

Arterial Blood Gas Analysis for Emergency Medicine Registrars

- very helpful investigation in critical care
- you should be an expert
- you need a system, and must be thorough
- they are not really that hard!!

Part 1 - The Basics

Part 2 - Compensation

Part 3 - Metabolic acidosis

Part 4 - Metabolic alkalosis

Part 5 - A quick word on respiratory disorders

Part 6 - More complex analysis

An excellent ABG online tutorial:

<http://www.anaesthesiamcq.com/AcidBaseBook/ABindex.php>

Part 1 – The Basics

1. What is the pH?

- normal is 7.35 to 7.45
- acidemia is pH <7.35
- alkalemia is pH >7.45

Use the terms acidemia / alkalemia when referring to the pH being low / high. The terms acidosis / alkalosis refer to a process rather than the resultant pH. It is well accepted that venous pH is approx 0.04-0.05 lower than arterial pH (see articles). Subsequently, all values will be assumed to be arterial.

2. What is the pCO₂?

- Normal is 35-45mmHg
- Hypercapnoea is >45 mmHg and is reflective of hypoventilation
- Hypocapnoea is <35 mmHg and is reflective of hyperventilation

It is fairly well accepted in Emergency Medical circles that a venous CO₂ is 4-5mmHg higher than an arterial pCO₂ (see articles). For the purposes of this guide, all pCO₂ values will be assumed to be arterial measurements.

A raised pCO₂ implies respiratory acidosis, as CO₂ reacts with water to make carbonic acid.

Likewise, a low pCO₂ implies respiratory alkalosis.

When determining whether the pCO₂ is a primary process or the result of respiratory compensation, compare the pCO₂ with the pH:

In the context of acidemia;

High pCO₂ reflects primary respiratory acidosis

Low pCO₂ reflects compensation for a metabolic acidosis

In the context of alkalemia;

Low pCO₂ reflects primary respiratory alkalosis

High pCO₂ reflects compensation for a metabolic alkalosis

Thus, for most blood gases, you will derive a great deal of information on acid-base status from just pH and pCO₂.

For example:

A young man with,

pH 7.15
pCO₂ 15

can be determined to be acidemic, caused by a primary metabolic acidosis with respiratory compensation.

ie; - the pH is low, hence the acidemia
- the CO₂ is low implying respiratory alkalosis, hence the respiratory component here must in compensation for a metabolic acidosis

3. What is the metabolic state (HCO₃ or base excess)?

- either HCO₃ or base excess (BE) can be used here
- normal HCO₃ is 22-26, use 24 for calculations
- normal BE is -3.0 to +3.0

A metabolic acidosis will be reflected by a low HCO₃ or more negative BE (<-3.0).

A metabolic alkalosis will be reflected by a raised HCO₃ or more positive BE (>3.0).

NOTE

It is possible to have a mixed picture, where there is:

Acidemia due to co-existent respiratory acidosis and metabolic acidosis
(Here, the pCO₂ will be high and the HCO₃ will be low)

Alkalemia due to co-existent respiratory alkalosis and metabolic alkalosis
(Here, the pCO₂ will be low and the HCO₃ will be high)

Part 2 - Compensation

- the idea is to bring the serum pH back closer to normal

- the correct terminology is to reserve the terms "acidosis" and "alkalosis" for the primary process and use "compensation" for the secondary process

Respiratory compensation

- occurs in response to a metabolic derangement
- ventilation is altered -> the pCO₂ is altered -> the pH is altered

In general, the adequacy of respiratory compensation in the acute setting can be judged by the following rule:

- the pCO₂ should roughly equal the last two digits of the pH value, within a range of 10 – 60 mmHg
- More accurately:
 - in metabolic acidosis, $pCO_2 = 1.5 [HCO_3] + 8$
 - in metabolic alkalosis, $pCO_2 = 0.7 [HCO_3] + 20$

For example, let's take a patient named Jon, with a *primary metabolic acidosis*, where the pH is 7.15; the expected pCO₂ would be about 15 mmHg. Significant difference from this value would be relevant.

For example, if Jon was found to have a pCO₂ of 30 mmHg, we would say that he still had respiratory compensation, but that it was inadequate (or much less than expected). This would imply that Jon had some sort of pathology interfering with normal respiratory function, such as opiates.

- respiratory compensation can not return the pH to normal limits

Metabolic compensation

- occurs in response to a ventilatory derangement
- occurs quickly via intracellular buffering, and more slowly via the kidney, where under normal conditions, HCO₃ is absorbed and H is secreted in varying amounts

The following rules are said to apply to determine the adequacy of metabolic derangement:

1. In respiratory acidosis
 - i. Acutely (<24hrs) – raise HCO₃ of 1mEq/L for every rise in 10mmHg of pCO₂
 - ii. Chronic (>24hrs) – raise HCO₃ of up to 4mEq/L for every rise in 10mmHg of CO₂

2. In respiratory alkalosis

iii. Acutely – fall in HCO_3 of 2 mEq/L for every fall in 10 mmHg of CO_2

iv. Chronic – fall in HCO_3 of 5 mEq/L for every fall in 10mmHg of CO_2

- chronic respiratory alkalosis is the only acid-base disorder where compensation can return the pH to normal

Part 3 - Metabolic Acidosis

FIRST, some more concepts

Anion Gap (plasma)

- = measured cations – measured anions
- = $\text{Na} - (\text{Cl} + \text{HCO}_3)$
- = the size of the unmeasured anions
 - normal value less than 12

When you have a metabolic acidosis, you need to measure this value, as the result has important diagnostic implications

- NOTE - albumin, due to its large number of negative charges, is largely responsible for the anion gap in a normal patient
- hence, hypoalbuminemia will result in a falsely low anion gap
 - to correct for low albumin, use the formula:

Corrected AG = measured AG + 0.25 (42 - serum albumin)

Osmolar Gap

= measured serum osmolality - calculated serum osmolality

- usual determinants of plasma osmolality are:

Sodium
Urea
Glucose

Such that formula for calculated plasma osmo is :

$$1.86 * [\text{Na}] + [\text{urea}] + [\text{glucose}]$$

sometimes rounded up to :

$$2 * [\text{Na}] + [\text{urea}] + [\text{glucose}]$$

If another substance that has osmotic activity is present in the plasma (such as methanol), then the measured osmolality will be much higher than the calculated value.

NOTE: you will have to ask the lab specifically for a measured value if you want one; it is not a standard test

- normal osmolar gap is less than 10
- NOTE - ethanol will cause an elevated osmolar gap

Metabolic acidosis is classified by anion gap

High anion gap metabolic acidosis

= anion gap greater than 12

Causes:

- Lactic acidosis
- Ketoacidosis
- Renal failure
- Toxic alcohols
- Other drugs
- Pyroglutamic acidosis

1) Lactic acidosis

Type A - caused by tissue hypoperfusion and subsequent anaerobic metabolism
- by far the most common

Type B

- B1 - due to organ failure / disease - cardiac, hepatic, renal, leukaemia
- B2 - due to drugs, such as metformin
- B3 - due to inborn errors of metabolism

2) Ketoacidosis

Ketones are - acetate, acetoacetate and beta-hydroxybutyrate

- Diabetic
- Alcoholic
- Starvation

NOTE - ketostix only measure acetate and acetoacetate

- in alcoholic ketoacidosis, the predominant ketone is usually beta-hydroxybutyrate, hence this condition may be missed by a simple urine dipstick

3) Renal failure

Due to failure of secretion of inorganic acids, such as phosphates and sulfates

4) Toxic alcohols

Such as methanol, ethylene glycol and isopropyl alcohol

- here the osmolar gap should also be elevated significantly
- care needs to be taken when ethanol co-ingestion exists, as it too will raise the osmolar gap
- the osmolar gap will fall as the toxic alcohol is metabolized, hence timing of any measurement is relevant to interpretation

5) Other drugs

- most cause acidosis via raised lactate
- some are acids or have acidic metabolites, such as toluene (from glue sniffing)

6) Pyroglutamic acidosis

- pyroglutamic acid is a breakdown product of glutamate
- a very rare cause, resulting from glutathione depletion due to sepsis or chronic paracetamol ingestion and/or malnutrition (glutathione inhibits pyroglutamic acid production)
- may also occur with flucloxacillin or vigabatrin which inhibit pyroglutamic acid breakdown
- diagnosis is by measurement of urinary pyroglutamic acid

Normal anion gap metabolic acidosis

= anion gap less than 12

- due to either a failure of H^+ secretion or HCO_3^- reabsorption
- result in hyperchloremia (hence the often used term "hyperchloremic metabolic acidosis")
 - due to reabsorption of Cl^- to maintain electrical neutrality in the face of a low HCO_3^-

Causes:

Failure of H^+ secretion

Kidney - types 1 and 4 renal tubular acidosis

Failure of HCO_3^- reabsorption

Kidney - type 2 renal tubular acidosis

- GIT*
- diarrhea
 - pancreatic / biliary / small bowel fistulae
 - uretero-enteric diversion

The urinary anion gap can be used to differentiate between renal and GIT causes of normal anion gap metabolic acidosis, if history and examination are not diagnostic

Urinary anion gap in normal anion gap metabolic acidosis

$$= [\text{Na}]_{\text{urine}} + [\text{K}]_{\text{urine}} - [\text{Cl}]_{\text{urine}}$$

(there is minimal HCO_3 in urine which is usually acidic)

- is largely dependent on urinary NH_4^+
- if the kidneys work (ie the problem is with the gut), then NH_4^+ will be produced in large quantities during metabolic acidosis, and the result will be a NEGATIVE urinary anion gap

- ie in the presence of a large amount of NH_4^+ , urinary Na and K will be relatively low (compared with Cl) to maintain electrical neutrality

- in contrast, if there is kidney dysfunction, then NH_4^+ production will be impaired, and the urinary anion gap will be POSITIVE

- so;

Renal cause – positive urinary anion gap

GIT cause – negative urinary anion gap

Part 4 - Metabolic alkalosis

- due to:

- 1) loss of acid from either the GIT or kidney
- 2) gain of alkali from exogenous administration eg milk alkali syndrome

- by far the most common causes (>90%) are:

- vomiting, with loss of HCl from the stomach
- diuretic use with loss of acid from the kidney

- other causes include:

- hyperaldosteronism / Cushing's syndrome
- significant potassium depletion
- rarities, such as liquorice poisoning, Bartter's syndrome

Conditions associated with chloride loss, such as vomiting and diuretic use, are sometimes termed "chloride sensitive metabolic alkalosis", implying that the mainstay of treatment is normal saline infusion.

Part 5 – A quick word on respiratory disorders

Respiratory acidosis

- the primary process where $p\text{CO}_2$ is $>45\text{mmHg}$
- is the result of hypoventilation, from any cause
- some common causes include:
 - o drugs, such as opiates or benzos
 - o head injury
 - o COPD

Respiratory alkalosis

- the primary process where $p\text{CO}_2$ is $<35\text{mmHg}$
- is the result of hyperventilation, from any cause
- some common causes include:
 - o hypoxia
 - o drug induced, for example, salicylates
 - o anxiety

Part 6 – More complex analysis

Delta gap (also known as the gap gap)

Often measured as a ratio:

Change in anion gap : change in HCO₃

(Measured anion gap - 12) : (24 - measured HCO₃)

A useful measurement when you are confronted with a raised anion gap metabolic acidosis (only)

A pure raised anion gap metabolic acidosis should have a ratio of between 1 and 2

Lactic acidosis usually about 1.6

Diabetic ketoacidosis often less than this

Renal failure may be less than 1

If the ratio is less than 1, a concomitant normal anion gap metabolic acidosis may be present

If the ratio is greater than 2, a concomitant metabolic alkalosis may be present

NOTE - the delta gap is not completely reliable and judgement should be supported by other clinical findings

- thus, the delta gap is measured to determine whether there is more than one metabolic process occurring concurrently

Alveolar - arterial oxygen gradient

Determines whether a shunt exists either bypassing the lung as in some forms of congenital heart disease, or more commonly, from within the lung when there is an area of lung that is perfused but not ventilated (a V/Q mismatch).

Alveolar- arterial gradient = PA O₂ - PaO₂

Alveolar gas equation

A - alveolar, i - inspired, a - arterial

assume P H₂O = 47mmHg

R - co-efficient of respiration, usually given to be 0.8

$$PA O_2 = PiO_2 - PaCO_2/R$$

$$PiO_2 = FiO_2 (P_{atmos} - P H_2O)$$

At sea level and room air

$$= 0.21 (760 - 47)$$

$$= 150mmHg$$

Therefore at sea level on room air:

$$PA O_2 = 150\text{mmHg} - PaCO_2/0.8$$

Then, A-a gradient can be calculated using this figure and subtracting the measured arterial PO₂

- normal value for A-a gradient can be estimated by the formula:

$$\text{age}/4 + 4$$