

Arterial Blood Gas : Basics and Interpretation

* Prasad R, ** Vineet Mahajan, ***Sanjay Verma, *0**Nikhil Gupta

* Professor and Head, ** Junior Resident, *** Senior Resident, ****Senior Resident,
Department of Pulmonary Medicine, CSMMU.
Department of Medicine, Era's Medical College.

Introduction

Blood gas analysis is an important routine investigation in patients who present with respiratory distress with the objective of knowing the acid-base status and to decide about the need for intervention. The usefulness of this diagnostic tool is dependent on being able to correctly interpret the results. Disorders of acid-base balance can be found in as many as 9 of every 10 patients in the Intensive Care Unit (ICU)¹ which means that acid-base disorders may be the most common clinical problems we will encounter in the ICU.

ACID-BASE BALANCE: BASIC PHYSIOLOGY

The pH is a measurement of the acidity or alkalinity of the blood. Hydrogen ions (H⁺) concentration in extracellular fluid is determined by the balance between the partial pressure of CO₂ and concentration of bicarbonate (HCO₃) in fluid. This relationship is expressed as follows¹:

$$H^+ (n \text{ Eq/L}) = 24 X (pCO_2/HCO_3)$$

pH and Hydrogen ions (H⁺) concentration²:

Hydrogen ions (H⁺) in extracellular fluid are expressed in nano equivalents per litre. Hydrogen ions (H⁺) is generally expressed as pH because nano equivalents is very small unit. A normal (H⁺) of 40 meq/l corresponds to a pH of 7.40. pH of a solution is inversely related to concentration of hydrogen ions. The pH of a solution is measured on a scale from 1 (very acidic) to 14 (very alkalotic). A liquid with a pH of 7, such as water, is neutral (neither acidic nor alkalotic). The normal blood pH range is 7.35 to 7.45. When the pH is below 7.35, the blood is said to be acidic. When the pH is above 7.45, the blood is said to be alkalotic.

The Intracellular and extracellular pH are generally maintained at very constant levels. The body pH is stabilized by the buffering capacity of the body fluids. A buffer is a substance that has the ability to bind or release hydrogen ions (H⁺) in solution, thus keeping the pH of the solution relatively constant despite the addition of considerable quantities of acids or bases.

THE RESPIRATORY BUFFER RESPONSE

A normal by-product of cellular metabolism is carbon dioxide (CO₂). Approximately 15,000 meq. Of CO₂

is generated daily and excreted by the lungs. CO_2 is carried in the blood to the lungs, where excess CO_2 combines with water (H_2O) to form carbonic acid (H_2CO_3). The blood pH will change according to the level of carbonic acid present. This triggers the lungs to either increase or decrease the rate and depth of ventilation until the appropriate amount of CO_2 has been re-established. Activation of the lungs to compensate for an imbalance starts to occur within 1 to 3 minutes³.

THE RENAL BUFFER RESPONSE

In an effort to maintain the pH of the blood within its normal range, the kidneys excrete or retain bicarbonate (HCO_3^-). About 1 meq/kg of non volatile acids are generated daily and are being excreted by the kidneys. As the blood pH decreases, the kidneys will compensate by retaining HCO_3^- and as the pH rises, the kidneys excrete HCO_3^- through the urine⁴. Although the kidneys provide an excellent means of regulating acid-base balance, the system may take from hours to days to correct the imbalance. When the respiratory and renal systems are working together, they are able to keep the blood pH balanced by maintaining 1 part acid to 20 parts base.

ACID BASE CONTROL

The concentration of hydrogen ions (H^+) in extracellular fluid is normally kept $< 10 \text{ nEq/L}$ and the stability of pH depends upon the constant $\text{pCO}_2/\text{HCO}_3^-$ ratio. So a change in one component is accompanied by proportional change in other component to maintain constant pH. Thus, an increase in pCO_2 (respiratory acidosis) must be accompanied by an increase in HCO_3^- to keep the pH constant. A respiratory disorder always initiates a compensatory metabolic response and vice-versa. The initial change in pCO_2 or HCO_3^- is called primary acid base disorder and the subsequent response is called compensatory or secondary acid base disorder. The compensatory responses are not enough to maintain the pH constant; they only limit the change in pH.

TYPES OF ACID-BASE DISORDERS:

Respiratory Acidosis

Respiratory acidosis is defined as a pH less than 7.35 with a PaCO_2 greater than 45 mm Hg⁵. Acidosis is caused by an accumulation of CO_2 which combines with water in the body to produce carbonic acid, thus, lowering the pH of the blood. Any condition that results in hypoventilation can cause respiratory acidosis. These conditions include:

- Central nervous system depression related to head injury.
- Central nervous system depression related to medications such as narcotics, sedatives, or anesthesia.
- Impaired respiratory muscle function related to spinal cord injury, neuromuscular diseases, or neuromuscular blocking drugs⁶.
- Hypoventilation due to pain, chest wall injury/deformity, or abdominal distension.

Respiratory Alkalosis

Respiratory alkalosis is defined as a pH greater than 7.45 with a PaCO_2 less than 35 mm Hg. Any condition that causes hyperventilation can result in respiratory alkalosis. These conditions include:

- Psychological responses, such as anxiety or fear.
- Pain.
- Increased metabolic demands, such as fever, sepsis, pregnancy, or thyrotoxicosis.
- Medications, such as respiratory stimulants.
- Central nervous system lesions.
- High altitude.
- Severe anaemia.
- Aspiration.
- Pulmonary disorders like pneumonia and pulmonary edema.

Metabolic Acidosis⁷

Metabolic acidosis is defined as a bicarbonate level of less than 22 mEq/L with a pH of less than 7.35.

Metabolic acidosis is caused by either a deficit of base in the bloodstream or an excess of acids, other than CO_2 . Diarrhoea and intestinal fistulas may cause decreased levels of base. Causes of increased acids include:

- Renal failure.
- Diabetic ketoacidosis⁸.
- Anaerobic metabolism.
- Starvation.
- Salicylate intoxication⁹.

Metabolic Alkalosis¹⁰

Metabolic alkalosis is defined as a bicarbonate level greater than 26 mEq/L with a pH greater than 7.45. Either an excess of base or a loss of acid within the body can cause metabolic alkalosis. Excess base occurs from ingestion of antacids, excess use of bicarbonate, or use of lactate in dialysis. Loss of acids can occur secondary to protracted vomiting, gastric suction, hypochloremia, excess administration of diuretics, or high levels of aldosterone.

COMPONENTS OF THE ARTERIAL BLOOD GAS

The arterial blood gas provides the following values:

pH

Measurement of acidity or alkalinity, based on the hydrogen (H^+) ions present.

The normal range is 7.35 to 7.45

PaO_2

The partial pressure of oxygen that is dissolved in arterial blood.

The normal range is 80 to 100 mm Hg.

SaO_2

The arterial oxygen saturation.

The normal range is 95% to 100%.

PaCO_2^*

The amount of carbon dioxide dissolved in arterial blood.

The normal range is 35 to 45 mm Hg.

HCO_3^{}**

The calculated value of the amount of bicarbonate in the bloodstream.

The normal range is 22 to 26 mEq/litre.

B.E.

The base excess indicates the amount of excess or insufficient level of bicarbonate in the system. The normal range is -2 to $+2$ mEq/liter. (A negative base excess indicates a base deficit in the blood.)

*when change occurs in PaCO_2 , then condition is called respiratory acid base disorder: an increase in PaCO_2 is respiratory acidosis and decrease in PaCO_2 is called respiratory alkalosis.

**when change occurs in HCO_3 , then condition is called metabolic acid base disorder: a decrease in HCO_3 is metabolic acidosis and increase in HCO_3 is called metabolic alkalosis.

PREPARATION FOR ARTERIAL SAMPLING¹¹

Following points must be considered before obtaining sample to avoid errors in blood gas measurement.

Steady State

Blood sampling must be done during steady state whenever there is initiation or change in oxygen therapy or changes in ventilatory parameters with patients on mechanical ventilation. In the patients without overt pulmonary disease a steady state is reached between 3-10 minutes and in patients with chronic airways obstruction it takes about 20-30 minutes.

Anticoagulants

Excess of heparin may affect the pH. Only 0.05 ml is required to anticoagulate 1 ml of blood¹¹. Because dead space volume of a standard 5 ml syringe with 1 inch 22 gauge needle is 0.2 ml, filling the syringe dead space with heparin provides sufficient volume to anticoagulate a 4 ml blood sample.

Delay in processing of sample

Because blood is a living tissue, O_2 is being consumed and CO_2 is produced in the blood sample. The delay may affect the blood gas values. In case of delay, the sample

should be placed in ice and such iced sample can be processed up to 2 hours without affecting the blood gas values.

Venous Sampling¹²

Arterial blood provides more information than venous blood with regard to acid-base and oxygenation status. However venous blood from a well perfused patient may provide a gross indication of acid-base status but is unacceptable for oxygenation status. Following points may help in recognizing inadvertent venous sampling (i) Failure to observe a sudden flash of blood in the needle on puncturing the vessel(ii) Incompatibility of values with clinical condition (iii) Low PaO₂ and high PaCO₂ (iv) SpO₂ by pulse oximetry more than SaO₂ by ABG analysis¹³.

STEPS TO AN ARTERIAL BLOOD GAS INTERPRETATION^{14,15}

STEP-I: TO IDENTIFY THE PRIMARY ACID BASE DISORDER

In the first part of approach, the measured PaCO₂ and pH are used to determine the acid base disturbance¹⁶.

PART 1 :

An acid base abnormality is present if either the PaCO₂ or pH is outside the normal range, however a normal PaCO₂ or pH does not exclude the presence of an acid base abnormality.

PART 2:

If the pH and PaCO₂ are both abnormal, then compare the directional change. If the both changes in same direction (both increase or decrease), the primary acid-base disorder is metabolic and if both changes are in opposite direction, the primary acid-base disorder is respiratory.

PART 3:

If either pH or PaCO₂ is normal, there is mixed metabolic and respiratory acid-base disorder. If the pH is normal, the direction of change in PaCO₂ identify the respiratory disorder and if PaCO₂ is normal, the direction of change in pH identifies the metabolic disorder.

STEP-II: TO EVALUATE THE COMPENSATORY RESPONSE¹⁷

The aim in this stage is to determine the additional acid base derangement.

PART 1

If there is primary metabolic acidosis or alkalosis then use the measured serum bicarbonate by equation 1 and 2.

$$\text{Expected PaCO}_2 = (1.5 \times \text{HCO}_3) + (8+2)$$

[for metabolic acidosis]

(Equation – 1)

$$\text{Expected PaCO}_2 = (0.7 \times \text{HCO}_3) + (21+2)$$

[for metabolic alkalosis]

(Equation – 2)

If measured PaCO₂ is equal to expected PaCO₂, then the condition is fully compensated. If measured value is more than the expected value, it shows that there is superimposed respiratory acidosis and if measured value is less than the expected value, it shows superimposed respiratory alkalosis.

PART 2

If there is primary respiratory acidosis or respiratory alkalosis then PaCO₂ should be used to calculate the expected pH by equation 3,4,5 and 6.

$$\text{Expected pH} = 7.40 - [0.008 \times (\text{PaCO}_2 - 40)]$$

[acute respiratory Acidosis]

(Equation – 3)

$$\text{Expected pH} = 7.40 + [0.008 \times (40 - \text{PaCO}_2)]$$

[acute respiratory Alkalosis]

(Equation – 4)

$$\text{Expected pH} = 7.40 - [0.003 \times (\text{PaCO}_2 - 40)]$$

[chronic respiratory Acidosis]

(Equation – 5)

$$\text{Expected pH} = 7.40 + [0.003 \times (40 - \text{PaCO}_2)]$$

[chronic respiratory Alkalosis]

(Equation – 6)

For respiratory acidosis: If measured pH is lower than expected pH for acute, uncompensated condition, there is superimposed metabolic acidosis and if measured pH is higher than expected pH for chronic compensated condition, there is superimposed metabolic alkalosis.

For respiratory alkalosis: If measured pH is higher than expected pH for acute, uncompensated condition, there is superimposed metabolic alkalosis and if measured pH is lower than expected pH for chronic compensated condition, there is superimposed metabolic acidosis.

Rule of thumb: In acute respiratory acidosis, HCO_3^- will increase by 1 mmol/L for every 10 mmHg increase in PaCO_2 . In chronic respiratory acidosis, HCO_3^- will increase by 4 mmol/L for every 10 mmHg increase in PaCO_2 . In acute respiratory alkalosis, HCO_3^- will decrease by 2 mmol/L for every 10 mmHg decrease in PaCO_2 . In chronic respiratory alkalosis, HCO_3^- will decrease by 4 mmol/L for every 10 mmHg decrease in PaCO_2 .

STEP-III: TO EVALUATE THE 'GAPS' FOR METABOLIC ACIDOSIS¹⁸

A measurement that is of some value in the differential diagnosis of metabolic acidosis is the anion gap. This gap is the difference between concentration of unmeasured cations and the concentration of unmeasured anions. It consists of most parts of proteins in anionic forms. The normal value of anion gap is 8-16 mEq/L. The anion gap is calculated by following equation, given below¹⁹

$$\text{Na} - (\text{Cl} + \text{HCO}_3^-) = \text{Unmeasured Anions} - \text{Unmeasured Cations.}$$

The difference between two groups is due to the albumin concentration.

Effect of albumin on anion gap²⁰: As described above that albumin is a major source of unmeasured anions and a 50% reduction in the albumin concentration will result in a 75% reduction in anion gap.

Interpretation of anion gap: On the basis of anion gap, the metabolic acidosis are grouped into, high anion gap acidosis or normal anion gap acidosis.

High anion gap metabolic acidosis²¹: The high anion gap acidosis is caused by the addition of a fixed (non volatile) acid to extracellular fluid.

Causes:

- Lactic acidosis
- Ketoacidosis (Diabetes, alcoholism, starvation)
- End stage renal failure
- Methanol ingestion
- Ethylene glycol ingestion
- Salicylates

Normal anion gap metabolic acidosis : The normal anion gap acidosis is caused by the net increase in chloride concentration in the extracellular fluid.

Causes:

- Diarrhea
- Isotonic saline infusion
- Early renal insufficiency
- Renal tubular acidosis

OTHER QUANTITATIVE MEASURES OF GAS EXCHANGE ABNORMALITY IN LUNGS:

1. Dead space ventilation
2. Intrapulmonary shunt fraction
3. The A-a PO_2 gradient

Dead space ventilation²²: The calculation of dead space ventilation (V_D/V_T) is based on the difference between the PaCO_2 in exhaled gas and end capillary (arterial) blood.

Intrapulmonary shunt fraction: The intrapulmonary shunt is derived by the relationship between the O_2 content in arterial blood, mixed venous blood and pulmonary capillary blood²³.

The A-a PO_2 gradient²⁴: The po_2 difference between alveolar gas and arterial blood ($\text{PAO}_2 - \text{PaO}_2$) is an indirect measure of ventilation-perfusion abnormalities. The $\text{PAO}_2 - \text{PaO}_2$ (A-a PO_2) gradient is determined with alveolar gas shown below :

$$PAO_2 = PIO_2 - (PaCO_2/RQ)$$

$$* PIO_2 = FIO_2 (P_B - P_{H_2O})$$

In a healthy subject breathing room air at sea level, $FIO_2 = 0.21$, $P_B = 760$ mmHg, $P_{H_2O} = 47$ mm Hg, $PAO_2 = 90$ mmHg, $PaCO_2 = 40$ mm Hg and $RQ = 0.8$.

$$A-a PO_2 = [0.21(760-47) - (40/0.8)] - 90 = 10 \text{ mmHg.}$$

SUMMARY

Understanding arterial blood gases can sometimes be confusing. A logical and systematic approach using these steps makes interpretation much easier. Applying the concepts of acid base balance will help the healthcare provider follow the progress of a patient and evaluate the effectiveness of care being provided.

Reference

- Gilfix BM, Bique M, Magder S. A physical chemical approach to the analysis of acid-base balance in the clinical setting. *J Crit Care* 1993;8:187-197.
- Breen PH. Arterial Blood Gas and pH Analysis. *Anesthesiology Clinics of North America* 2001;19: 885-902.
- Kellum JA. Determinants of plasma acid-base balance. *Crit Care Clin* 2005;21:329-346.
- Rose BD. Clinical physiology of acid-base and electrolyte disorders. 3d ed. New York: McGraw-Hill, 1989:464-77
- Madias NE, Cohen JJ. Respiratory acidosis. In: Cohen JJ, Kassirer JP, eds. Acid-base. Boston: Little, Brown, 1982:307-48.
- Respiratory pump failure: primary hypercapnia (respiratory acidosis). In: Adrogue HJ, Tobin MJ. Respiratory failure. Cambridge, Mass.: Blackwell Science, 1997:125-34.
- Casaletto JJ. Differential diagnosis of metabolic acidosis. *Emerg Med Clin North Am* 2005;23:771-787.
- Charfen MA, Fernandez-Frackelton M. Diabetic ketoacidosis. *Emerg Med Clin North Am* 2005;23:609-628.
- Hill JB. Salicylate intoxication. *N Engl J Med* 1973;288:1110-1113.
- Khanna A, Kurtzman NA. Metabolic alkalosis. *Respir Care* 2001;46:354-365.
- Blonshine S, M Foss CM, Mottram C, Ruppel G, Wanger J. Blood Gas Analysis and Hemoximetry: 2001 Revision & Update. *Respir Care* 2001;46:498-505.
- Sherman SC, Schindlbeck M. When is venous blood gas analysis enough? *Emergency Medicine* 2006; 38:44-48.
- Weil MH, Rackow EC, Trevino R. Difference in acid-base state between venous and arterial blood during cardiopulmonary resuscitation. *N Engl J Med* 1986;315:153-156.
- Rutecki GW, Whittier FC. An approach to clinical acid-base problem solving. *Compr Ther* 1998; 24:553-559.
- Morganroth M. An analytic approach to diagnosing acid-base disorders. *The Journal of Critical Illness* 1990; 5:138-150.
- Das B. Acid-base disorders. *Indian J Anaesth* 2003;47:373-379.
- Whittier WL, Rutecki GW. Primer on clinical acid-base problem solving. *Dis Mon* 2004;50:117-162.
- Emmet M, Narins RG. Clinical use of the anion gap. *Medicine* 1977;56:38-54.
- Gabow PA, Kaehny WD, Fennessey PV, Goodman SI, Gross PA, Schrier RW. Diagnostic importance of an increased serum anion gap. *N Engl J Med* 1980;303:854-858.
- Figge J, Jabor A, Kazda A, Fencl V. Anion gap and hypoalbuminemia. *Crit Care Med* 1998;26:1807-1810.
- Gabow PA, Kaehny WD, Fennessey PV, Goodman SI, Gross PA, Schrier RW. Diagnostic importance of an increased serum anion gap. *N Engl J Med* 1980;303:854-858.
- Lanken PN. Ventilation-perfusion relationships. In: Grippi MA, ed. Pulmonary pathophysiology. Philadelphia: JB Lippincott, 1995:195-210.
- D'Alonzo GE, Dantzger DR. Mechanisms of abnormal gas exchange. *Med Clin North Am* 1983;67:557-571.
- Gammon RB, Jefferson LS. Interpretation of arterial oxygen tension. UpToDate Web Site 2006. (Accessed 3/11/2006).