Chapter 12 / Capítulo 12

Electrolyte and Internal Environment Imbalances in Emergencies (English Edition)
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Step-by-Step Interpretation of Arterial Blood Gas Analysis

Interpretación de la gasometría arterial paso a paso

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Objectives:

- Define the normal values of arterial blood gas and their variations.
- Establish a step-by-step interpretation of arterial blood gas.
- Understand the specific characteristics of each disorder to optimize arterial blood gas interpretation.

INTRODUCTION

Arterial blood gas (ABG) is an essential tool in medicine for assessing the body's acid-base balance, oxygenation status, and pulmonary ventilation. For general practitioners, understanding it in a simple way allows for the diagnosis and management of common disorders such as acidosis or alkalosis in patients with respiratory, metabolic, or mixed problems. This chapter focuses on a step-by-step approach using the Henderson-Hasselbalch equation, which relates pH to the respiratory component ($PaCO_2$) and the metabolic one (HCO_3^-). The goal is to provide a practical guide, avoiding unnecessary complexities, to interpret ABG in everyday clinical contexts such as emergencies or outpatient consultations.

Acid-base balance keeps blood pH between 7,35 and 7,45. The lungs regulate $PaCO_2$ (volatile acid) and the kidneys regulate HCO_3^- (metabolic base). Alterations in these lead to disorders: acidemia due to acidosis (low pH) or alkalemia due to alkalosis (high pH), which can be respiratory or metabolic. ABG measures pH, PaO_2 (partial pressure of oxygen), $PaCO_2$ (partial pressure of carbon dioxide), HCO_3^- (bicarbonate), and sometimes the anion gap (AG).

Validated Methods for ABG Interpretation

In a basic approach, three methodologies have been proposed to assess acid-base imbalances: 1) the traditional Henderson-Hasselbalch method, also called the physiological approach; 2) the standard base excess method (based on the Van Slyke equation); and 3) Stewart's quantitative method (which uses the strong ion difference and total weak acids).

Currently, the last two methods mentioned are the most widely used, particularly Stewart's approach (greater practical application), which presents an algorithm that downplays the physiological importance of carbonic acid and HCO_3^- from the traditional method, creating a contradiction with the physiological proposal. However, this system requires several variables, and when any of them is missing or assumed to be normal, the method becomes indistinguishable from the traditional ones. On the other hand, the Van Slyke equation does not indicate whether a fixed acid or base is responsible for a given balance, nor does it resolve the issue of base excess present in the Henderson-Hasselbalch procedure.

The physiological approach is considered easier to use from a clinical perspective, provides a physiological assessment of the disorder, offers a logic that is easily understandable for evaluating severity, and also provides a stronger foundation for developing effective therapies. However, its main drawback lies in the dissimilar formulas proposed for its interpretation, especially when dealing with metabolic acidosis.

Regardless of the physiological and chemical differences they present, authors such as Lovesio C *et al* argue that the three algorithms provide virtually identical results when used to quantify acid-base imbalances in a blood sample. In other words, there is no method superior to another.

Regardless of this, Sánchez-Díaz JS *et al*, state that the most common mistake is trying to interpret everything at once and in a disorganized manner. Estimating and performing incorrect calculations leads to erroneous diagnoses and results in poor treatments.

Having reliable and easy-to-use equations available at the patient's bedside is essential.

Approximate normal values

- pH 7,35 – 7,45.
- $PaCO_2$ 35 – 45 mmHg.
- HCO_3^- 22 – 26 mmol/L.
- PaO_2 80 – 100 mmHg.
- Base Excess (BE): ±2,5 mEq/L.

These approximate normal values may vary in different clinical contexts. We will refer to two that are particularly relevant for medical practice:

- Pregnancy: during pregnancy, various anatomical and physiological changes occur in the mother's respiratory system, leading to changes in lung volumes and alterations in arterial blood gases depending on the trimester of pregnancy (table 12.1).
- Altitude: as we ascend above sea level, barometric pressure decreases, and consequently, the inspired oxygen pressure (PiO_2) and arterial oxygen pressure (PaO_2) also decrease. Hypoxemia increases minute ventilation (VE), reducing $PaCO_2$.

This phenomenon clearly alters the blood gas pattern due to the primary and compensatory mechanisms previously described (Every respiratory compensation entails a metabolic response). According to the authors of this chapter, the reference values are established at sea level (table 12.1).

Table 12.1. Approximate normal values in pregnancy and at altitude

Values	1st Trimester	3rd Trimester	1388 meters	4200 meters
pH	7,40 - 7,44	7,41 - 7,45	7,43 - 7,49	7,39 - 7,42
PaO_2 (mmHg)	99 - 111	100 - 112	84 - 98	48 - 76
$PaCO_2$ (mmHg)	27 - 32	23 - 29	24 - 28	14 - 32
HCO_3^- (mmol/L)	17 - 19	15 - 19	17 - 18	12 - 20
O_2 Saturation (%)	98	98	96	90

Before interpreting, check the consistency of the sample: collect it in a heparinized syringe, remove air bubbles, and analyze promptly to avoid errors.

Acidemia/alkalemia or acidosis/alkalosis?

From this sequence of values described above, it can be determined that a change in $PaCO_2$ and/or HCO_3^- values will influence the extracellular pH, modifying it regardless of the physiological compensatory systems.

Thus, when one of these two variables shifts enough to cause a change in the usual pH values, the suffix ***-emia*** is used, and depending on that change, the condition is referred to as acidemia (pH < 7,35) or alkalemia (pH > 7,45). This terminology is fairly general and encompasses both

respiratory and metabolic disorders.

Thus, it can also be inferred that if the disorder affects $PaCO_2$, it is classified as a respiratory acid-base disorder, whereas if it involves HCO_3^-, then it is considered a metabolic acid-base disorder.

Step-by-step approach to interpretation

The initial change in PCO_2 and/or HCO_3^- is called the primary acid-base disorder; while the subsequent response is referred to as the compensatory or secondary acid-base disorder. It is important to note that compensatory responses are almost never strong enough to keep the pH constant (they do not correct the disorder); they only limit the pH change based on the other two variables.

Step 1. Assess the pH

- pH <7,35: Acidemia
- pH >7,45: Alkalemia.
- pH normal (7,35-7,45): Possible mixed or compensated disorder; check $PaCO_2$ and HCO_3^-.

Important: what to do if the pH is euphemic and the blood gas values are altered? As explained earlier, when reviewing a blood gas analysis, you may find a pH within normal limits (euphemic); however, both bicarbonate and PCO_2 are altered. This situation indicates a mixed disorder. The goal, then, is to refer to the Base Excess and compare it with normal values.

Base Excess refers to the amount of base required to return an individual's blood pH to the normal value (pH 7,4). Its normal reference value is ±2,5. If the values are within the ±2,5 range, then the primary disorder is respiratory. If they are outside the ±2,5 range, the disorder is metabolic.

Step 2. Determine the primary disorder

Acidemia (pH < 7,35): if HCO_3^- < 22 mmol/L, it is metabolic acidosis; or if $PaCO_2$ > 45 mmHg, it is respiratory acidosis.

Alkalemia (pH > 7,45): if HCO_3^- > 26 mmol/L, it is metabolic alkalosis; or if $PaCO_2$ < 35 mmHg, it is respiratory alkalosis.

Therefore, the primary disorder could be: an Acidemia (pH < 7,35) due to Metabolic Acidosis (HCO_3^- < 22 mmol/L).

Step 3. Determine the secondary or compensatory disorder

The interpretation of the compensation phase will be done by comparing the measured $PaCO_2$ (in the metabolic case) with the expected $PaCO_2$ according to the calculation.

In respiratory disorders, the measured HCO_3^- (in the blood gas analysis) is compared with the expected HCO_3^- according to the calculated result. Metabolic acid-base disorders trigger rapid ventilatory responses, mediated by peripheral chemoreceptors located in the carotid body at the carotid bifurcation in the neck. Metabolic acidosis stimulates these chemoreceptors and initiates an increase in ventilation, leading to a consequent decrease in arterial $PaCO_2$. Metabolic alkalosis silences the chemoreceptors and causes a rapid decrease in ventilation and an increase in arterial PCO_2.

Respiratory compensation for metabolic acidosis

In the steady state of metabolic acidosis, there is a precise, well-defined, and predictable relationship between the magnitude of the bicarbonate decrease and the degree of hypocapnia.

Expected $PaCO_2$ = (1,5 x HCO_3-) + 8 (±2)

- If the measured $PaCO_2$ equals the expected $PaCO_2$, then the disorder is compensated (compensated metabolic acidosis).
- If the measured $PaCO_2$ is lower than the expected $PaCO_2$, then we are dealing with an associated respiratory alkalosis (the disorder would then be called primary metabolic acidosis with associated respiratory alkalosis).
- If the measured $PaCO_2$ is higher than the expected $PaCO_2$, then there is an associated respiratory acidosis, and the disorder would be called primary metabolic acidosis with associated respiratory acidosis.

Respiratory compensation for metabolic alkalosis

To establish the diagnosis of the compensatory phase in this disorder, the plasma HCO_3^- concentration that classifies metabolic alkalosis must be considered.

For mild to moderate metabolic alkalosis ($HCO_3^- \leq 40$ mmol/L):
Expected $PaCO_2$ = (0,9 x HCO_3^-) + 15 (±2)

For severe metabolic alkalosis ($HCO_3^- > 40$ mmol/L):
Expected $PaCO_2$ = (0,9 x HCO_3^-) + 9 (±2)

- If the measured $PaCO_2$ equals the expected $PaCO_2$, then the disorder is compensated (compensated metabolic alkalosis).
- If the measured $PaCO_2$ is lower than the expected $PaCO_2$, then we are dealing with an associated respiratory alkalosis (the disorder would then be called primary metabolic alkalosis with associated respiratory alkalosis).
- If the measured $PaCO_2$ is higher than the expected $PaCO_2$, then there is an associated respiratory acidosis, and the disorder would be called primary metabolic alkalosis with associated respiratory acidosis.

Metabolic compensation for respiratory acidosis

Before determining the compensatory phase in respiratory acid-base disorders, it is essential to consider whether both disorders (respiratory acidosis and respiratory alkalosis) are acute or chronic.

This is because the expected compensation comes from the metabolic system, which is mostly generated by renal function, a mechanism that is activated later, unlike the respiratory compensatory system for metabolic disorders. Although this mechanism begins to act about 48 hours after the onset of the respiratory disorder, it is often very complex to determine this exact moment. Therefore, it is suggested to apply formulas to determine whether it is an acute or chronic disorder (based on the pH value measured in the blood gas analysis).

For acute respiratory acidosis: Calculated pH = 7,40 – (0,008 x [measured $PaCO_2$ - 40])

For chronic respiratory acidosis: Calculated pH = 7,40 – (0,003 x [measured $PaCO_2$ - 40])

For interpretation, the results of both formulas are compared with the pH measured in the blood gas analysis, and the result that is closest to the measured pH will indicate the type of respiratory acidosis.

Once it is determined whether the disorder is acute or chronic, one of the two formulas will be used to establish metabolic compensation.

For acute respiratory acidosis: Expected HCO_3- = 24 + (0,1 x [measured $PaCO_2$ - 40])

For chronic respiratory acidosis: Expected HCO_3- = 24 + (0,4 x [measured $PaCO_2$ - 40])

- If the measured HCO_3^- equals the expected value, it is a compensated disorder.
- If the measured HCO_3^- is lower than the expected value, it indicates an associated metabolic acidosis.
- If the measured HCO_3^- is higher than the expected value, we are dealing with an added metabolic alkalosis.

Metabolic compensation for respiratory alkalosis

Determine whether it is an acute or chronic disorder:

- Acute: calculated pH = 7,40 – (0,014 x [40 – measured $PaCO_2$])
- Chronic: calculated pH = 7,40 – (0,003 x [40 - measured $PaCO_2$])

For interpretation, the results of both formulas are compared with the pH measured in the blood gas analysis, and the result that is closest to the measured pH will indicate the type of respiratory alkalosis.

Once it is determined whether the disorder is acute or chronic, one of the two formulas will be used to establish metabolic compensation.

For acute respiratory alkalosis: Expected HCO_3- = 24 - (0,2 x [40 – measured $PaCO_2$])

For chronic respiratory acidosis: Expected HCO_3- = 24 - (0,5 x [40 - measured $PaCO_2$])

- If the measured HCO_3^- equals the expected value, it is a compensated disorder.
- If the measured HCO_3^- is lower than the expected value, it indicates an associated metabolic acidosis.
- If the measured HCO_3^- is higher than the expected value, we are dealing with an added metabolic alkalosis.

Considerations of interest for Acidemia due to Metabolic Acidosis

If it is a METABOLIC ACIDOSIS, calculate the Anion Gap (GAP or Anion GAP). What is the Anion Gap?

The concentrations of anions and cations in plasma must be equal to maintain electrical neutrality. Therefore, there is no real anionic gap in plasma. However, only certain anions and cations are routinely measured in the clinical laboratory. The cation normally measured is Na^+, and the anions are usually Cl^- and HCO_3^-. The anion gap (which is only a diagnostic concept) is the difference between the unmeasured anions and the unmeasured cations.

The difference (Unmeasured Anions [A^-] – Unmeasured Cations [Ca^+]) is a measure of the relative abundance of unmeasured anions, which is called the Anion Gap, Anionic Gap, GAP, or Anion GAP.

Anion Gap (AG) = Na+ - (Cl- + [HCO_3-]); normal 8-12 mmol/L.

- High AG (>12): Acidosis with high gap (e.g., ketoacidosis, lactic acidosis).
- Normal AG: Acidosis without gap (hyperchloremic, e.g., diarrhea).

Influence of Albumin

One source of error in interpreting the Anion Gap is failing to account for the contribution of albumin. A common finding in patients admitted to emergency services is a hypoalbuminemic state, secondary to underlying complications. This represents the most significant limitation in calculating the Anion Gap. Therefore, it should be adjusted according to serum albumin concentrations. The anionic equivalence of albumin is equal to 0,25 mEq/L for each gram of albumin per liter.

Adjustment for albuminemia

Corrected AG = AG + [2,5 × (4 – albumin g/dL)].

Important Note

Only adjust the Anion Gap for albumin if this calculation is normal or low.

The influence on the Anion Gap is not only from albumin, there are other factors that can alter it: hypercalcemia, IgG in multiple myeloma, salicylate overdose, hypermagnesemia, lithium intoxication, and hypertriglyceridemia.

If metabolic acidosis presents with an elevated Anion Gap, calculate the corrected HCO_3^- to determine whether another associated metabolic disorder exists.

When there is metabolic acidosis with an increased Anion Gap, it is possible to identify another metabolic acid-base disorder (a normal-gap metabolic acidosis or a metabolic alkalosis) by comparing the excess Anion Gap (difference between the measured and normal values) with the HCO_3^- deficit (difference between the measured plasma HCO_3^- concentration and the normal value).

Corrected HCO_3^- = measured HCO_3^- + (Anion Gap - 12)

- If Corrected HCO_3^- < 22 mmol/L: associated non-anion gap metabolic acidosis (hyperchloremic metabolic acidosis).
- If Corrected HCO_3^- > 26 mmol/L: associated metabolic alkalosis.

Determination of the Urinary Anion Gap (UAG) for normal-gap (hyperchloremic) acidosis

In the presence of metabolic acidemia with a normal anion gap, determining the urinary anion gap (UAG) is useful to define whether the alteration is due to renal or extrarenal causes. Practically speaking, if the anion gap is normal in metabolic acidosis, there is either a loss of HCO_3^- (mainly from the gastrointestinal tract or at the renal level) or an increase in extracellular fluid volume (hyperchloremic acidosis induced by parenteral administration of sodium chloride solution).

UAG = $(Na^+ + K^+) - Cl^-$

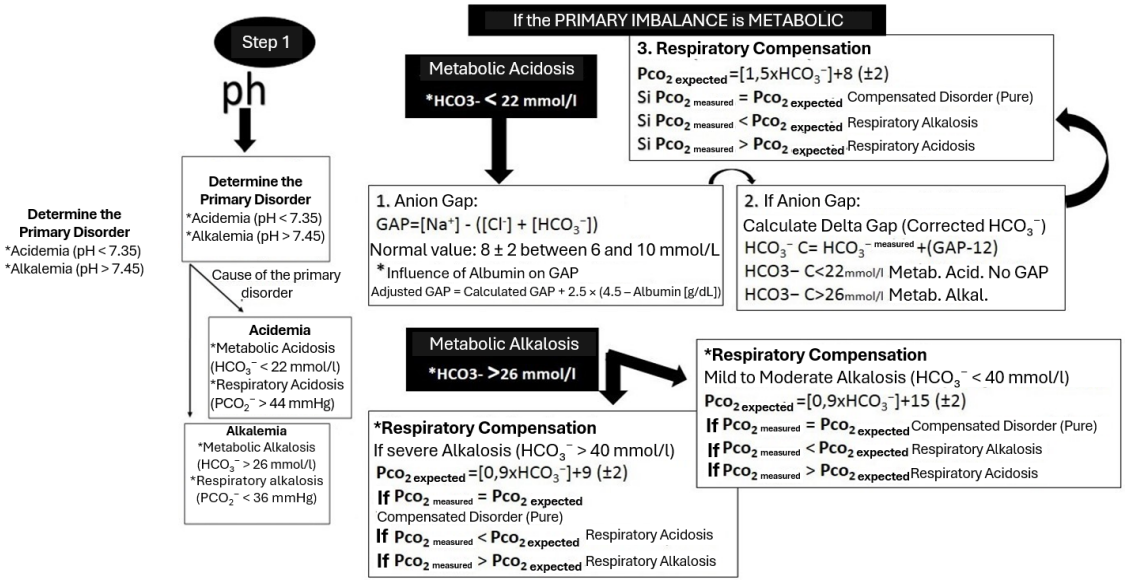
- If Chloride > $(Na^+ + K^+)$: extrarenal cause: indicates the presence of NH_4^+ ion in urine or normal urine acidification (diarrhea, ileus, pancreatic and biliary fistulas, ureterointestinal diversion, laxatives, calcium chloride, magnesium sulfate, or cholestyramine).
- If Chloride < $(Na^+ + K^+)$: renal cause: no NH_4^+ excretion occurs. There is renal loss of bicarbonate. Furthermore, if there is hypokalemia + urinary pH > 5,5: Renal tubular acidosis type II: primary (transient, genetic, or idiopathic), carbonic anhydrase alteration (acetazolamide deficiency), sulfanilamide, and Fanconi syndrome.

Important:

At the end of the chapter, a summary table is provided to guide the interpretation of the Anion Gap for each disorder (figures 12.1 and 12.2).

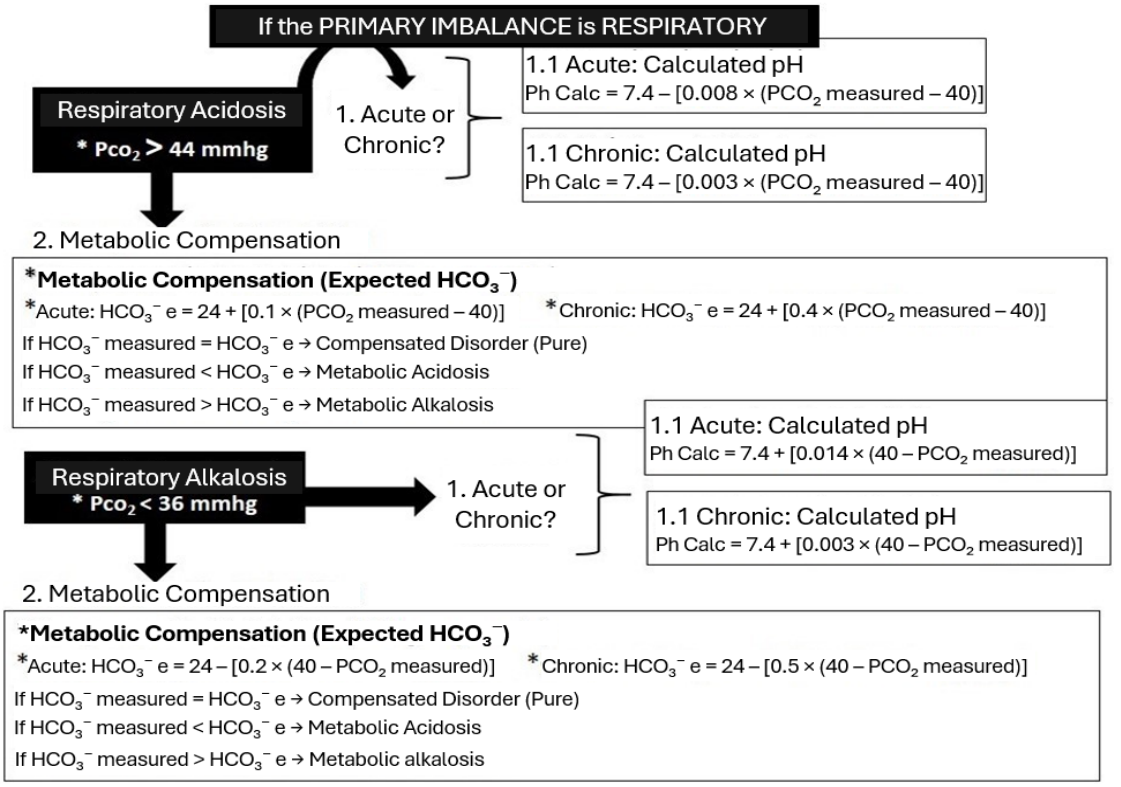
CHAPTER CONCLUSIONS

It is expected that, by using this tool, medical students and professionals can consequently minimize errors in the systematic reading and interpretation of the Anion Gap and provide an appropriate clinical and therapeutic approach to the patient.



Note: Own elaboration

Figure 12.1. Identification of the primary imbalance and sequence to follow if the disorder is metabolic



Source: Own elaboration

Figure 12.2. Identification of the primary imbalance and sequence to follow if the disorder is respiratory

The most complex acid-base disorder to fully determine is Acidemia due to Metabolic Acidosis, because along with this disorder, up to two additional associated defects may appear (Acidemia due to Metabolic Acidosis with anion GAP and non-anion GAP, along with a compensatory respiratory alkalosis).

The correct calculation of each formula (regardless of the algorithm chosen) represents the semiology of the disorder, which will establish the definitive diagnosis.

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