/presenting problem as well the acid-base status. However, general guidelines include correcting the fluid balance and electrolyte status. Continuous regular ABG monitoring and recording for trends. Possible dialysis to remove excess lactate, manipulation of ventilation settings to adjust $CO_2$ levels, but always maintaining tissue perfusion. Depending on severity of $HCO3^-$ levels and pH - possible sodium bicarbonate infusion (this is controversial however). Of course any of the adjustments made should only be done with an order from a medical officer, and ***must*** go through the intensive care team before any changes to treatments are applied. [4]

## OXYHAEMOGLOBIN DISSOCATION CURVE (ODC)

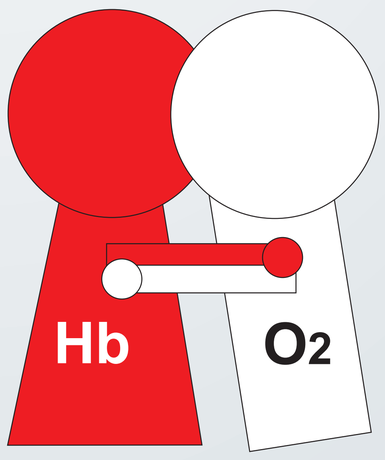
What is it and why is it important?
- The ODC looks at the relationship between oxygen tension (pressure) and oxygen saturation. It helps us better understand how our blood interacts with oxygen, i.e. how and why it picks up and lets oxygen go.
- The S shape tells us that after an amount of oxygen has accumulated in the blood, there isn't room for any more, no matter how much oxygen you throw at the haemoglobin molecule, it wont change
- Affinity basically means how much we (or blood) are attracted to someone (oxygen). The ODC shows us what changes the affinity of blood for oxygen.

RIGHT SHIFT (ACIDOSIS)
Shift of the curve to right decreases affinity - meaning that the Hb isn't very attracted to oxygen, and when it does pick up oxygen, it lets it go very quickly.
Increased temperature and $CO_2$ help this right shift.

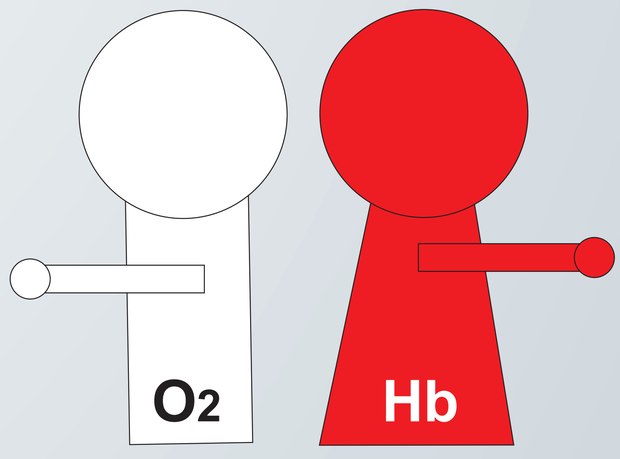
LEFT SHIFT (ALKALOSIS)
Shift of the curve to the left increases affinity - meaning that the Hb is very attracted (oh la la) to oxygen and when oxygen is picked up, the Hb has a hard time letting it go at the cellular level. Decreased temperature and $CO_2$ help the curve move to the left. [2]




## ALKALOSIS




## ACIDOSIS




**BICARBONATE/CARBONIC ($HCO3^-$/H2CO3) BUFFERING SYSTEM**
- This is the most active buffering system.
- Needs good renal function ($HCO3^-$ regulation)
- Converts H+ (hydrogen) to H2CO3 (carbonic acid) which is then able to be "blown off" via the lungs as $CO_2$.

$H_2O + CO_2 \leftrightarrow H2CO3 \leftrightarrow H+ + HCO3^-$
- The $H_2O$ and $CO_2$ is regulated via the lungs and $HCO3^-$ and H+ via the kidneys. An increase in one value can shift the equation and helps maintain equilibrium.
- A problem in either the lungs or kidneys or both can throw this equation out of whack, resulting in an acid base imbalance.
- Other buffers include protein and phosphate buffers.

**RESPIRATORY REGULATION OF ACID BASE BALANCE**
- Eliminates volatile acids ($CO_2$)
- Works within minutes
- Dependent on $HCO3^-$ stores and adequate gas exchange
- $CO_2$ diffuses passively into the cerebrospinal fluid and the medulla controls rate and depth of respiration

**RENAL REGULATION OF ACID BASE BALANCE**
- Eliminates fixed acids (harder to remove than volatile) H+
- Regulates $HCO3^-$ depending on need
- Controls phosphate and ammonia buffer systems
- Bicarbonate/carbonic acid system can only work if bicarbonate stores are replaced by the kidneys.[1]
- Works slowly, from hours to days