Chapter

26

How to Interpret Arterial Blood Gas Data?

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INTRODUCTION

Arterial blood gas (ABG) analysis, considered to be one of the most precise measurements in medicine, is a crucial investigation that is frequently employed in the critical care setting[1,2]. A structured approach to rational interpretation of the ABG report requires a clear understanding of the basic concepts and physiological principles underlying the disorders of acid-base homeostasis, oxygenation and ventilation. In this review we have provided a brief overview regarding the essential fundamental physiological concepts and have attempted to provide a practical approach for bed-side interpretation of ABG results.

BASIC PHYSIOLOGICAL AND CHEMICAL CONCEPTS

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^+) concentration[3].

$$pH = -\log [H^+]$$

The pH is a 'dimensionless representation of the H^+, has no units and is just a number. There are several advantages of the expression pH compared to H^+. Measured by the pH electrode, pH, is related to the logarithm of H^+ "activity" rather than "concentration" of H^+ and this seems physiologically more appropriate. The relationship between pH and H^+ is shown in Table 1. For each 0.1 pH unit increment, H^+ falls by 20%; by remembering this relationship, the intermediate values can be derived accurately[4-7].

Table 1: Relationship between pH and $[H^+]$

pH	*$[H^+]$ (nmol/l)*
6.8	158
6.9	125
7.0	100
7.1	79
7.2	63
7.3	50
7.4	40
7.5	31
7.6	25
7.7	20
7.8	15

Overview of Acid-base Balance in the Human Body

The normal extracellular fluid (ECF) H^+ concentration is 40 nmol / l. Under normal physiological conditions, the H^+ concentration varies little from its normal value due to the processes of acid base regulation, as maintenance of H^+ concentration at this level is considered to be essential for normal cellular processes. In order to achieve this, close interaction between three physiological systems of the body, namely, the chemical buffers of the body, kidneys and the lungs is considered essential[4-7].

Dissolved carbon dioxide is in equilibrium with carbonic acid

$$CO_2 + H_2O \underset{}{\overset{\text{Carbonic Anhydrase}}{\rightleftharpoons}} H_2CO_3$$

Carbonic acid ionizes according to the equation

$$H_2CO_3 \xrightarrow{\text{Carbonic Anhydrase}} H^+ + HCO_3^-$$

By the law of mass action:

$$\frac{[H^+]\,[HCO_3^-]}{[H_2CO_3]} = K'$$

Taking logarithm on both sides we have:

$$\log \frac{[H^+]\,[HCO_3^-]}{[H_2CO_3]} = \log K'$$

Therefore,

$$\log [H^+] + \log \frac{[HCO_3^-]}{[H_2CO_3]} = \log K'$$

and

$$\log [H^+] = \log K' - \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

By changing signs on both sides of the equation, we have log

$$- \log [H^+] = - \log K' + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

Since –log $[H^+]$ is called pH and –log K' is called pK, the equation can be rewritten as

$$pH = pK + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

This has been called the *Henderson-Hasselbach equation*. The pK represents the pH at which maximum buffering capacity can be achieved for that reaction. The carbonic acid concentration is dependent on the amount of dissolved carbon dioxide which in turn is dependent on its solubility and the partial pressure of carbon dioxide ($PaCO_2$) in the arterial blood. Therefore, in this equation, (H_2CO_3) can be substituted by ($s \times PaCO_2$), where s = solubility coefficient. In the clinical setting, pK = 6.1; s = 0.0301.

Therefore,

$$pH = 6.1 + \log \frac{[HCO_3^-]}{0.0301 \times PaCO_2}$$

This equation may also be rewritten as

$$H^+ = 24 \times \frac{PaCO_2}{[HCO_3^-]}$$

The body responds to the perturbations in the acid-base balance by buffering which is the immediate response followed by respiratory (alteration in ventilation) and renal (alteration in bicarbonate excretion) responses.

The major buffer system in the ECF is the carbon dioxide-bicarbonate buffer system. Other buffer systems that play a role in buffering are protein and phosphate buffer systems. In the blood, concentration of phosphate is very low and it is quantitatively unimportant. Protein buffers in blood include hemoglobin and plasma proteins. Hemoglobin is an important blood buffer particularly for buffering carbon dioxide. According to the "isohydric principle", at a given moment, all buffer systems which participate in defence of acid-base changes are in equilibrium with each other. It is important to remember that the carbonate and phosphate salts in bone act as a long-term supply of buffer especially during prolonged metabolic acidosis.

Alveolar Ventilation

Alveolar ventilation is best defined in terms of ventilation of carbon dioxide. The $PaCO_2$ directly reflects the adequacy of alveolar ventilation. Elevation of $PaCO_2$ > 50 mmHg indicates alveolar hypoventilation and low $PaCO_2$ <30 mmHg is suggestive of alveolar hyperventilation.

In a steady state:

$$\dot{V}_A = \frac{863 \times \dot{V}\,CO_2}{PACO_2}$$

or,

$$PACO_2 = \frac{863 \times \dot{V}\,CO_2}{\dot{V}_A}$$

Where

$\dot{V}CO_2$ = carbon dioxide production by the body (l / min) corrected to standard temperature and pressure, dry (STP)

$\dot{V}_A$ = alveolar volume per unit of time corrected to body temperature and pressure, saturated with water (BTPS)

863 = conversion factor to express $\dot{V}_A$ in l / min BTPS

$PACO_2$ = partial pressure of alveolar carbon dioxide expressed in mm Hg, BTPS which is essentially equal to $PaCO_2$.

Oxygenation

Oxygenation is the transfer of adequate oxygen from the alveoli to the blood. Several issues such as oxygen

content, oxygen delivery, the oxygen-hemoglobin dissociation curve, ventilation perfusion mismatch, shunt, and the alveolar-arterial gradient [$P(A\text{-}a)O_2$] are all important in evaluating the oxygenation status.

Oxygen Content

Oxygen is present in the blood bound to hemoglobin and dissolved in plasma. The oxygen content (CaO_2) represents the sum of oxygen attached to the hemoglobin and that dissolved in plasma and is calculated by the formula

$$CaO_2 \text{ (ml/dl)} = [\text{Hb (g/dl)} \times 1.34 \times \% \text{ saturation}] + [0.003 \times PaO_2]$$

Blood at BTPS with 15 g/dl hemoglobin and 100% saturation has a CaO_2 of 17 - 20 ml/dl (vol%). Mixed venous oxygen content (CvO_2) is the amount of oxygen carried by venous blood in ml/dl (vol %)

$$C\bar{v}O_2 \text{ (ml/dl)} = [\text{Hb (g/dl)} \times 1.34 \times \% \text{ saturation}] \times [0.003 \times P\bar{v}O_2]$$

Blood at BTPS with 15g/dl haemoglobin and 100% saturation has a $C\bar{v}O_2$ of 12 - 15 ml/dl (vol%).

The arterial-venous oxygen content difference [$CaO_2 - C\bar{v}O_2$] = 5 ml/dl (vol%)

Oxygen-hemoglobin Dissociation Curve

Oxygen-hemoglobin dissociation curve represents the relationship between the partial pressure of arterial oxygen (PaO_2) and the oxygen saturation[8]. The P_{50} is the PaO_2 at which hemoglobin is 50% saturated. It also reveals that 90% saturation of hemoglobin corresponds to a PaO_2 of about 60 mmHg. A right shift in this curve results in increased unloading of oxygen and a left shift results in decreased unloading of oxygen at the tissue level.

Alveolar-arterial Oxygen Pressure Difference

$P(A\text{-}a)O_2$ takes into account the fact that alveolar and therefore arterial oxygen tension changes depending on the alveolar ventilation.

It is calculated using a simplified form of alveolar gas equation

$P(A\text{-}a)O_2 = PAO_2 - PaO_2$

Where, $PAO_2 = FIO_2 \times (PB - PH_2O) - PaCO_2/R$

PAO_2 = partial pressure of alveolar oxygen

PaO_2 = partial pressure of arterial oxygen

FIO_2 = fractional concentration of inspired oxygen (0.21 when breathing room air at sea level)

PB = barometric pressure (760 mm Hg at sea level)

PH_2O = water vapour pressure (47 mm Hg when air is fully saturated at 37°C)

R = respiratory quotient (the ratio of carbon dioxide production to oxygen consumption assumed to be 0.8 under steady state conditions). The respiratory quotient depends on the substrates being metabolized as fuel source.

Substituting the above values,

$$PAO_2 = 150 - 1.25 \times PaCO_2$$

In healthy young persons aged 20 years, the $P(A\text{-}a)O_2$ is normally 4 to15 mm Hg and the value increases with increasing age. $P(A\text{-}a)O_2$ can also be roughly calculated as [(age (years)/4)+4]. When hypoxaemia is purely due to a low inspired oxygen or alveolar hypoventilation, $P(A\text{-}a)O_2$ is normal. If $P(A\text{-}a)O_2$ and $PaCO_2$ are both elevated, additional mechanism such as ventilation-perfusion mismatch or shunt are likely to be contributing to hypoxia.

NOMENCLATURE FOR CLINICAL INTERPRETATION OF ARTERIAL BLOOD GAS MEASUREMENTS

The nomenclature for clinical interpretation of ABG measurements are listed in Tables 2a, 2b, and 2c. Normal laboratory values and acceptable therapeutic ranges for pH and $PaCO_2$ are shown in Table 3. The typical alterations in the ABG results and the expected compensatory mechanisms in simple acid-base disorders are listed in Table 4.

Indices for Acid-base Balance

These include $PaCO_2$, indices such as actual bicarbonate, standard bicarbonate, and buffer base. Standard bicarbonate is the measurement made in the clinical laboratory after the blood has been equilibrated at 37°C with a $PaCO_2$ of 40 mmHg. Actual bicarbonate is calculated from $PaCO_2$ and pH using Henderson-Hasselbach equation. Base excess is defined as the number of mEq of acid or base needed to titrate 1 L of blood to pH 7.40 at 37°C while $PaCO_2$ is held constant. For quick bed-side calculation, it can be assumed that at $PaCO_2$ held constant at 40 mm Hg, 7 mEq of acid or base are required to change pH by 1 unit (0.10). Base excess (or deficit) which is a true reflection of non-respiratory component of acid-base balance, is an estimate and not an actual measurement. Therefore, it will be reliable only if the measurements used to derive it, the pH and $PaCO_2$ are estimated accurately.

Table 2a: Nomenclature for clinical interpretation of ABG measurements: acid-base status

Acidosis is an abnormal process or condition which would lower arterial pH if there were no secondary changes in response to the primary etiological factor
Alkalosis is an abnormal process or condition which would raise arterial pH if there were no secondary changes in response to the primary etiological factor
Acidemia = Arterial pH < 7.36
Alkalemia = Arterial pH > 7.44
Simple acid-base disorders are those in which there is a single primary aetiological acid-base disorder.
Mixed acid-Base Disorders are those in which two or more primary etiological disorders are present simultaneously
Respiratory disorders are caused by abnormal processes which tend to alter pH because of a primary change in $PaCO_2$ levels
Metabolic disorders are caused by abnormal processes which tend to alter pH because of a primary change in $[HCO_3^-]$

Table 2b: Nomenclature for clinical interpretation of ABG measurements: ventilatory status

Ventilatory status is reflected by $PaCO_2$ levels. Accordingly,
$PaCO_2$ < 30 mmHg = alveolar hyperventilation (respiratory alkalosis)
$PaCO_2$ 30-50 mmHg = acceptable alveolar ventilation
$PaCO_2$ > 50 mmHg = ventilatory failure (respiratory acidosis)
Ventilatory state in conjunction with pH
Alveolar hyperventilation ($PaCO_2$ < 30 mmHg)
pH >7.5 = Acute alveolar hyperventilation
pH 7.40 – 7.5 = Chronic alveolar hyperventilation
pH 7.30 – 7.50 = Compensated metabolic acidosis
pH <7.3 = Partially compensated metabolic acidosis
Acceptable alveolar ventilation ($PaCO_2$ 30-50 mmHg)
pH >7.50 = Metabolic alkalosis
pH 7.30 – 7.50 = Acceptable alveolar ventilation and metabolic acid-base status
pH <7.30 = Metabolic acidosis
Ventilatory failure ($PaCO_2$ > 50 mmHg)
pH >7.50 = Partially compensated metabolic alkalosis
pH 7.30 – 7.50 = Chronic ventilatory failure
pH <7.30 = Acute ventilatory failure

Adapted from reference 4

Base excess is determined as follows[4]:

Calculating $PaCO_2$ Variance

First, the $PaCO_2$ variance is determined by calculating the difference between the measured $PaCO_2$ and 40; then, the decimal point is shifted two places to the left.

Example:

pH 7.06, $PaCO_2$ = 74

Table 2c: Nomenclature for clinical interpretation of ABG measurements: oxygentation status

These values correspond to a person under 60 years of age breathing room air at sea level
PaO_2 < 80 mmHg = mild hypoxemia
PaO_2 < 60 mmHg = moderate hypoxemia
PaO_2 < 40 mmHg = severe hypoxemia
Effect of oxygen therapy
Uncorrected hypoxemia = PaO_2 < room air acceptable limit*
Corrected hypoxemia = PaO_2 > room air acceptable limit; < 100 mm Hg
Excessively corrected hypoxemia = PaO_2 > 100 mmHg; <minimal predicted†
At any age, PaO_2 <40 mm Hg reflects severe hypoxaemia. For each year over the age range of 60-90 years, 1 mmHg should be substracted for limits of mild and moderate hypoxaemia
* Room air acceptable limits for PaO_2 for a child and adult *person under 60 years old* breathing room air at sea level: normal (97–100 mm Hg); acceptable (80 - 97 mm Hg); hypoxemia (<80 mmHg)
† If PaO_2 is < FIO_2 × 5, the patient can be assumed to be hypoxemic while breathing room air
Adapted from reference 4

Table 3: Normal laboratory values and acceptable therapeutic ranges for pH and $PaCO_2$

Variable	*Mean*	*1SD*	*2SD*	*Acceptable therapeutic range*
pH	7.40	7.38-7.42	7.35-7.45	7.30-7.50
$PaCO_2$ (mmHg)	40	38-42	35-45	30-50

SD = standard deviation

74 - 40 = 34

$PaCO_2$ variance = 0.34

pH 7.48, $PaCO_2$ = 20

40 - 20 = 20

$PaCO_2$ variance = 0.20

Calculating Predicted pH

Predicted respiratory pH is calculated as follows. If the $PaCO_2$ is greater than 40, half the $PaCO_2$ variance is subtracted from 40. If $PaCO_2$ is less than 40, the $PaCO_2$ variance is added to 7.4

Example:

1. pH 7.06, $PaCO_2$ = 74

   74 - 40 = 34

$PaCO_2$ variance = 0.34

0.34 × ½ = 0.17

Predicted respiratory pH = 7.40 - 0.17 = 7.23

2. pH 7.48, $PaCO_2$ = 20

   40 - 20 = 20

   $PaCO_2$ variance = 0.20

Predicted respiratory pH = 7.40 + 0.20 = 7.60

Estimate of Base Excess/Deficit

The difference between the predicted respiratory pH and the measured pH is calculated. This reflects the metabolic pH change. Normally, a 10 mEq/l variance from normal buffer baseline represents a pH change of 0.15 units. If the pH decimal point is moved two places to the right, a 10 (10 mEq/l) to 15 (0.15 unit) relationship will be evident and this has been expressed as "⅔ relationship". By determining the difference between the measured pH and the predicted respiratory pH, moving the decimal point two places to the right and multiplying by ⅔, base excess/deficit can be calculated.

When the measured pH is greater than the predicted pH, it is termed *base excess*. When the measured pH is less than the predicted pH, it is termed *base deficit*.

Example:

1. pH 7.06, $PaCO_2$ = 74, predicted pH = 7.23

   Difference between predicted and measured pH = 7.23 - 7.06 = 0.17

   Applying ⅔ rule 0.17 × ⅔ = 0.11

   Base deficit = 11 mEq/l

2. pH 7.48, $PaCO_2$ = 20, predicted pH = 7.60

   Difference between predicted and measured pH = 7.6 - 7.48 = 0.12

   Applying ⅔ rule 0.12 × ⅔ = 0.08

   Base deficit = 8 mEq/l

3. pH 7.20, $PaCO_2$ = 90

   90 - 40 = 50

   $PaCO_2$ variance = 0.50

   0.50 × ½ = 0.25

   Predicted respiratory pH = 7.40 - 0.25 = 7.15

   Difference between predicted and measured pH = 7.20 – 7.15 = 0.05

   Applying ⅔ rule 0.05 × ⅔ = 0.033

Base excess = 3 mEq/l

Base excess/deficit calculation serves as a useful guide for bicarbonate administration

Anion Gap

Anion gap (AG)[9] represents the concentration of all the unmeasured anions in the plasma and is measured by the following formula:

$$AG = [Na^+] - [K^-] + [HCO_3^-]$$

Normal AG is 12 ± 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.

Delta Ratio

Delta ratio[10] is related to the AG and buffering, and is defined as:

Delta ratio = [increase in AG/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.

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[10,11]. The following sequence may be followed to interpret the ABG report.

Validity of the ABG Report

Firstly, whether pH, $PaCO_2$ and HCO_3^- are compatible should be confirmed using the Henderson-Hasselbach equation or acid-base nomograms.

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.

Table 4: Typical alterations in the ABG results and the expected compensatory mechanisms

Acid-base Disorder	*Initial change*	*Compensatory response*	*Equations for expected range of compensation*
Metabolic Acidosis	↓ HCO_3^-	↓ $PaCO_2$	Expected $PaCO_2$ = [1.5 × (HCO_3^-) + 8] ± 2 ↓$PaCO_2$ = 1.2 × Δ (HCO_3^-) Expected $PaCO_2$ = Last two digits of pH
Metabolic Alkalosis	↓ HCO_3^-	↓ $PaCO_2$	Expected $PaCO_2$ = [0.9 × (HCO_3^-) + 16] ± 2 ↑$PaCO_2$ = 0.7 × Δ (HCO_3^-)
Respiratory Acidosis	↑ PCO_2	↑HCO_3^-	[HCO_3^-] increases 1 mEq/l for every 10 mmHg ↑ in $PaCO_2$
Acute			[HCO_3^-] increases 3.5 mEq/l for every 10 mmHg ↑ in $PaCO_2$
Chronic			
Respiratory alkalosis	↓ PCO_2	↓ HCO_3^-	
Acute			[HCO_3] falls 2 mEq/l for every 10 mmHg ↓ in $PaCO_2$
Chronic			[HCO_3^-] falls 5 mEq/l for every 10 mmHg ↓ in $PaCO_2$

$PaCO_2$ and HCO_3^-

Simple acid-base disorders result in a predictable change in the $PaCO_2$ and HCO_3^- (Table 4). Low $PaCO_2$ and HCO_3^- indicate respiratory alkalosis or metabolic acidosis; but a mixed disorder cannot be excluded. Elevated $PaCO_2$ and HCO_3^- indicate respiratory acidosis or metabolic alkalosis; but a mixed disorder cannot be excluded. If $PaCO_2$ and HCO_3^- show a change in opposite directions, it is indicative of a mixed disorder.

Compensatory Response

The expected compensatory response for simple acid-base disorders is shown in Table 4. 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.

Calculating the Anion Gaps

The AG should be measured in all patients with metabolic acidosis. 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 overdosage; **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 urine electrolytes are available, assuming a urine pH of less 6.1 and euvolemia, urinary anion gap [UAG = (Na^+ + K^+) - Cl^-] can help in distinguishing renal from non-renal causes. A negative urinary anion gap indicates a non-renal cause of acidosis. Delta ratio is also useful in the assessment of elevated AG metabolic acidosis to determine if a mixed acid base disorder is present (Table 5).

Table 5: Utility of delta ratio when assessing metabolic acid-base disorders

Delta ratio	*Interpretation*
< 0.4	Hyperchloremic normal anion gap acidosis
< 1	High AG and normal AG acidosis
1 to 2	Usual for uncomplicated high AG acidosis Lactic acidosis: average value 1.6DKA more likely to have a ratio closer to 1 due to urine ketone loss
> 2	High AG acidosis and a concurrent metabolic alkalosisor a pre-existing compensated respiratory acidosis

AG = anion gap

Osmolar gap[12] 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.

Calculated osmolality =

$$2[Na^+\ (mEq/l)] + \frac{Glucose\ (mg/dl)}{18} + \frac{Blood\ urea\ nitrogen\ (mg/dl)}{2.8}$$

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, ethylene

glycol, methanol, acetone, isopropyl ethanol and propylene glycol poisoning.

Assessment of Ventilatory and Oxygenation Status

The approach shown in Table 2b can be used to interpret the ABG report and classify the ventilatory status in conjunction with the pH. Finally, looking at the PaO_2 in the context of the FIO_2 (Table 2c) can aid in the evaluation of hypoxemia.

CONCLUSIONS

Detailed history taking and thorough physical examination are vital for obtaining clues regarding the underlying etiological conditions. Assessment of pH, $PaCO_2$, and HCO_3^- facilitates determination of whether a primary metabolic or respiratory disorder is present. Estimation of the predicted compensatory response for simple acid-base disorders might suggest the presence of an additional disease process if compensation is not appropriate. Calculation of the various gaps (AG, UAG, osmolar gap) can be helpful in differential diagnosis. With a clear understanding of the pathophysiological processes and by following the stepwise approach described, majority of the ABG reports can be interpreted with clarity.

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