Arterial Blood Gases: A Guide to Interpretation in Emergency Medicine

Fundamental Physiology Equations

1. Dissociation of a weak acid (HA):

   $HA \leftrightarrow H^+ + A^-$ Where A- is the resultant anion

2. The acid is buffered in the ECF mainly by the Bicarbonate system:

   $H^+ + HCO_3^- \leftrightarrow CO_2 + H_2O$ Where CO_2 is expired via ventilation

A. Compensation for Acid / Base Primary Pathology

Primary Pathophysiology	Primary Derangement	Compensatory Response	Secondary Change	Applicable Formula for Expected Secondary Change	Comments
Metabolic Acidosis	Decreased HCO_3	Hyperventilation	Decreased CO_2	**$pCO_2 = 1.5(HCO_3) + 8$ +/- 2**	Takes a few hours. pCO_2 will NOT fall below 10mmHg.
Metabolic Alkalosis	Increased HCO_3	Hypoventilation	Increased CO_2	**$pCO_2 = 0.7(HCO_3) + 20$ +/- 5**	pCO_2 will NOT exceed 55mmHg. Expect large variations in pCO_2.
Acute Respiratory Acidosis	Increased CO_2	Shift in equation 2 above to the LEFT	Increased HCO_3	**$HCO_3 = 24 + [(pCO_2 - 40) / 10]$**	That is, HCO_3 will increase by 1 mmol/L for every 10mmHg rise in pCO_2 above 40 mmHg. A simple physiochemical event that occurs almost immediately.
Chronic Respiratory Acidosis	Increased CO_2	Increased renal excretion of acid	Increased HCO_3	**$HCO_3 = 24 + 4 \times [(pCO_2 - 40) / 10]$**	That is, HCO_3 will increase by 4 mmol/L for every 10mmHg rise in pCO_2 above 40 mmHg. Renal compensation takes a few days.
Acute Respiratory Alkalosis	Decreased CO_2	Shift in equation 2 above to the RIGHT	Decreased HCO_3	**$HCO_3 = 24 - 2 \times [(40 - pCO_2) / 10]$**	That is, HCO_3 will decrease by 2 mmol/L for every 10mmHg drop in pCO_2 below 40 mmHg. HCO_3 does NOT fall below 18mmol/L.
Chronic Respiratory Alkalosis	Decreased CO_2	Decreased renal excretion of acid	Decreased HCO_3	**$HCO_3 = 24 - 5 \times [(40 - pCO_2) / 10]$ +/- 2**	That is, HCO_3 will decreased by 5 mmol/L for every 10mmHg drop in pCO_2 below 40 mmHg. Renal compensation takes a few days. HCO_3 does NOT fall below 12mmol/L.

All formulae assume that physiological mechanisms are functioning normally. As such, they should only be used as guides.

B. pH and Potassium

H+ and K+ ions compete for trans-membrane channels. Excess H+ ions (as in low pH states) compete for K+ ions for transport into cells. Therefore, low pH (ie high H+ ions) lead to high extracellular K+. Note that this hyperkalaemia in acidosisis NOT a spurious result. While total body K+ may be normal, the serum K+ is actually high. However, it is expected to reduce as soon as the pH is corrected. The "corrected" K+, per the formulae below, is therefore important, as it predicts the serum K+ when pH is corrected by treatment.

- Serum K rises by 0.5mmol/L for every 0.1 fall in pH from 7.4.
  - **In acidaemia: Predicted K+ = (Measured K+) – [(7.40 – measured pH) x 10 x 0.5]**

- Serum K drops by 0.5mmol/L for every 0.1 rise in pH from 7.4.
  - **In alkalaemia: Predicted K+ = (Measured K+) + [(Measured pH – 7.40) x 10 x 0.5]**

C. Serum Glucose and Measured Na+

Hyperglycaemia impairs the ability of laboratory analyzers to measure Na+ concentration, owing to its effect of osmolarity. High serum glucose leads to spuriously low levels of Na+.

- Serum Na spuriously drops by 1.0 mmol/L for every 3mmol/L rise in serum glucose from 5mmol/L.
  - **In hyperglycaemia: Corrected Na+ = (Measured Na+) + [(Measured glucose – 5) / 3]**

D. Anion Gap

This phenomenon applies to metabolic acidosis. It determines if the acidosis is from excess added acid to, or loss of HCO_3 from the body. Electrical neutrality is mandatory in body fluids. Total cation charge must equal total anion charge. The sum of measured cations is higher than the sum of measured anions, as several anions (eg PO_4) are not routinely measured. This quantum of unmeasured anions is called the anion gap.

- **Anion Gap = (Measured Na+) – (Measured Cl^- + Measured HCO_3)**
  Anion Gap should be 12 or less

A measured anion gap of greater than 12 indicates extra anions in the extracellular fluid. These extra anions are added acids to the body, such as lactate, ketones, salicylate, oxalate, formate, urea. This condition is called High Anion Gap Metabolic Acidosis (HAGMA).
A normal anion gap acidosis (NAGMA) indicates no added acids, implying that the acidosis results mainly from HCO_3 loss, through the renal system (renal tubular acidosis) or GI system (diarrhoea). As the serum Cl^- is often high in such scenarios, the condition is also called hyperchloraemic metabolic acidosis. Of course, a mixed metabolic acidosis can occur, leading to the utility of the Delta Ratio calculation (see below).

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E. Delta Ratio

Also known confusingly (and inappropriately!) as the Delta Gap, this applies principally in high anion gap metabolic acidosis. It helps determine if the extraneous acid balances with the resultant loss of HCO_3 (given its buffering effect) from the extracellular environment. The term "delta" describes "change". That is, it compares the change in extracellular acid to the change in extracellular HCO_3. It is a ratio of changes, or a ratio of deltas. Hence the term Delta Ratio.

- **Delta Ratio = (Amount of Acid Added) / (Amount of HCO_3 lost)**

Intuitively, this ratio should be 1.0. However, most physiological buffering is done by intracellular mechanisms such as the phosphate system, rather than the HCO_3 system. That is, the drop in HCO_3 does not match the rise in acid. Moreover, some added acids (being charged) do not cross trans-cellular membranes, leading to relatively greater concentrations of extracellular acid. Consequently, the Delta Ratio is typically greater than 1.0. In pure lactic acidosis, the Delta Ratio approximates 1.6. In pure DKA, the added acids (namely ketones) are excreted more in urine, leading a lower Delta Ratio of approximately 1.4. Proceeding with the calculation of Delta Ratio:

- **Delta Ratio = (Measured Anion Gap – Normal Anion Gap) / (Normal HCO_3 – Measured HCO_3)**

  = (Measured Anion Gap – 12) / (24 – Measured HCO_3)

Delta Ratio	Pathophysiological Processes	Explanation(s)
> 2.0	Acid added to system **HCO_3 reduction is less than expected**, *AND / OR* Pre-existent high HCO_3	Usual potential causes **Co-existent metabolic alkalosis,** *AND / OR* Chronic CO_2 retainer (chronic respiratory disease) with renal compensation
1.0 to 2.0	Acid added to system "Pure" HAGMA	Usual potential causes Lactic acidosis typically causes delta ratio of 1.6; lactic acidosis results from tissue hypoxia DKA leads to a smaller delta ratio (about 1.4), because of high urinary excretion of the added acid, *viz* ketone
< 1.0, > 0.4	Acid added to system **HCO_3 reduction is greater than expected**, owing to co-existent HCO_3 loss (renal, GI) **This is a mixed metabolic acidosis**	Usual potential causes, AND **Co-existent normal anion gap acidosis** Example: severe diarrhoea causing GI HCO_3 loss AND dehydration with consequent lactic acidosis
< 0.4	Probably no acid added to system; just HCO_3 loss	**Normal anion gap metabolic acidosis**

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F. A-a Gradient and the Alveolar Gas Equation

A-a gradient is used to determine the efficacy of oxygen exchange between the alveoli and the arteries, where "A" denotes the pressure of alveolar O_2 (pAO_2), and "a" the pressure of systemic arterial O_2 (paO_2). To calculate A-a gradient, the pAO_2 and paO_2 must be known. The latter is measured. The former is calculated using the Alveolar Gas Equation. The premise of this equation is that some inspired oxygen is exchanged (*ie* respired) with expired carbon dioxide as it travels through the airways into the alveoli. The ratio of this exchange is fixed (see below, on RQ). At the alveoli, the remaining oxygen, represented by pAO_2, is available for transfer to pulmonary capillaries.

- **The Alveolar Gas Equation states: $pAO_2 = PiO_2 - (pACO_2) / RQ$**

Where:

- Unit of measurement of all pressures are in mmHg
- PiO_2 equals partial pressure of inspired oxygen
- PiO_2 equals FiO_2 x (Atmospheric Pressure minus water vapour pressure)
  - Atmospheric pressure is 760 (at sea level)
  - Water vapour pressure is 47 (at sea level)
  - FiO_2 is fraction of inspired oxygen (in room air, it is 0.21)
- $pACO_2$ equals alveolar CO_2
- RQ equals Respiratory Quotient. RQ is the ratio of exchange between carbon dioxide and oxygen. It varies according to several parameters, but for practical purposes, is assumed to be a constant, at 0.8

Therefore, at sea level:

- **$pAO_2 = FiO_2 \times (760 - 47) - (pACO_2) / 0.8$**

- As diffusion of $pACO_2$ from alveoli to pulmonary veins is almost flawless, $pACO_2$ can rightly be assumed to equal $paCO_2$, which is obtained from ABG analysis.

The equation then becomes:

- **$pAO_2 = FiO_2 \times (760 - 47) - (paCO_2) / 0.8$**

Alveolar oxygen diffuses into the pulmonary capillaries. The oxygen travels thence to the pulmonary vein, right heart, left heart and systemic arteries. In physiological states, imperfections in oxygen diffusion, perfusion and addition of oxygen-deplete blood from bronchial veins into the pulmonary veins reduce the partial pressure of oxygen. In pathological states, deficits in exchange are exacerbated by impediments to diffusion (*eg* alveolar infiltration in pneumonia) and / or perfusion (*eg* shunt from pulmonary embolism).
By the time it reaches the systemic arteries, oxygen's partial pressure (paO_2) is therefore reduced by physiological factors. **Determining if pathological processes co-exist is aim of A-a gradient calculation**. In the absence of pathology, **estimate of normal A-a gradient is (age in years) / 4**. Of course, paO_2 is obtained from ABG analysis. The A-a gradient can finally be calculated:

- **A-a gradient = $pAO_2 - paO_2$**

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