Arterial Blood Gas Interpretation: The Basics

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Sampling of arterial blood for blood gas analysis will yield information on:

- Oxygenation of blood through gas exchange in the lungs.
- Carbon dioxide (CO_2) elimination through respiration.
- Acid-base balance or imbalance in extra-cellular fluid (ECF).

Oxygenation

- A normal healthy person at rest consumes oxygen (O_2) at a rate of approximately 4 ml/kg/min. This consumption can be increased 10 times in a moderately fit person and as much as 20 times in an Olympic class athlete during exercise.
- This oxygen is used in oxidative (aerobic) metabolism of carbohydrate and fat, the end product of which is water (H_2O) and carbon dioxide (CO_2).
- When oxygenation is inadequate to meet tissue needs, the condition is called *hypoxia*.
- In the presence of hypoxia, aerobic metabolism cannot proceed and the end product of anaerobic metabolism is lactic acid.

Acid, hydrogen ions, and pH

- An acid is a hydrogen ion (H^+) donor. Conversely an alkali is an H^+ acceptor.
- The loss of 1 mmol of alkali is equivalent to the gain of 1 mmol of acid and vice versa.
- The acidity and alkalinity of a solution is measured by the pH scale.
- The pH of a solution is equal to the negative log of the hydrogen ion concentration in that solution: pH = - log [H^+]. Thus pH is low when hydrogen ion concentration is high and vice versa.
- Arterial blood pH is maintained normal at 7.40 (range 7.35 to 7.45). When arterial blood pH is <7.35, the pH value is acidotic, the blood is said to be acidemic, and the condition is called *acidosis*. When arterial blood pH is >7.45, the pH value is alkalotic, the blood is said to be alkalemic, and the condition is called *alkalosis*.

CO_2 as a source of H^+ ions and its elimination

- As much as 15,000—20,000 mmol of CO_2 is produced through oxidative metabolism per day. CO_2 is called a "volatile acid" because it can combine reversibly with H_2O to yield a strongly acidic H^+ ion and a weak basic bicarbonate ion (HCO_3^-) according to the following equation:

$$CO_2 + H_2O \leftrightarrow H^+ + HCO_3^- \qquad (1)$$

- Normally CO_2 produced in this manner is eliminated by the lungs through respiration and there is no net gain of H^+ ion by the body.

- When respiration and CO_2 elimination are inadequate, the retained CO_2 will drive equation (1) to the right and release more strongly acidic H^+ ion. As a consequence arterial blood becomes more acidemic (pH falls below normal range) and the condition is called *respiratory acidosis*.
- With hyperventilation (ventilation increased above normal), more CO_2 is eliminated and equation (1) is driven to the left with a fall in H^+ ion concentration. As a consequence arterial blood becomes more alkalemic (pH rises above normal range) and the condition is called *respiratory alkalosis*.

Other sources of H^+ ions

- Metabolism of dietary protein produces the "non-volatile" hydrochloric and sulfuric acids, and metabolism of ingested phosphate produces phosphoric acid. The normal H^+ ion load from this source is approximately 1 mmol of H^+ ions per kilogram of body weight per day.
- The daily H^+ ion load from non-volatile acids may be increased through other processes. For example:
    - Anaerobic metabolism yielding lactic acid as the end product.
    - Incomplete oxidation of fatty acids.
    - Gastrointestinal loss of alkali through diarrhea (a loss of 1 mmol of alkali is equivalent to a gain of 1 mmol of acid).
    - Ingestion of acidifying compounds (e.g., NH_4Cl).
    - Hyperalimentation.

- The daily H^+ ion load from non-volatile acids may be decreased through some other processes. For example:
    - Loss of hydrochloric acid (HCl) in gastric secretions through vomiting or gastric suction.
    - Metabolism of vegetables and fruits in dietary intake produces alkali (a gain of 1 mmol of alkali is equivalent to a loss of 1 mmol of acid).

Renal excretion of H^+ and HCO_3^- ions

- The kidneys play a pivotal role in acid-base balance. They excrete H^+ ions in exchange of HCO_3^- ions when the H^+ ion load rises above normal and retain H^+ ions in exchange of HCO_3^- ions when the H^+ ion load falls below normal.
- When excretion of H^+ ions in exchange of HCO_3^- ions by the kidneys is not enough to eliminate the H^+ ion load, H^+ ion concentration rises above normal, HCO_3^- ion concentration falls below normal, arterial blood pH becomes more acidic, and the condition is called *metabolic acidosis*.
- When retention of H^+ ions in exchange of HCO_3^- ions by the kidneys is not enough to maintain H^+ ion concentration normal because of a low H^+ ion load, H^+ ion concentration falls below normal, HCO_3^- ion concentration rises above normal, and arterial blood pH becomes more alkalemic. The condition is called *metabolic alkalosis*.

Compensations

- In the presence of respiratory acidosis the kidneys compensate for the fall in pH by excreting H^+ ions and retaining HCO_3^- ions. As a result, pH rises towards normal and HCO_3^- concentration rises above normal. Renal compensation (also called metabolic compensation) to respiratory acidosis is a slow process. Compensation is not obvious for several hours and takes 4 days to complete. Even then compensation is not total and pH is never completely corrected to normal.
- In the presence of respiratory alkalosis the kidneys compensate for the increase in pH by retaining H^+ ions and excreting HCO_3^- ions. As a result, pH falls towards normal and HCO_3^- concentration falls below normal. Again renal compensation to respiratory alkalosis is a slow process and the pH does not completely return to normal.
- In the presence of metabolic acidosis, ventilation of the lungs increases through stimulation of central chemoreptors (H^+ ion receptors) in the medulla and peripheral chemoreceptors in the carotid and aortic bodies. Consequently PCO_2 falls below normal, equation (1) is pushed towards the left and H^+ ion concentration falls. This respiratory compensation contributes to increase the acidic pH towards normal, but not completely. The respiratory system responds to metabolic acidosis quickly and predictably by hyperventilation, so much so that pure metabolic acidosis is seldom seen, except in patients who are mechanically ventilated with a fixed minute volume in the Intensive Care Unit or in debilitated patients whose respiratory muscles are too weak to cope with the increased workload of breathing.
- The respiratory response to metabolic alkalosis is hypoventilation. PCO_2 rises above normal. Respiratory compensation to metabolic alkalosis is variable and unpredictable. It is unlikely that a conscious patient breathing spontaneously will hypoventilate to a $PCO_2 > 7.3$ kPa (55 mmHg) to compensate for metabolic alkalosis.

A systematic approach to the interpretation of ABG results

Step 1: Check the origin of the ABG results and make sure they belong to the patient you are about to review. Check the name and identification number on the report against the patient's other records (e.g., identification bracelet). Attributing the results from another patient to the one you are about to review can cause confusion and cost you dearly in court if the consequence is medical misadventure.

Step 2: Review the patient's clinical condition and the indication for the ABG examination. This information will help you to focus your attention.

Step 3: Obtain previous results, if there is any, for comparison. In medical practice changes in findings are as important as findings at any discrete moment.

Step 4: Check if the patient is breathing room air (21% oxygen) or an oxygen supplement. Note the oxygen concentration if it is an oxygen supplement.

Step 5: Look at the PO_2 and decide if hypoxia is present.

	Hypoxia	Normal	Hyperoxia
PO_2	<12 kPa (<90 mmHg)	12 to 13 kPa (90 to100 mmHg)	>13 kPa (>100 mmHg)
SO_2	<95%	95—100 %	

- In the International System of Units (SI Units), the unit of pressure measurement is pascal (Pa). kPa stands for kilopascal and equals 1,000 Pa. The pascal unit is meant to be used for all pressure measurements, including blood pressure and gas tension like PO_2 and PCO_2. If the pascal unit is used for blood pressure measurement, a normal blood pressure of 120/80 mmHg would be an unwieldy 15960/10640 Pa. Therefore doctors around the world have agreed to retain mmHg as the unit of blood pressure measurement. With respect to gas tension, there is no universal agreement. Some doctors use the pascal unit and others use mmHg. The conversion factors between kPa and mmHg are:
  - 1 atmosphere = 100 kPa = 760 mmHg
  - 1 kPa = 7.6 mmHg
  - 1 mmHg = 0.132 kPa.
- PO_2 should be close to 13.2 kPa or 100 mmHg in young healthy adults breathing room air (21% oxygen) at sea level. Due to an increase in V/Q mismatch, PO_2 falls with age even when the individual is fit and healthy. The following equation gives a reasonable estimation of PO_2 (in mmHg) in patients older than 40 years:

  $PO_2 = 104 - (age \times 0.27)$ mmHg
- SO_2 stands for oxygen saturation or percentage of hemoglobin combined with oxygen. In ABG results, SO_2 is derived from PO_2 using the oxygen-hemoglobin dissociation curve. It can also be measured using a pulse oximeter. SO_2 may be as low as 90% in healthy patients of advanced age.

Step 6: Look at the pH and decide whether its value is acidotic, normal, or alkalotic.

	Acidotic	Normal	Alkalotic
pH	<7.35	7.35 to 7.45	>7.45

- The pH is the best indicator of whether the underlying disorder is acidosis or alkalosis. Even when compensatory mechanisms are fully functional, by themselves, they cannot correct the underlying disturbance completely.
- Acidosis is more sinister than alkalosis. Very low arterial blood pH may be incompatible with life. A pH of 7.2 is a dire sign requiring urgent action.

Step 7: Look at the PCO_2 and the standard bicarbonate concentration [$sHCO_3^-$] or base excess (BE) to decide whether the acid-base disturbance is of respiratory or metabolic origin.

	Respiratory acidosis	Normal	Respiratory alkalosis
PCO_2	>6 kPa (>45 mmHg)	4.6 to 6 kPa (35 to 45 mmHg)	<4.6 kPa (<35 mmHg)
	Metabolic acidosis	**Normal**	**Metabolic alkalosis**
[$sHCO_3^-$]	<22 mmol/L	22 to 26 mmol/L	>26 mmol/L
BE	>-2 mmol/L	-2 to +2 mmol/L	>+2 mmol/L

- Standard bicarbonate concentration is not the bicarbonate concentration reported in serum electrolyte results or the so-called actually bicarbonate concentrated reported in some ABG results. It is what the bicarbonate concentration would have been after the PCO_2 is corrected to 5.3 kPa or 40 mmHg. According to equation 1, an increase in PCO_2 can cause a rise in HCO_3^- ion concentration and a decrease in PCO_2 can cause a fall in HCO_3^- ion concentration. Standard bicarbonate concentration is the derived bicarbonate concentration after changes associated with respiratory disturbances are eliminated. It reflects purely metabolic changes.
- Base excess (BE) is the mmol/L of base that needs to be removed to bring the pH back to normal when PCO_2 is corrected to 5.3 kPa or 40 mmHg. BE is reported together with or instead of standard bicarbonate concentration in some ABG results. It works just as well and is gaining popularity. (**N.B.** A negative BE is sometime called a base deficit or BD. That is, a BE of -2 is equal to a BD of 2.)

Step 8: Look at the pH, PCO_2, & BE / [$sHCO_3^-$] together to decide whether compensatory mechanisms are at work. (**N.B.** When respiratory or renal compensatory mechanisms are invoked to correct acid-base abnormalities, the pH is not corrected completely back to normal.)

Acidosis

- pH <7.35 (acidotic) PCO_2 ↑ (acidosis) BE / [$sHCO_3^-$] ↔ (normal)

  The pH is acidotic and the increase in PCO_2 means that the primary process is respiratory acidosis. Since BE / [$sHCO_3^-$] is normal, the condition is simple respiratory acidosis.

- pH <7.35 (acidotic) PCO_2 ↑ (acidosis) BE / [$sHCO_3^-$] ↑ (alkalosis)

The pH is acidotic and the increase in PCO_2 means that the primary process is respiratory acidosis. Since BE / [$sHCO_3^-$] is increased, the process is respiratory acidosis with compensatory metabolic (renal) alkalosis.

- pH <7.35 acidotic — PCO_2 ⇔ normal — BE / [$sHCO_3^-$] ↓ acidosis

The pH is acidotic and the decrease in BE / [$sHCO_3^-$] means that the primary process is metabolic acidosis. Since PCO_2 remains normal in this instance, the condition is metabolic acidosis without respiratory compensation. As was pointed out in a previous section, the respiratory system responds to metabolic acidosis quickly and predictably by hyperventilation, so much so that metabolic acidosis without respiratory compensation is not seen, except in patients who are not able to mount such a response (e.g., because of debilitation or dependence on fixed volume mechanical ventilation).

- pH <7.35 acidotic — PCO_2 ↓ alkalosis — BE / [$sHCO_3^-$] ↓ acidosis

The pH is acidotic and the decrease in BE / [$sHCO_3^-$] means that the primary process is metabolic acidosis. Since the PCO_2 is below normal, the process is metabolic acidosis with compensatory respiratory alkalosis.

Alkalosis

- pH >7.45 alkalotic — PCO_2 ↓ alkalosis — BE / [$sHCO_3^-$] ⇔ normal

The pH is alkalotic and the decrease in PCO_2 means that the primary process is respiratory alkalosis. Since BE / [$sHCO_3^-$] is normal, the condition is simple respiratory alkalosis.

- pH >7.45 alkalotic — PCO_2 ↓ alkalosis — BE / [$sHCO_3^-$] ↓ acidosis

The pH is alkalotic and the decrease in PCO_2 means that the primary process is respiratory alkalosis. Since the BE / [$sHCO_3^-$] is lower than normal, the condition is respiratory alkalosis with compensatory metabolic (renal) acidosis.

- pH >7.45 alkalotic — PCO_2 ⇔ normal — BE / [$sHCO_3^-$] ↑ alkalosis

The pH is alkalotic and an increase in BE / [$sHCO_3^-$] means that the primary process is metabolic alkalosis. Since PCO_2 remains normal the condition is simply metabolic alkalosis without respiratory compensation.

- pH >7.45 alkalotic | PCO_2 ↑ acidosis | BE / [$sHCO_3^-$] ↑ alkalosis

  The pH is alkalotic and the increase in BE / [$sHCO_3^-$] means that the primary process is metabolic alkalosis. Respiratory compensation causes an increase in PCO_2. Therefore the condition is metabolic alkalosis with compensatory respiratory acidosis. The respiratory response to metabolic alkalosis is hypoventilation. This response is variable and unpredictable. It is unlikely that a conscious patient breathing spontaneously will hypoventilate to a PCO_2 > 7.3 kPa (55 mmHg) to compensate for metabolic alkalosis.

Step 9: In the presence of metabolic acidosis, look at the serum electrolyte results and determine the *anion gap*.

- Based on the principle of electrical neutrality, the serum concentration of cations (positive ions) should equal the serum concentration of anions (negative ions). However, serum Na^+ ion concentration is higher than the sum of serum Cl^- and HCO_3^- concentration. (**N.B.** K^+ is also a cation like Na^+, but its serum concentration is very low and is ignored by most doctors in this calculation of electrical neutrality.) This is because there are some other anions (AG^-)in serum that are not measured and not reported in serum electrolyte results:

  Na^+ mmol/L > Cl^- mmol/L + HCO_3^- mmol/L

  Or

  Na^+ mmol/L = Cl^- mmol/L + HCO_3^- mmol/L + AG^- mmol/L

  The anion gap is a measure of the concentration of these other unmeasured anions (AG^-):
  Anion Gap (AG^-) = Na^+ - (Cl^- + HCO_3^-) mmol/L

- Normal anion gap is 12 mmol/L (range 10—14 mmol/L).

Normal anion gap (hyperchloraemic) metabolic acidosis

- In normal anion gap acidosis, loss of HCO_3^- ions is accompanied by an increase in serum Cl^- concentration. That is why the anion gap remains normal and the condition is also called hyperchloraemic acidosis.
- Disease processes that can lead to normal anion gap (hyperchloraemic) acidosis can be summarized by the mnemonic **DURHAM**:

  - **D**iarrhea

    In diarrhea both HCO_3^- and water is lost from the lower GI tract and the remaining Cl^- becomes more concentrated.

- **U**reteral diversion

  Urine from the ureter may be diverted to the sigmoid colon due to disease (uretero-colonic fistula) or after bladder surgery. In such an event urinary Cl^- is absorbed by the colonic mucosa in exchange for HCO_3^-, thus increases the gastrointestinal loss of HCO_3^-.

- **R**enal tubular acidosis

  In renal tubular acidosis dysfunctional renal tubular cells causes an inappropriate wastage of HCO_3^- and retention of Cl^-.

- **H**yperalimentation

  Ammonium chloride (NH_4Cl) and amino acids in hyperalimentation feeds are metabolized to hydrochloric acid (HCl) and produce a transient normal anion hyperchloraemic metabolic acidosis.

- **A**cetazolamide

  Acetazolamide is a carbonic anhydrase inhibitor used to treat glaucoma, a condition in which the intraocular pressure (pressure within the eye) is above normal. Normal carbonic anhydrase function is necessary for renal tubular cells to generate and retain HCO_3^- (review the appropriate lectures on renal tubular physiology). Therefore all carbonic anhydrase inhibitors can cause a metabolic acidosis similar to that found in renal tubular acidosis.

- **M**iscellaneous conditions

  They include pancreatic fistula, cholestyramine, and calcium chloride (CaCl) ingestion, all of which can increase the gastrointestinal wastage of HCO_3^-.

Increased anion gap metabolic acidosis

- In increased anion gap metabolic acidosis, the nonvolatile acids are organic or other inorganic acids (e.g., lactic acid, acetoacetic acid, formic acid, sulphuric acid).
- The anions of these acids are not Cl^- ions. The presence of these acid anions, which are not measured, will cause an increase in the anion gap.
- A useful mnemonic to help remembering the common causes of metabolic acidosis accompanied by an increased anion gap is **MUD PILES**:

  - **M**ethanol poisoning

    Methanol is metabolized by alcohol dehydrogenase in the liver to formic acid. There is also concomitant accumulation of lactic acid. Besides causing

increased anion gap acidosis, formic acid is toxic and causes visual loss, coma, and death.

- **U**remia

  In end-stage renal failure in which glomerular filtration rate falls below 10—20 ml/min, acids from protein metabolism are not excreted and accumulate in blood.

- **D**iabetic ketoacidosis

  In diabetic ketoacidosis incomplete oxidation of fatty acids causes a build up of beta-hydroxybutyric and acetoactic acids (ketoacids).

- **P**araldehyde poisoning

  Paraldehyde poisoning can cause a metabolic acidosis of the increased anion gap type, but the organic acid responsible has not been identified.

- **I**schaemia

  Ischaemia causes lactic acidosis (see section below).

- **L**actic acidosis

  Lactic acid is the end product of glucose breakdown if pyruvic acid, the end product of anaerobic glycolysis, is not oxidized to CO_2 and H_2O via the Tricarboxylic Acid Cycle (review the relevant lectures on glucose metabolism). Lactic acidosis occurs most commonly in a setting of oxygen lack, whether oxygen deficiency is absolute or relative (hypoxia, ischemia, hypotension, sepsis).

- **E**thylene glycol poisoning

  Ethylene is metabolized by alcohol dehydrogenase to oxalic acid in the liver. Usually there is also a coexisting lactic acidosis. Oxalic acid is toxic to brain tissue and oxalic acid crystals can cause renal failure by obstructing urine outflow (obstructive nephropathy).

- **S**alicylate poisoning

  Salicylate, found in aspirin, stimulates the respiratory centre and the first manifestation of poisoning is respiratory alkalosis. Subsequently, uncoupling of oxidative phosphorylation and inhibition of enzymes in the Tricyclic Acid Cycle lead to lactic acidosis.

Step 10: Review the patient again to see whether his clinical status is consistent with the ABG results.

- Treat the patient. Do not treat the numbers!