Cleveland Clinic Lerner College of Medicine of Case Western Reserve University

Frequently Asked Questions
Acid-Base Physiology

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[1] What are arterial blood gases?

ANSWER - Arterial blood gases (ABGs) are gases dissolved in arterial blood. Their magnitudes (tensions) are usually expressed in units of mm Hg. The usual gases of interest are oxygen and carbon dioxide. Most ABG reports also provide information about arterial blood pH and the arterial blood bicarbonate concentration, even though, technically speaking, they are not gases. (Of interest, most ABG reports do not provide information about dissolved nitrogen, even though is ordinarily the gas in highest concentration in blood.)

[2] Why are blood gas tensions expressed in mm Hg?

ANSWER - Blood gas tensions in America are expressed in mm Hg because of tradition, just like blood pressure measurements. There is a strong case to be made for eventually "going scientific" on this matter and adopting the SI system, but medical traditions die hard. Still, in Europe and most elsewhere in the world, the SI system is used to express blood gas tensions and blood pressure information in units of kilopascals (kPa).

A pressure of 1000 pascals (1 kPa) is 10.2 cm H2O or 7.75 mm Hg. Standard atmospheric pressure (760 mm Hg) is about 1000 cm H2O (1034 to be exact) or 100 kPa (101.9 to be exact). To convert pressure in mm Hg to kPa, simply divide the value in mm Hg by 7.5. Example: 760 mm Hg = 760 / 7.5 kPa = 10.13 kPa

For more information on unit conversions of all kinds, visit the Web at http://www.convert-me.com

[3] How are arterial blood gas samples obtained?

ANSWER- Arterial blood gas samples obtained by sticking a needle into an artery and aspirating a blood sample. Sometimes, instead of a needle, an indwelling catheter is placed into an artery so that repeated samples can be taken. Either way, this can really hurt, although use of a local anesthetic and a small bore needle (e.g., size 25) can help reduce the discomfort.

Left: Typical syringe and needle setup for arterial blood gas sampling. Inside the syringe can be found a small amount of heparin to prevent the blood from clotting. Usually 2 ml of blood is more than sufficient for analysis.
Image Credit: http://www.vital-signs.com/products/respiratory/abgs/gaslyte.jpg

Right: Intubated / ventilated patient in an intensive care unit (ICU) getting an arterial blood gas "stick" from the radial artery. Note the use of gloves by the operator. Also, note that most ICU patient will have an indwelling arterial line (catheter) placed to allow blood samples to be taken repeatedly simply by opening up a stopcock. This particular arrangement also allows for continuous (beat-by-beat) BP measurement displayed on a patient monitor.
Image Credit: http://www.uky.edu/LCC/RCP/mv-stick-13.jpg

[4] Can taking an arterial blood gas sample be harmful to the patient?

ANSWER- All invasive procedures (indeed, some noninvasive procedures too) have the potential to harm patients. Taking an ABG sample has the potential to cause direct arterial damage, to put the artery into spasm ("vasospasm") or to impair the circulation of tissues supplied by the artery being stabbed. Hematoma formation is particularly common, something especially important to think about in patients with a coagulopathy (impaired clotting system), such as patients on anti-coagulants like heparin or patients with poor platelet function. Also, remember that an expanding hematoma can even compress an artery to the extent that it no longer supplies blood to an extremity.

[5] What kinds of clinical information can one obtain from an arterial blood gas sample?

ANSWER- Three kinds of clinical information are usually made available from an arterial blood gas sample:

[i] Information about ***how well the patient is oxygenating***, as in the arterial oxygen tension and the arterial oxygen saturation. (Remember that arterial oxygen tension and arterial oxygen saturation are linked through the oxy-hemoglobin dissociation curve).

[ii] Information about the ***patient's acid-base status***, obtainable from any TWO of the three piece of information: [1] arterial pH (or, equivalently, arterial hydrogen ion concentration), [2] arterial carbon dioxide tension, and [3] arterial blood bicarbonate concentration. (Remember that given any two of these parameters, the third can be obtained using the Henderson-Hasselbalch equation.)

[iii] ***Serum electrolyte information.*** Most modern blood gas analyzers provide information on some or all of the following electrolytes: sodium concentration, potassium concentration, chloride concentration, magnesium concentration, chloride concentration.

Finally, some blood gas machines provide even more information than the above – such as information on carboxyhemoglobin and methhemoglobin levels.

[6] What is the relationship between pH and $[H^+]$?

ANSWER

$[H^+]$ *in nEq/L* = $10^{(9\text{-pH})}$

or

pH = 9 – $\log_{10}$ [H+]

Example: if $[H^+]$ = 100 nEq/L, then

pH = 9 – $\log_{10}$ [100]
= 9 – 2
= 7

Note: 1 nEq/L = 10^{-9} Eq/L; 1 mEq/L = 10^{-3} Eq/L

Table: pH and hydrogen ion concentration

pH	$[H^+]$ nEq/L
6.0	1000
7.0	100
8.0	10
9.0	1

[7] What are normal values for arterial blood gases?

ANSWER – For the acid-base side of things, the "normal values" are as follows:

pH = 7.35 to 7.45
P_{CO2} = 35 to 45 mm Hg
$[HCO_3^-]$ = 22 to 26 mEq/L

[8] What is the Henderson-Hasselbalch equation?

ANSWER - The hydrogen ion concentration $[H^+]$ in extracellular fluid is determined by the ratio between the partial pressure (tension) of carbon dioxide (P_{CO2}) and the bicarbonate concentration $[HCO_3^-]$. This relationship is known as the *Henderson-Hasselbalch equation*:

$[H^+]$ *in nEq/L* = $24 \times P_{CO2} / [HCO_3^-]$.

or

$[HCO_3^-] = 24 \times P_{CO2} / [H^+]$

or

$P_{CO2} = [HCO_3^-] \times [H^+] / 24$

This important relationship may also be stated in its classical but awkward form as:

$pH = 6.1 + \log_{10} ([HCO_3^-] / (0.03\ P_{CO2}))$

Example 1: Since a normal arterial P_{CO2} is 40 mm Hg and a normal $[HCO_3^-]$ is 24 mEq/L, the normal $[H^+]$ in arterial blood is $24 \times (40/24) = 40$ nEq/L.

Example 2: What is the P_{CO2} in a patient with a bicarbonate concentration of 10 mEq/L and a hydrogen ion concentration of 100 nEq/L (pH=7.0)?

ANSWER

$P_{CO2} = 10 \times 100 / 24 = 1000 / 24 = 42$ mm Hg

[9] What is the normal respiratory response to metabolic acidosis?

ANSWER - The physiological response to metabolic acidosis is hyperventilation, with a resulting compensatory drop in PCO2 according to **"Winter's formula":**

$$\text{Expected } P_{CO2} \text{ in metabolic acidosis} = 1.5 \times [HCO_3^-] + 8 \quad (\text{range: } +/- 2)$$

If the actual measured P_{CO2} is much greater than the expected P_{CO2} from Winter's formula, then the respiratory system is not fully compensating for the metabolic acidosis, and a respiratory acidosis is concurrently present. This may occur, for instance, when respiratory depressants like morphine or fentanyl are administered to the patient to reduce pain.

[10] What is the anion gap?

Anion Gap (plasma) =
plasma sodium – plasma chloride – plasma bicarbonate

This information is useful in the setting of metabolic acidosis. It is helpful to divide cases of metabolic acidosis into those of normal plasma anion gap and those with elevated plasma anion gap.

Anions and cations in any compartment must be equal (because of the electroneutrality principle). $HCO3^-$ is a measured anion in plasma, while unmeasured anions are protein, lactate, phosphate, and ketoanions. In metabolic acidosis, when $HCO3^-$ is replaced by Cl^-, a measured anion, the anion gap is normal. When $HCO3^-$ is replaced by an unmeasured anion (e.g., lactate), the anion gap is increased.

In general, normal values for the plasma anion gap are 4-12 mEq/L (varies by laboratory). (Note that, at least theoretically, the presence of unmeasured cations can falsely narrow the anion gap, making an otherwise widened gap appear normal. For example, if ammonium were somehow present in high amounts it might cover up an elevated anion gap metabolic acidosis.)

[11] What are the usual causes of an elevated anion gap metabolic acidosis?

The most common etiologies of a metabolic acidosis with an increased anion gap are shown below:

- Lactic acidosis (from poor perfusion)
- Renal failure
- Ketoacidosis (as in diabetic ketoacidosis)
- Starvation
- Ingestion of:
  - Ethylene glycol
  - Methanol
  - Salicylate

[12] What are the usual causes of a normal anion gap metabolic acidosis?

Normal anion gap metabolic acidosis, also termed "hyperchloremic" metabolic acidosis, results from systemic bicarbonate loss (with consequent concentration of the remaining chloride). A frequent cause in children is diarrhea. Other causes of normal anion gap metabolic acidosis include:

- Renal tubular acidosis (various types).
- Use of carbonic anhydrase inhibitor medications
- Plasma volume expansion with chloride-rich intravenous fluid (e.g., Normal Saline), resulting in bicarbonate dilution
- Exposure of ileal mucosa to urine (as in urinary diversion surgery).
- Administration of hydrochloric acid (HCl) or metabolic equivalents (ammonium chloride, arginine hydrochloride, etc.)

[13] What is the urine anion gap?

The urine anion gap is calculated as follows:

Urine Anion gap = urine sodium + urine potassium - urine chloride.

Urinary electrolytes studies can often be helpful in the setting of a normal anion gap metabolic acidosis. In this situation, if the urine anion gap is negative, gastrointestinal loss of bicarbonate (such as from diarrhea) is the likely cause of the metabolic acidosis. If the urine anion gap is positive, this suggests a renal cause (such as one of the various types of renal tubular acidosis).

NOTE: Some authorities give +/- 10 mEq/L as being a "normal range" for the urine anion gap. Thus, if the urine anion gap is more negative than -10 mEq/L, gastrointestinal bicarbonate loss is the likely cause of the metabolic acidosis, and if the urine anion gap is more than 10 mEq/L, this suggests a renal cause.

[14] What are the expected pH and bicarbonate for the various respiratory disturbances?

Acute respiratory acidosis:
Expected pH = 7.4 - 0.008 (PCO2 - 40)
Expected HCO3 = 24 + 0.1 (PCO2 - 40)
(The increase in PCO2 shifts the equilibrium between CO2 and HCO3 to increase HCO3. This simple physicochemical event occurs almost immediately.)

Chronic respiratory acidosis:
Expected pH = 7.4 - 0.003 (PCO2 - 40)
Expected HCO3 = 24 + 0.4 (PCO2 - 40)
(With chronic respiratory acidosis, the kidneys respond by retaining HCO3. This takes a few days to fully occur.)

Acute respiratory alkalosis:
Expected pH = 7.4 - 0.008 (PCO2 - 40)
Expected HCO3 = 24 + 0.2 (PCO2 - 40)
(The decrease in PCO2 shifts the equilibrium between CO2 and HCO3 to decrease HCO3. This occurs very quickly.)

Chronic respiratory alkalosis:
Expected pH = 7.4 - 0.003 (PCO2 - 40)
Expected HCO3 = 24 + 0.5 (PCO2 - 40)
(With chronic respiratory alkalosis, the kidneys respond by reducing HCO3. This takes a few days to fully occur.)

[15] What is an acid-base nomogram (acid-base map)?

ANSWER - An acid-base nomogram or acid-base map is a two-dimensional display that allows one to determine the acid-base status of the patient given any TWO of the following: [1] arterial pH (or, equivalently, arterial hydrogen ion concentration), [2] arterial carbon dioxide tension, and [3] arterial blood bicarbonate concentration. *(Remember that given any two of these parameters, the third can be obtained using the Henderson-Hasselbalch equation.)*