REVIEW ARTICLE

Analyzing the Arterial Blood Gases: A Comprehensive Approach

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Abstract

The arterial blood gas analysis provides extensive and crucial information to the intensivist. It indicates the state of alveolar ventilation, the oxygenation, as well as the acid–base balance. It is an invaluable tool in expert hands. This review attempts to clarify the concepts related to respiratory and metabolic acid–base disturbances with several examples.

Keywords: Anion gap, Metabolic acidosis, Metabolic alkalosis, Oxygenation, Respiratory acidosis, Respiratory alkalosis.

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Introduction

Understanding arterial blood gas (ABG) reports is a crucial task in determining the management of patients requiring intensive and continuous monitoring. They give us a deep insight to the oxygenation deficiencies to be differentiated from primary ventilatory deficiencies and primary metabolic acid–base abnormalities. Hence, learning to interpret them in a proper way becomes an essential agenda in any anesthesiologist's learning diary.[1]

The aims of doing a blood gas analysis are to detect the existence and intensity of oxygen- and carbon dioxide-related abnormalities in the blood levels and the degree of compensation in maintaining the acid–base equilibrium, thereby diagnosing the condition and planning their management.

The maintenance of potential hydrogen (pH) equilibrium by the three physiological processes of alveolar ventilation, oxygenation, and acid–base balance is reflected through the ABG report. There is a strong interrelation of all these three processes.

1. Alveolar Ventilation

The maintenance of carbon dioxide (CO_2) levels reflected by arterial CO_2 pressure ($PaCO_2$) depends on the production and excretion of CO_2 via alveolar ventilation by the body. Thus, alveolar ventilation is best assessed by $PaCO_2$. Normal $PaCO_2$ is 35–45 mm Hg.

2. Assessment of Oxygenation Status

Pulse oximetry (SpO_2) and ABG analysis (SaO_2) are useful for measuring oxygen saturation and thereby evaluating the oxygenation status. They determine the arterial oxygen content better than the partial pressure of arterial oxygen (PaO_2). If the saturation shown by a pulse oximeter is less than 90% and the PaO_2 is less than 60 mm Hg, the patient is termed hypoxemic.[2]

Whether the hypoxia occurs due to hypoventilation (due to increase in $PaCO_2$) or due to deficiency in oxygenation can be determined by calculating an alveolar-arterial (A-a) oxygen gradient ($P(A\text{-}a)O_2$).[3] The alveolar-arterial difference in partial pressure of O_2 is $P(A\text{-}a)O_2$. The partial pressure of alveolar oxygen (PAO_2) can be computed using fractional inspired oxygen concentration (FiO_2), partial pressure of arterial CO_2 ($PaCO_2$), and barometric pressure while PaO_2 is measured by ABG.

$PAO_2 = FiO_2$ (P(baro) – P(wv)) – $PaCO_2/R$ = 0.21 (760 – 47) – 40/0.8 = 150–50 = 100 mm Hg

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PaO_2 = 90–100 mm Hg

$P(A\text{-}a)O_2$ is used as an index for shunting and ventilation-perfusion (V/Q) mismatch and is normally about 5–25 mm Hg. It is however affected by age and FiO_2. A-a gradient = Age/4 + 4. So, it increases with increase in the age. For every 10% rise in FiO_2, the A-a gradient rises by 5–7 mm Hg.

The PaO_2/PAO_2 ratio (independent of FiO_2) can also be calculated. Normal values are 90–99/92.6–100 = 0.8–1.0. Grading of this ratio is done as follows: <0.25 is critical shunt, 0.25–0.5 is significant shunt, and 0.5–0.8 is moderate shunt.

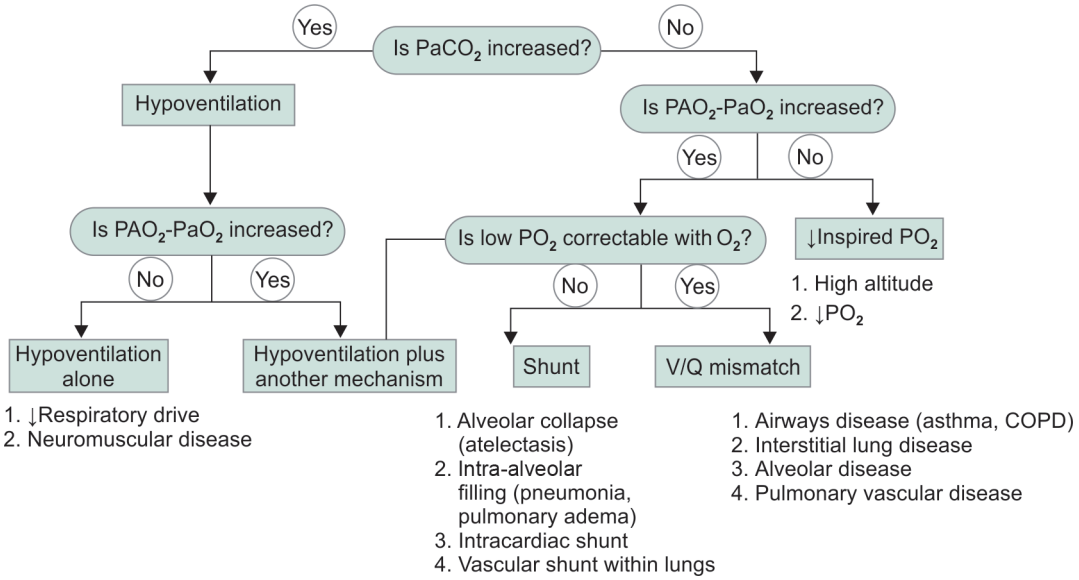
The PaO_2/FiO_2 ratio is important since the PaO_2 value should always be correlated with the amount of oxygen received by the patient. For example, FiO_2 of 20% gives PaO_2 of 100 mm Hg. FiO_2 1.0 gives PaO_2 of 500 mm Hg in normal individuals.

The stepwise evaluation of hypoxemia is shown in Flowchart 1.

3. Acid–Base Balance

Potential hydrogen is defined as negative logarithm to base 10 of hydrogen ions measured in nmol per liter. The pH equilibrium can be maintained by the buffer system of the kidneys, lungs, and blood and is computed by the Henderson-Hasselbalch equation: $pH = 6.1 + \log HCO_3^-/0.3 \times [PaCO_2]$. The Henderson equation has been simplified by Kassirer and Bliech as $H^+ = 24 \times PCO_2/HCO_3^-$.[4] Thus, maintaining pH within range of 7.35–7.45 is the primary objective of regulating acid–base balance. Decrease in arterial pH (pH < 7.35) is acidemia whereas rise in arterial pH (pH > 7.45) points

Flowchart 1: Flow diagram showing approach to hypoxemic respiratory failure



toward alkalemia. Thus, acidosis is defined as process tending to decline the pH by increasing H^+ or eliminating HCO_3^-, whereas the process causing surge in the pH on account of decreasing H^+ or rise of HCO_3^- is termed alkalosis.

Respiratory and Metabolic Conditions

Primary modifications in ventilation lead to respiratory acidosis or alkalosis due to excessive removal or accumulation of CO_2. Metabolic conditions cause acidosis or alkalosis by alterations in the bicarbonate ions concentration.

Compensation

Compensation of primary acid–base disorder is done either by the lungs or kidneys. The respiratory system compensates for the metabolic changes due to deviations in HCO_3^- (metabolic acidosis or alkalosis) while kidneys compensate for fluctuations in CO_2 (respiratory acidosis/alkalosis), which take around 2–5 days.

Simple vs Mixed Acid–Base Disorder

Simple acid–base disorder consists of only a primary single process of either acidosis or alkalosis whereas mixed acid-base disorder may have more than one acid–base disorder coexisting together.

In metabolic conditions, movement of pH and HCO_3^- is in same direction whereas in respiratory conditions the movement of pH and $PaCO_2$ is in opposite direction. In simple disorders, the movement of HCO_3^- and $PaCO_2$ is in same direction while in mixed disorders, fluctuations of HCO_3^- and $PaCO_2$ are in opposite directions (Table 1).[5,6]

While evaluating for the acid–base status, the pH values range between 7.35 and 7.45; $PaCO_2$ ranges between 35 and 45 mm Hg and HCO_3^- values range between 22 and 26 mEq/L. For calculation purposes, the base is taken as 7.40 for pH, $PaCO_2$ is taken as 40 mm Hg, and 24 mEq/L for HCO_3^-.[7,8]

The **stepwise approach to the ABG Analysis**[4] was given by Narins and Emmett and modified by Morganroth in 1991.[9]

Table 1: Features of primary acid–base disorders

Disorder	*Primary responses*		*Compensatory responses*	
Metabolic acidosis	↑ $[H^+]$	↓ pH	↓ HCO_3^-	↓ $PaCO_2$
Metabolic alkalosis	↓ $[H^+]$	↑ pH	↑ HCO_3^-	↑ $PaCO_2$
Respiratory acidosis	↑ $[H^+]$	↓ pH	↑ HCO_3^-	↑ HCO_3^-
Respiratory alkalosis	↓ $[H^+]$	↑ pH	↓ HCO_3^-	↓ HCO_3^-

- Step 1. Evaluate if the patient has alkalosis or acidosis using the arterial pH. Low pH (<7.35) is acidosis, if associated with low HCO_3^- is metabolic acidosis; high $PaCO_2$ is respiratory acidosis. High pH (>7.45) is alkalosis, if associated with high HCO_3^- is metabolic alkalosis. Low $PaCO_2$ is respiratory alkalosis.
- Step 2. Based on $PaCO_2$ and serum HCO_3^- levels, check if there is a primary respiratory or metabolic disruption. pH moves in the same direction as $PaCO_2$/HCO_3^- in case of metabolic conditions. pH moves in the opposite direction as $PaCO_2$/ HCO_3^- in case of respiratory conditions.
- Step 3. In case of a primary respiratory disorder, evaluate whether it is chronic or acute. This can be done by checking $\Delta H^+/\Delta PaCO_2 < 0.3$ indicating chronic condition, >0.8 indicating acute condition, while 0.3–0.8 indicates acute or chronic condition.
- Steps 4 and 5. In metabolic disorders, evaluate if the compensation by the respiratory system is adequate or not. In respiratory disorders, evaluate if compensation by the metabolic system is adequate or not (Table 2).
- Step 6. Check for the oxygenation status (PaO_2 and SaO_2)—if there is hypoxemia or not?
- Step 7A. In case of metabolic acidosis, calculate the anion gap (AG).
  $AG = Na^+ - (Cl^- + HCO_3^-)$ normal < 12 and osmolar gap. In case of severe hypoalbuminemia, AG should be corrected by AGc = AG + {(4-[Albumin]) × 2.5}.
- Step 7B. In high anion gap metabolic acidosis (HAGMA), compute the sum of bicarbonate ions (HCO_3^-) and the change in anion gap (ΔAG) to check for coexisting metabolic imbalances.
  $\Delta AG = [HCO_3^-] + (AG-12)$

Table 2: Compensation of simple acid–base imbalance

Simple acid–base imbalance	*Compensation*	*Formula*
Metabolic acidosis	$PaCO_2$ decreases 1.2 mm Hg per 1.0 mEq/L drop in HCO_3^-	Expected $PaCO_2 = [(1.5 \times HCO_3^-) + 8] \pm 2$ (Winter's formula)
Metabolic alkalosis	$PaCO_2$ rises 0.7 mm Hg per 1.0 mEq/L rise in HCO_3^-	Expected $PaCO_2 = [(0.7 \times HCO_3^-) + 21] \pm 1.5$
Respiratory acidosis	$[HCO_3^-]$ increases	
Acute	$[HCO_3^-]$ increases 1.0 mEq/L per 10 mm Hg rise of $PaCO_2$	$\Delta HCO_3^- = \Delta PaCO_2 \times 0.1 \pm 3$
Chronic (3–5 days)	$[HCO_3^-]$ increases 4 mEq/L per 10 mm Hg rise of $PaCO_2$	$\Delta HCO_3^- = 4\ (\Delta PaCO_2 \times 0.1)$
Respiratory alkalosis	$[HCO_3^-]$ decreases	
Acute	$[HCO_3^-]$ decreases 2.0 mEq/L per 10 mm Hg fall of $PaCO_2$	$\Delta HCO_3^- = 2\ (\Delta PaCO_2 \times 0.1)$
Chronic	$[HCO_3^-]$ decreases 5.0 mEq/L per 10 mm Hg fall of $PaCO_2$	$\Delta HCO_3^- = 5(\Delta PaCO_2 \times 0.1)$

If > 24, consider additional hidden metabolic alkalosis. If < 24, consider a hidden nonanion gap metabolic acidosis.

We can also calculate Delta ratio[10,11] in case of HAGMA:

Delta ratio = Increase in AG/decrease in HCO_3^-

Delta ratio < 1 suggests a greater decrease in HCO_3^- than expected from the change in AG, i.e., additional nonanionic gap metabolic acidosis

Delta ratio ranging between 1 and 2 suggests HAGMA.

Delta ratio > 2 suggests a less decrease in HCO_3^- than expected, i.e., additional metabolic alkalosis

- Step 7C. In case of nonanion gap acidosis, calculate the urinary anion gap that is necessary to differentiate between renal and nonrenal causes.

If compensation is as per calculation, it is a single acid–base disorder. If compensation is less than or greater than calculated expected value, then it is mixed acid–base problem.

Metabolic Acidosis

The pathophysiological causes of metabolic acidosis[11] include: (1) HCO_3^- loss either due to renal or gastrointestinal tract (GIT) causes; (2) greater formation of nonvolatile acids, exogenous acids, lactates, ketoacids, and poisons; and (3) fall in renal acid secretion.

Causes of HAGMA include: (1) lactic acidosis; (2) diabetic ketoacidosis, alcoholic ketoacidosis, and starvation; (3) chronic renal failure; (4) rhabdomyolysis; (5) salicylate toxicity, methanol toxicity, and ethylene glycol toxicity.

Causes of normal AG metabolic acidosis (NAGMA): (1) HCO_3^- loss due to GIT causes—biliary or pancreatic drainage, diarrhea, ketoacidosis during treatment, renal-proximal (type II) renal tubular acidosis (RTA), urinary diversions (ureterosigmoidostomy); (2) reduced renal acid excretion: renal failure, hyperkalemia (type IV) RTA, distal (type I) RTA; (3) miscellaneous: ammonium chloride (NH_4Cl) therapy, HCl therapy (treatment of severe metabolic alkalosis), hyperalimentation, and cholestyramine ingestion.

Diagnosing cause of metabolic acidosis:

Plasma osmolality = $2[Na^+]$ + [Gluc]/18 + [BUN]/2.8. Plasma osmolality gap is defined as the difference between the calculated and measured plasma osmolality.

In HAGMA:

If the plasma osmolality gap is more than 15–20 mOsm/kg, then it shows presence of high osmolar substances like alcohol in the blood.

Ketones and hyperglycemia indicate diabetic ketoacidosis while ketones with normal glucose point to alcoholism and starvation. Serum lactate levels more than 2 indicate lactic acidosis.

In NAGMA:

Urine AG (UAG) is calculated. If positive UAG, then it indicates RTA; urine pH > 5.5 indicates type I RTA. If pH < 5.5 and high K^+, it indicates type IV RTA; if pH < 5.5 and low potassium (K^+), it indicates type II RTA.

If negative UAG, the urine osmolality gap is calculated. If increased it's due to iatrogenic acid gain and if normal it's due to GIT causes.

Example 1: Patient comes with diabetic ketoacidosis at 0 hours at presentation. pH 7.06, $CO_2 = 10.3$, $HCO_3^- = 6.1$, Na^+/Cl^- 142/106

- pH acidemia, primary condition metabolic with pH and HCO_3^- both decreased
- AG = 142 – (106 + 6.1) = 30↑↑ = HAGMA
- Compensation for metabolic acidosis: Winter's formula: Expected $CO_2 = (1.5 \times 6) + 8 = 17$, but actual value 10 < 17, indicating respiratory alkalosis (hyperventilation)
- Delta ratio = AG-12/HCO_3^- -24 = 18/18 = 1, no non-AG acidosis (Cl^- normal)

Thus, patient has HAGMA with respiratory alkalosis.

Same patient after 6 hours of treatment with insulin and saline, pH 7.22, $CO_2 = 24$, $HCO_3^- = 10.2$, Na^+/Cl^- 140/120

- pH = acidemia, primary condition metabolic with pH and HCO_3^- both decreased
- AG = 140–130 = 10, now normal indicating NAGMA
- Metabolic acidosis compensation: Expected $CO_2 = (1.5 \times 10) + 8 = 23$ matches the measured CO_2 (Winter's formula). Thus, compensation is adequate.
- Delta AG = (AG – 12) + measured $HCO_3^- = 2 + 10 = 12 < 24$, indicating hidden NAGMA (Cl^- 120). Delta ratio which is the ratio of difference in AG and bicarbonates = (AG - 12)/(HCO_3^-–24) = 2/14 < 1.

With saline, hyperchloremic acidosis sets in. Thus, the patient now has NAGMA owing to the treatment. Ketoacids are being cleared (AG ↓), thus indicating a good response. Only HCO_3^- will not indicate adequacy of response.

Treatment of metabolic acidosis includes treating underlying cause, HCO_3^- therapy as per the formula 0.5 × weight (kg) × HCO_3^- deficit (mEq/L). The objective is to raise the pH to >7.2 and HCO_3^- > 10 mEq/L.

Metabolic Alkalosis

Metabolic alkalosis is commonly (up to 50% of all disorders) associated with significant mortality.

Pathophysiological causes of metabolic alkalosis are the following:[11]

(1) HCO_3^- accumulation: massive blood transfusion, giving large amounts of HCO_3^-, ingestion (milk-alkali syndrome); (2) H^+ loss: GIT—loss of gastric secretions, chloride losing diarrheal diseases; renal: mineralocorticoid excess, diuretics (loop/thiazide), Bartter's syndrome, high-dose intravenous penicillin, post-chronic hypercapnia, hypercalcemia; (3) H^+ movement into cells—hypokalemia, refeeding. Most in ICU are Cl^- responsive (urinary $Cl^- < 15$ mEq/L) due to vomiting, nasogastric suction, diuretics, laxative, and volume depletion. Cl^- resistant (urinary $Cl^- > 25$ mEq/L) due to severe hypokalemia and high aldosterone.

Example 2: A 70-year-old male with history of poor oral intake associated with areflexia and weakness. pH 7.54↑, CO_2 54↑, HCO_3^- 44, $Na^+/K^+/Cl^-$ 145/2.1/86

- pH = alkalemia, condition being metabolic since both pH and HCO_3^- high
- AG = 145 − (86 + 44) = 15 normal
- Expected $CO_2 = 0.7 \times HCO_3^- + 21 \pm 2 = 0.7 \times 44 + 21 \pm 2 = 51.8 \pm 2$, which is seen as per reading.
- Diagnosis is severe metabolic alkalosis due to severe hypokalemia ($K^+ = 1.9$).

Treatment: Immediate goal of therapy is partial correction of the alkalosis. The objective is reducing pH less than 7.55 and decreasing HCO_3^- less than 40 mEq/L.

Cl^- responsive type metabolic alkalosis is the most severe form. Treating the causes responsible for chloride and volume depletion. Administration of 3–5 L of 150 mEq/L sodium chloride should be done in patients with established signs of volume contraction. In case of chloride-resistant metabolic alkalosis, chloride should be repleted as potassium chloride. For metabolic alkalosis due to mineralocorticoid excess, spironolactone, which is a K^+-sparing diuretic, proves beneficial.

Respiratory Acidosis

Acute respiratory acidosis is caused by obstruction in elimination of CO_2: (1) ventilation-related causes, airway obstruction like aspiration, bronchospasm (severe), laryngospasm; pulmonary edema; (2) perfusion-related causes: cardiac arrest, pulmonary thromboembolism; (3) restricted chest wall: tension pneumothorax, hemothorax, flail chest; (4) musculoskeletal system: myasthenia gravis crisis, hypokalemia; (5) failure of mechanical ventilator and other problems like (a) central nervous system (CNS): trauma, stroke, drugs (anesthetics, sedatives); (b) peripheral nerves: neurotoxins (OPC, tetanus, botulism), cervical cord injury, skeletal muscle relaxants (succinyl choline, curare, pancuronium and allied drugs, aminoglycosides)

Causes of chronic respiratory acidosis are obstruction in elimination of CO_2 like (1) ventilation: interstitial lung disease, chronic obstructive pulmonary disease; (2) restricted chest wall movement: muscular dystrophy, polymyositis, etc., and (1) CNS: chronic trauma, obesity hypoventilation syndrome, brain malignancies, myxoedema, (2) peripheral nerves: diaphragmatic paralysis, poliomyelitis, multiple Sclerosis.

Example 3: A 21-year-old female presented with history of dyspnea and wheezing for 5 days. She was tachypneic with central cyanosis and bilateral wheezing present. Chest X-ray showed tubular heart with hyperinflated lung fields. pH = 7.31, CO_2 = 64, HCO_3^- = 30, PaO_2 = 68 mm Hg, Na^+/Cl^- = 136/98

- pH = 7.31 with increased CO_2, hence dominant disorder is respiratory acidosis. Condition is chronic due to 5 days.
- AG = 136 − (98 + 30) = 8, which is normal.
- Compensation = $\Delta HCO_3^- = 0.4 \times \Delta PaCO_2 = 0.4 \times 24 = 9.6$, HCO_3^- = 24 + 9.6 = 33. 2 compensation is adequate.

The diagnosis is chronic respiratory acidosis.

Treatment of respiratory acidosis is correction of underlying cause, appropriate oxygenation preventing subduing of respiratory drive, and avoid rapid correction of increased $PaCO_2$ to avoid post-hypercapnic metabolic alkalosis (seizures, arrhythmias).

Respiratory Alkalosis

Causes of respiratory alkalosis comprises of head trauma, brain tumor, cerebrovascular accidents and other causes like pain, anxiety, fever, and peripheral respiratory stimulation like pulmonary V/Q imbalance, pulmonary shunts, hypovolemia, heights, diffusion problems, and miscellaneous causes like drugs—NSAIDs, progesterone, thyroid hormone, catecholamines, gram–ve sepsis, pregnancy, heat exposure, mechanical ventilation.

Example 4: 15-year-old boy presented with complaints of difficulty in breathing and upper abdominal discomfort for 3 hours. On examination per abdomen and respiratory system are normal except for hyperventilation. pH = 7.5, $PaCO_2$ = 26 mm Hg, HCO_3^- = 20 mEq, PaO_2 = 100 mm Hg, Na^+/Cl^- = 137/99.

- pH = 7.5 with decreased CO_2, hence dominant disorder is respiratory alkalosis.
- AG = 137 − (99 + 20) = 18, which is raised. AG is slightly raised in respiratory alkalosis. Since it occurred only 3 hours back, it is an acute respiratory alkalosis.
- Compensation $\Delta HCO_3 = 0.2 \times \Delta PaCO_2 = 0.2 \times 14 = 2.8$, HCO_3^- = 24 − 2.8 = 21.2; compensation is adequate. Diagnosis is acute respiratory alkalosis.

Treatment of respiratory alkalosis consists of treatment of underlying cause, administration of O_2. If pH > 7.55, the patient is sedated and paralyzed and put on ventilator.

In Mixed Acid–base Disorders

Step 1. Determine the primary component as explained previously depending on the direction of pH, pCO_2, HCO_3^-.

Step 2. Calculate AG = Na − (Cl + HCO_3), $n = 12 \pm 4$.

Step 3A. Calculate expected CO_2 if metabolic.

Metabolic acidosis: $CO_2 = (1.5 \times HCO_3^-) + 8$

Metabolic alkalosis: $\Delta CO_2 = 0.7 \times \Delta HCO_3^-$

3B. Calculate expected HCO_3^- if respiratory

$\Delta HCO_3^- = 0.1 \times \Delta CO_2$ (respiratory acidosis)

$0.2 \times \Delta CO_2$ (if respiratory alkalosis)

$0.4 \times \Delta CO_2$ (if chronic respiratory acid/alkalosis)

Step 4. Calculate delta ratio = (AG – 12)/(HCO_3 – 24)

Examples of mixed acid–base disorders

Example 5: A 68-year-old male taking diuretics for congestive heart failure admitted for lobar pneumonia: pH 7.62↑, CO_2 32↓, HCO_3^- = 34↑, Na^+/K^+ 142/96

- pH = Primary alkalemia, metabolic alkalosis due to high HCO_3^-
- AG = 142 – 129 = 13 normal
- Since metabolic is primary check for respiratory compensation
- Expected $CO_2 = 0.7 \times HCO_3^- + 21 = 0.7 \times 9 + 21 \pm 2 = 44.8 \pm 2$
- But actual CO_2 32 << 44.8 indicates that there is also associated respiratory alkalosis (hyperventilation)
- Diagnosis is metabolic alkalosis (loss of K^+ and Cl^- due to diuretics) + respiratory alkalosis

Example 6: A 60-year-old male, known case of hypertension, underwent emergency abdominal exploration and he suffered intraoperative infarct and arrest pH 7.27 ↓, CO_2 66↑, HCO_3^- = 20 Na^+/K^+ = 138/102

- pH = acidemia along with increased CO_2 levels indicating respiratory acidosis
- AG = 138 – (102 + 20) = 16 normal. Since, it was emergency abdominal exploration, the changes were acute.
- $\Delta HCO_3^- = 0.1 \times \Delta\ CO_2 = 0.1 \times 26 = 2.6$

Expected $HCO_3^- = 24 + 2.6 = 26.6$ but actual $20 < 26$ indicating metabolic acidosis developing due to poor circulation. Hence diagnosis is post-arrest respiratory acidosis and developing metabolic acidosis.

Example 7: A 75-year-old male with severe abdominal pain and shock, pH 7.35, CO_2 22, HCO_3^- = 14↓↓, $Na^+/K^+/Cl^-$ 137/4.9/107

- pH tends to acidemia = metabolic acidosis due to low HCO_3^-
- AG = (137+5) – (107 + 13) = 12 (NAGMA)
- Respiratory compensation Winter formula CO_2 $(1.5 \times 14) + 8 = 29$

but actual CO_2 $22 < 29$, there is overcompensation of ventilation resulting in respiratory alkalosis.

- Urine AG and Urine Osmolarity Gap needs to be calculated.

Thus, the diagnosis is non-anion gap metabolic acidosis secondary to shock + respiratory alkalosis secondary to hyperventilation due to pain.

Example 8: A 45-year-old male known case of chronic renal failure admitted to the intensive care unit with severe vomiting pH 7.40, $PaCO_2$ = 42, HCO_3^- = 22, $Na^+/K^+/Cl^-$ 150/3.8/99, serum creatinine 8.7

- pH = normal
- AG = 150 – (99 + 22) = 29↑↑ (HAGMA)
- Respiratory compensation $CO_2 = (1.5 \times 24) + 8 = 44$, compensation is adequate

ΔRatio = (AG – 12)/(HCO_3^- – 24) = 15/2 >2—presence of metabolic alkalosis. Thus, the diagnosis is HAGMA (uremic) + M alkalosis (vomiting), thus normalizing the pH values.

Conclusion

Thus, we conclude that knowledge about the triad of oxygenation, ventilation, and acid–base status helps us in thorough analysis of ABG. However, ABG results are only meaningful when inferred taking into account patients' symptomatology, pathophysiology, and treatment in details. Hence, emphasis is to treat the patient and not the ABG.

References

1. Gattinoni L, Pesenti A, Matthay M. Understanding blood gas analysis. Intensive Care Med 2018;44(1):91–93. DOI: 10.1007/s00134-017-4824-y.
2. Barthwal MS. Analysis of arterial blood gases - a comprehensive approach. J Associat Physic India 2004;52:573–577.
3. Castro D, Keenaghan M. Arterial blood gas. [updated 2020 mar 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020.
4. Rao SM, Nagendranath V. Arterial blood gas monitoring. Indian J Anaesth 2002;46(4):289–297.
5. Sood P, Paul G, Puri S. Interpretation of arterial blood gas. Indian J Crit Care Med 2010;14(2):57–64. DOI: 10.4103/0972-5229.68215.
6. Buche V. Arterial blood gases: a simplified bedside approach. J Neonat Biol 2014;3(04):153. DOI: 10.4172/2167-0897.1000153.
7. Cowley NJ, Owen A, Bion JF. Interpreting arterial blood gas results. Br Med J 2013;346(jan16 1):f16. DOI: 10.1136/bmj.f16.
8. Larkin BG, Zimmanck RJ. Interpreting arterial blood Gases Successfully. Associat Reg Nurs Jl 2015;102(4):343–354. DOI: 10.1016/j.aorn.2015.08.002; quiz 355-7.
9. Morganroth ML. Six steps to acid-base analysis: clinical applications. J Crit Ill 1990;5(5):460–469.
10. Salem MM, Mujais SK. Gaps in the anion gap. Arch Intern Med 1992;152(8):1625–1629. DOI: 10.1001/archinte.1992.00400200063011.
11. Black RM. Metabolic acidosis and metabolic alkalosis. In: Irwin RS, Rippe JM. Intensive Care Medicine. 5th ed., Philadelphia: Lippincott Williams Wilkins; 2003. 852–864.