

**INTRODUCTION**

Acid-Base balance is an intricate concept which requires an intimate and detailed knowledge of the body's metabolic pathways used to eliminate the H<sup>+</sup> ion. Accurately interpreting acid-base balance requires simultaneous measurements of arterial pH and plasma electrolytes, as well as knowledge of compensatory physiologic mechanisms. In this article, we'll review normal acid-base physiology, acid-base disturbances, and lab techniques and mathematical calculations used to identify the cause of acid-base derangements.

**BASIC PHYSIOLOGY****Acid-base Chemistry****pH**

The concept of pH was put forward by the Danish chemist, Soren Peter Sorensen in 1909 to refer to the negative logarithm of hydrogen ion (H<sup>+</sup>) concentration. An increase in the pH indicates a proportionate decrease in the [H<sup>+</sup>] and a decrease in the pH indicates a proportionate increase in the [H<sup>+</sup>].<sup>1</sup>

$$\text{pH} = -\log [\text{H}^+]$$

The new generation of blood gas machines will report the H<sup>+</sup> as well as the pH. Acid is an H<sup>+</sup> donor and base is H<sup>+</sup> acceptor.<sup>2</sup> The intra and extracellular buffer systems minimize the changes in H<sup>+</sup> that occur as a result of addition of an acid or alkali load to the extracellular fluid (ECF), 60% of the acid load is buffered in the intracellular fluid (ICF). The most important buffer is the imidazole ring of the histidine in the hemoglobin molecule. The bicarbonate/carbonic acid is a weak buffer.<sup>3</sup>

However the presence of carbonic anhydrase, the high solubility of CO<sub>2</sub> and the ability of kidney to synthesize new bicarbonate and above all the efficient removal of CO<sub>2</sub> by lungs make it a powerful buffer. All buffers in a common solution are in equilibrium with the same H<sup>+</sup> ion concentration. Therefore, whenever there is a change in the H<sup>+</sup> ion concentration in the CSF, the balance of all the buffer system changes at the same time – the isohydric principle. It is therefore enough to study one buffer system in order to evaluate the acid base status of ECF.<sup>4</sup>

The Henderson-Hasselbalch equation, with its reliance on logarithms and antilogarithms is long and cumbersome and when attempting to deal with clinical situations. Kassirer and Blich have rearranged the Henderson equation that relates H<sup>+</sup> (instead of pH) to PCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup> and have derived an expression, which has great clinical utility.

$$\text{H}^+ = 24 \times \text{PCO}_2 / \text{HCO}_3^-$$

It is important to emphasize that H<sup>+</sup> ion concentration is

defined by the ratio of PCO<sub>2</sub> to HCO<sub>3</sub><sup>-</sup> and not by absolute value of either one alone.<sup>5,6</sup>

**Overview of Fundamentals of Acid-Base Disorder**

Normal metabolism of proteins and nucleotides generates about 100 mmol H<sup>+</sup> per day in the form of sulphuric and phosphoric acids. By comparison, hydration of CO<sub>2</sub> to form H<sub>2</sub>CO<sub>3</sub> generates 12,500 mmol H<sup>+</sup> per day.

**Carbon dioxide transport**

1. Transport of carbon dioxide in the blood is considerably more complex. A small portion of carbon dioxide, about 5 percent, remains unchanged and is transported dissolved in blood.
2. The remainder is found in reversible chemical combinations in red blood cells or plasma. Some carbon dioxide binds to blood proteins, principally hemoglobin, to form a compound known as carbamate.
- 3., About 88 percent of carbon dioxide in the blood is in the form of bicarbonate ion.

Carbon dioxide enters blood in the tissues because its local partial pressure is greater than its partial pressure in blood flowing through the tissues. As carbon dioxide enters the blood, it combines with water to form carbonic acid (H<sub>2</sub>CO<sub>3</sub>), a relatively weak acid, which dissociates into hydrogen ions (H<sup>+</sup>) and bicarbonate ions (HCO<sub>3</sub><sup>-</sup>). Blood acidity is minimally affected by the released hydrogen ions because blood proteins, especially hemoglobin, are effective buffering agents.<sup>7</sup>

The natural conversion of carbon dioxide to carbonic acid is a relatively slow process; however, carbonic anhydrase, a protein enzyme present inside the red blood cell, catalyzes this reaction with sufficient rapidity that it is accomplished in only a fraction of a second. Because the enzyme is present only inside the red blood cell, bicarbonate accumulates to a much greater extent within the red cell than in the plasma. The capacity of blood to carry carbon dioxide as bicarbonate is enhanced by an ion transport system inside the red blood cell membrane that simultaneously moves a bicarbonate ion out of the cell and into the plasma in exchange for a chloride ion. The simultaneous exchange of these two ions, known as the chloride shift, permits the plasma to be used as a storage site for bicarbonate without changing the electrical charge of either the plasma or the red blood cell.<sup>8</sup>

Hemoglobin acts in another way to facilitate the transport of carbon dioxide. Amino groups of the hemoglobin molecule react reversibly with carbon dioxide in solution to yield carbamates. A few amino sites on hemoglobin are

554 labile that is, their ability to bind carbon dioxide depends on the state of oxygenation of the hemoglobin molecule.<sup>9</sup>

Only 5 percent of carbon dioxide in the blood is transported free in physical solution without chemical change or binding, yet this pool is important, because only free carbon dioxide easily crosses biologic membranes. Virtually every molecule of carbon dioxide produced by metabolism must exist in the free form as it enters blood in the tissues and leaves capillaries in the lung. Between these two events, most carbon dioxide is transported as bicarbonate or carbamate.<sup>10</sup>

### Bicarbonate

Bicarbonate is a weak base that is regulated by the kidneys as part of acid–base homeostasis. The  $\text{HCO}_3^-$  measured in arterial blood reflects the metabolic component of arterial blood. Together,  $\text{CO}_2$  and  $\text{HCO}_3^-$  act as metabolic and respiratory buffers respectively. They are related via the equation:



### Compensatory changes

For any disturbance of gas tensions in arterial blood, a compensatory system exists to maintain homeostasis. In a metabolic disorder, where  $\text{HCO}_3^-$  may be retained or excreted by the kidneys, respiratory compensation can occur almost immediately to alter the rate and depth of ventilation to retain or remove  $\text{CO}_2$ . This occurs due to the exquisite sensitivity of chemoreceptors in the medulla to carbonic acid ( $\text{H}_2\text{CO}_3$ ) or  $\text{H}^+$ . Renal compensation in response to a respiratory disorder takes much longer, sometimes between three and five days, to retain or remove  $\text{HCO}_3^-$  as required.<sup>9</sup>

As a general rule, when compensation is present the arterial blood gas result shows two imbalances – derangement of both  $\text{HCO}_3^-$  and  $\text{PaCO}_2$ . A clue to which imbalance is the primary disturbance is obtained from the pH. If pH is leaning toward acidosis or alkalosis, then the parameter that matches the pH trend (that is, is increased or decreased corresponding to pH) is the primary problem and the other is due to compensation.<sup>10</sup>

### Oxygenation

A  $\text{PaO}_2$  that is less than expected indicates hypoxemia. This can result from hypoventilation or a mismatch of ventilation and perfusion. If alveolar ventilation is adequate (that is,  $\text{PaCO}_2$  is normal), then the hypoxemia is almost certainly caused by a ventilation–perfusion disturbance. The nature of the hypoxemia can be further assessed by the difference between the alveolar and arterial oxygen tensions.

### The alveolar–arterial oxygen tension difference

If an arterial blood gas result shows hypoxemia (low  $\text{PaO}_2$ ) and inadequate alveolar ventilation (high  $\text{PaCO}_2$ ), it must be determined whether the hypoxemia is related to hypoventilation, or is secondary to a disturbance in ventilation–perfusion, or both. This is assessed by calculating the difference between the alveolar ( $\text{PAO}_2$ ) and arterial ( $\text{PaO}_2$ ) oxygen tensions.

The alveolar–arterial difference, or gradient, can be estimated only if the oxygen fraction of inspired air

( $\text{FiO}_2$ , usually 0.21 on room air), barometric pressure and water vapor pressure are known. A normal reference range is 5–15 mmHg. The difference, expressed as  $\text{P (A–a) O}_2$ , increases with age, cigarette smoking and increasing  $\text{FiO}_2$ . An expected  $\text{P (A–a) O}_2$  can be calculated using the formula  $\text{P (A–a) O}_2 = 3 + (0.21 \times \text{patient's age})$ .

All causes of hypoxemia, apart from hypoventilation, increase the alveolar–arterial difference. In a patient breathing room air, a  $\text{P (A–a) O}_2$  greater than 15 mmHg suggests a ventilation–perfusion mismatch related to disease of the airways, lung parenchyma or pulmonary vasculature. However, the result is non-specific in defining the actual pathology and again the patient's clinical features are essential for diagnosis.

## NOMENCLATURE FOR CLINICAL INTERPRETATION OF ARTERIAL BLOOD GAS MEASUREMENTS

### Acid base disorders

Several definitions that are used to describe disturbances in acid base status are useful in understanding acid base disorders

Acidemia – A  $\text{H}^+$  ion above the normal range of 36–44 nmolL<sup>-1</sup>, pH less than 7.36

Acidosis – A process that would cause acidemia, if not compensated

Alkalemia – A  $\text{H}^+$  below the range of 36–44 nmolL<sup>-1</sup>, pH greater than 7.44

Alkalosis – A process that would cause alkalemia if not compensated

There are mainly two types of disorders, respiratory and metabolic. They may be compensatory or non-compensatory. Changes in pH that are primarily a result of changes in  $\text{PCO}_2$  are termed respiratory disorders. On the other hand changes in pH brought about by changes in bicarbonate and other buffer bases are termed primary metabolic disorders. Basically there are four primary acid–base disorders viz. respiratory acidosis, metabolic acidosis, respiratory alkalosis and metabolic alkalosis.<sup>13</sup> Compensation usually occurs in a primary acid base disturbance with an appropriate change in other components, e.g. a primary metabolic acidosis is compensated for by secondary respiratory alkalosis (by hyperventilation). On the other hand primary respiratory acidosis as it occurs in chronic obstructive airway disease (COPD) is secondarily compensated for by metabolic alkalosis brought about by  $\text{H}^+$  secretion and  $\text{HCO}_3^-$  absorption by the kidneys. While the former takes a few minutes to achieve the result, the latter may take days to weeks to be fully established.<sup>14</sup>

### Respiratory acidosis

In respiratory acidosis, the patient's pH is less than 7.35 and his  $\text{PaCO}_2$  is above 45 mm Hg (the upper limit of normal). Alveolar hypoventilation is the only mechanism that causes hypercarbia, or a  $\text{PaCO}_2$  above the upper limit of normal. The amount of alveolar ventilation necessary to maintain normal  $\text{PaCO}_2$  varies depending upon  $\text{CO}_2$  produced. The relationship between  $\text{PaCO}_2$  and plasma  $\text{HCO}_3^-$  determines arterial pH. Generally, acute increases in  $\text{PaCO}_2$  are accompanied by only minimal changes in

serum  $\text{HCO}_3^-$ . However, over a period of 1 to 3 days, renal conservation of  $\text{HCO}_3^-$  - results in an increase in pH.<sup>15</sup>

Chronic respiratory acidosis occurs secondary to a chronic reduction in alveolar ventilation—for example, in chronic lung diseases such as chronic obstructive pulmonary disease COPD, other main causes are: sedation, coma, neuromuscular disorders, severe kyphoscoliosis or obesity, pulmonary Fibrosis, sarcoidosis, pneumothorax or effusion, chronic obstructive airway disease, airway obstruction, severe pulmonary parenchymal disease etc.<sup>16</sup>

### Respiratory alkalosis

Common in critical care, respiratory alkalosis occurs when  $\text{PaCO}_2$  is reduced, causing an increase in pH. The most common cause of respiratory alkalosis is increased alveolar ventilation, which can happen in hyperventilation, mechanical overventilation, hepatic disease, pregnancy, and septicemia. Determining appropriate compensatory changes in  $\text{HCO}_3^-$  is key to determining if the patient also has a concomitant metabolic disorder. In chronic respiratory alkalosis, the compensatory mechanisms result in mild reduction in plasma  $\text{HCO}_3^-$  levels to maintain a near normal or normal pH. This causes a mixed acid-base disorder. Treatment of respiratory alkalosis is directed at discovering and correcting the underlying etiology. For example, if a patient is hyperventilating from anxiety, have him breathe into a paper bag. In mechanically ventilated patients with mechanical overventilation, reducing the minute ventilation or tidal volume will increase  $\text{PaCO}_2$  and reduce pH.<sup>17</sup>

### Metabolic acidosis

Metabolic acidosis is an increase in the amount of absolute body acid, either from excess production of acids or excessive loss of bicarbonate, sodium, and potassium. Causes of metabolic acidosis include lactic acidosis, diabetic ketoacidosis, and loss of bicarbonate through severe diarrhea or bicarbonate wasting through the kidneys or gastrointestinal (GI) tract.<sup>18</sup>

In general, the kidneys attempt to preserve sodium by exchanging it for excreted  $\text{H}^+$  or potassium. In the presence of an  $\text{H}^+$  load,  $\text{H}^+$  ions move from the extracellular fluid into the intracellular fluid. For this process to occur, potassium moves outside the cell into the extracellular fluid to maintain electro neutrality. In severe acidosis, significant overall depletion of total body potassium stores can occur despite serum hyperkalemia. This is why I.V. potassium is given to patients with diabetic ketoacidosis early in treatment, despite the often-elevated serum potassium level. External and internal potassium balances are regulated to maintain an extracellular fluid concentration of 3.5 to 5.5 mEq/L and a total body content of about 50 mEq/kg (40mEq/kg in females). Main causes are: hypovolemia, cardiogenic or septic shock, severe hypoxia, diabetic ketoacidosis, renal failure, diarrhea, pancreatic fistula.<sup>19</sup>

### Metabolic alkalosis

Metabolic alkalosis occurs when  $\text{HCO}_3^-$  is increased, usually as the result of excessive loss of metabolic acids. Causes of metabolic alkalosis include diuretics, secretory adenoma of the colon, emesis, hyperaldosteronism, Cushing's syndrome, and exogenous steroids. Some

causes of metabolic alkalosis respond to treatment with 0.9% sodium chloride solution. If the patient's urine chloride concentration is less than 15 mmol/L, his metabolic alkalosis is saline-responsive; urine chloride levels above 25 mmol/L indicate non-saline-responsive metabolic alkalosis. The mechanisms resulting in saline-responsive metabolic alkalosis include GI loss, diuresis, or renal compensation from hypercapnia. Non-saline responsive metabolic alkalosis results from mineralocorticoid excess or potassium depletion. Fluid administration is the foundation for treatment for saline-responsive metabolic alkalosis. In cases of extreme alkalosis, the patient may be given dilute hydrochloric acid. Saline-resistant alkalosis is treated by addressing the underlying etiology.<sup>19</sup>

ABG	pH	$\text{PaCO}_2$	$\text{HCO}_3^-$
Respiratory Acidosis	↓	↑	Normal
Respiratory Alkalosis	↑	↓	Normal
Metabolic Acidosis	↓	Normal	↓
Metabolic Alkalosis	↑	Normal	↑

### Estimate of Base Excess/Deficit

The metabolic component of the acid-base balance is reflected in the base excess. This is a calculated value derived from blood pH and  $\text{PaCO}_2$ . It is defined as the amount of acid required to restore a liter of blood to its normal pH at a  $\text{PaCO}_2$  of 40 mmHg. The base excess increases in metabolic alkalosis and decreases (or becomes more negative) in metabolic acidosis, but its utility in interpreting blood gas results is controversial.

While the base excess may give some idea of the metabolic nature of a disorder, it may also confuse the interpretation. The alkalaemia or acidaemia may be primary or secondary to respiratory acidosis or alkalosis. The base excess does not take into account the appropriateness of the metabolic response for any given disorder, thus limiting its utility when interpreting results.<sup>12</sup>

### Anion Gap

Anion gap (AG) represents the concentration of all the unmeasured anions in the plasma and is measured by the following formula:

$$\text{AG} = [\text{Na}^+] - [\text{Cl}^-] + [\text{HCO}_3^-]$$

Normal AG is  $12 \pm 4$  mEq/L. Conditions resulting in metabolic acidosis other than hydrochloric acidosis usually lead to a decrease in the serum bicarbonate concentration without a concomitant rise in serum chloride thereby increasing the AG.<sup>11</sup>

### Delta Ratio

Delta ratio is related to the AG and buffering, and is defined as:

$$\text{Delta ratio} = [\text{increase in AG} / \text{decrease in bicarbonate}]$$

A high delta ratio can occur when the bicarbonate levels are already elevated at the onset of the metabolic acidosis either due to a pre-existing metabolic alkalosis, or as a compensation for pre-existing respiratory acidosis. A low delta ratio occurs with hyperchloremic normal anion gap acidosis.<sup>18</sup>

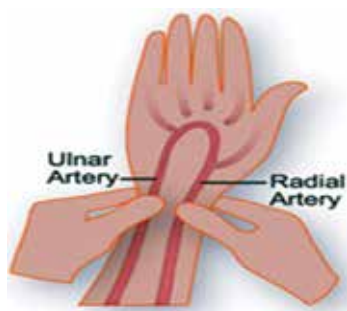


Fig. 1: Allen's Test

## INTERPRETATION OF THE ABG REPORT

### Clinical Assessment

Patients with acid-base disturbances may present with symptoms due to the etiological cause that resulted in the disturbance. They may also present with manifestations that develop as a consequence of the disturbance as well as with symptoms that have nothing to do with the acid-base disturbance. Therefore, a carefully obtained history and a thorough physical examination are essential for the interpretation of ABG report. The following sequence may be followed to interpret the ABG report.<sup>11</sup>

### Sampling and analysis

Blood is usually withdrawn from the radial artery (Figure 1) as it is easy to palpate and has a good collateral supply. The patient's arm is placed palm-up on a flat surface, with the wrist dorsiflexed at 45°. A towel may be placed under the wrist for support. The puncture site should be cleaned with alcohol or iodine, and a local anesthetic (such as 2% lignocaine) should be infiltrated. Local anesthetic makes arterial puncture less painful for the patient and does not increase the difficulty of the procedure.<sup>1</sup> The radial artery should be palpated for a pulse, and a pre-heparinized syringe with a 23 or 25 gauge needle should be inserted at an angle just distal to the palpated pulse to ensure accuracy, it is important to deliver the sample for analysis promptly. If there is any delay in processing the sample, the blood can be stored on ice for approximately 30 minutes with little effect on the accuracy of the results.

### Validity of the ABG Report

Firstly, whether pH, PaCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup> are compatible should be confirmed using the Henderson- Hassel Bach equation or acid-base nomograms.<sup>16</sup>

### Arterial pH

Net deviation in the arterial pH will indicate whether an acidosis or an alkalosis is present. If pH is normal, either no acid-base disorder is present or compensating disorders are present.<sup>18</sup>

### PaCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup>

Simple acid-base disorders result in a predictable change in the PaCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup>. Low PaCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup> indicate respiratory alkalosis or metabolic acidosis; but a mixed disorder cannot be excluded.<sup>19</sup>

Elevated PaCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup> indicate respiratory acidosis or metabolic alkalosis; but a mixed disorder cannot be excluded. If PaCO<sub>2</sub> and HCO<sub>3</sub><sup>-</sup> show a change in opposite directions, it is indicative of a mixed disorder.

Table 1: Comparing Acid-base imbalances

Condition	pH	Paco <sub>2</sub>	HCO <sub>3</sub>
Pure respiratory alkalosis	High	Low	Normal
Pure respiratory acidosis	Low	High	Normal
Pure metabolic alkalosis	High	Normal	High
Pure metabolic acidosis	Low	Normal	Low
Metabolic alkalosis with partial respiratory compensation	High	High	High
Metabolic acidosis with partial respiratory compensation	Low	Low	Low

### Compensatory Response

The expected compensatory response for simple acid-base disorders is shown in Table 1 below. If the expected values and the actual values match, a mixed disorder is unlikely. If the expected values and the actual values differ, a mixed disorder is present.<sup>18</sup>

### Calculating the Anion Gaps

The serum anion gap, calculated from the electrolytes measured in the chemical laboratory, is defined as the sum of serum chloride and bicarbonate concentrations subtracted from the serum sodium concentration. This entity is used in the detection and analysis of acid-base disorders, assessment of quality control in the chemical laboratory, and detection of such disorders as multiple myeloma, bromide intoxication, and lithium intoxication.<sup>20</sup> Low values most commonly indicate laboratory error or hypoalbuminemia but can denote the presence of a paraproteinemia or intoxication with lithium, bromide, or iodide. Elevated values most commonly indicate metabolic acidosis but can reflect laboratory error, metabolic alkalosis, hyperphosphatemia, or paraproteinemia. Metabolic acidosis can be divided into high anion and normal anion gap varieties, which can be present alone or concurrently (Table 2). The AG should be measured in all patients with metabolic acidosis.<sup>17</sup> Causes of elevated AG metabolic acidosis can be remembered with the mnemonic

MUDPILES [M = methanol; U = uremia; D = diabetic ketoacidosis (also alcoholic ketoacidosis and starvation); P = paraldehyde ingestion; I = isoniazid overdose; L = lactic acidosis; E = ethylene glycol poisoning; S = salicylate poisoning].

Normal AG metabolic acidosis can be grouped as per the serum potassium levels. Normal AG acidosis with a normal to high potassium include hyperaldosteronism, type IV renal tubular acidosis, moderate degree of renal failure, administration of hydrochloric acid and post-hypocapnia. Conditions causing normal AG acidosis include gastrointestinal losses of bicarbonate (diarrhea, ureteral diversion, biliary or pancreatic fistulas), carbonic anhydrase inhibitors, proximal and distal renal tubular acidosis. When  $\{(Na^+ + K^+) - Cl^-\}$  can help in distinguishing renal from non-renal causes. A negative urinary anion gap indicates a non renal cause of acidosis.<sup>16</sup>

**Table 2: Causes of Acidosis****High anion gap**

- Renal failure (severe)
- Lactic acidosis
- L: tissue hypoxia, tumors
- D: short bowel syndrome, mental status changes; not measured by routine lab
- Ketoacidosis: diabetic, alcoholic
- Poisonings
- Salicylate (usually associated with respiratory alkalosis)
- Ethylene glycol
- Methanol
- Acetaminophen - induced 5 - oxoprolinuria (pyroglutamic aciduria)

**Normal anion gap**

- Diarrhea (loss HCO<sub>3</sub>)
- Renal tubular acidosis
- Renal failure
- Ureterosigmoidostomy
- Carbonic anhydrase inhibitors (e.g. acetazolamide for glaucoma)
- Dilution with hyperchloremic solutions (e.g. saline)
- Pancreatic or biliary diversion
- Administration of inorganic acid or acid equivalents
- Ketoacidosis, well hydrated or excretion of Na<sup>+</sup> ketones

of arterial blood. When combined with a patient's clinical features, blood gas analysis can facilitate diagnosis and management.

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**OSMOLAR GAP**

Osmolar gap is also useful in differentiating the causes of elevated AG metabolic acidosis. Osmolar gap is calculated by subtracting the calculated serum osmolality from measured osmolality using the formula shown below.<sup>15</sup>

$$\text{Calculated osmolality} = \text{Glucose (mg/dl)} + 2[\text{Na} + (\text{mEq/l})]/18 + \text{Blood urea nitrogen (mg/dl)} / 2.8$$

Osmolar gap = Measured osmolality - Calculated osmolality

Osmolar gap >10 mOsm/l is considered abnormal when calculated using this formula. Conditions causing high AG metabolic acidosis include ethanol, ethyleneglycol, methanol, acetone, isopropyl ethanol and propylene glycol poisoning.<sup>17</sup>

**CONCLUSIONS**

Measuring arterial blood gases can be a useful adjunct to the assessment of patients with either acute or chronic diseases. The results show if the patient is acidaemic or alkalaemic and whether the cause is likely to have a respiratory or metabolic component. The PaCO<sub>2</sub> reflects alveolar ventilation and the PaO<sub>2</sub> reflects the oxygenation