

Interpretation of Arterial Blood Gas Analysis

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Introduction

Blood gas analysis (arterial and/or venous) is a routine test and commonly used monitoring modality. Blood gas has vast information; when this information is interpreted with background clinical condition it helps in diagnosis

Indications for Arterial Blood Gas (ABG)

- Severe respiratory or metabolic disorders
- Clinical features of hypoxia or hypercarbia
- Shock
- Sepsis
- Decreased cardiac output
- Renal failure
- Multiorgan dysfunction
- Diabetic ketoacidosis

Samples Collection and Transport

Ideal artery for sampling is radial. One must perform “Allen Test” to ensure collateral blood supply by ulnar artery before puncturing radial artery (Table 1).

Indwelling arterial line of radial or femoral artery can be used for sample collection.

Table 1.

Comparison of blood gas analysis at different sites

Variables	Arterial	Venous
pH	Same	Lower
PaCO ₂	Lower	Higher
PaO ₂	Higher	Lower
HCO ₃	Same	Same

Precautions for collection of blood sample

1. Heparin is acidic and lowers pH. Use heparin of lower strength (1000 instead of 5000 units per ml) or heparin solution.
2. Use small volume of heparinised saline just for lubricating syringe and plunger. If volume is more, dissolved oxygen in heparinised saline may increase PaO₂.
3. Avoid air bubble and let syringe fill spontaneously.
4. It is desirable to use a glass syringe as plastic syringes are permeable to air.
5. The sample should be processed immediately, preferably within 30 minutes. Blood is a living medium. The cells consume oxygen and produce CO₂. Drop in PaO₂ depends on initial PaO₂. If the latter is very high, significant drop may be noticed. The changes are as depicted in Table 2. Slush of ice (not cubes) should be used for storing samples till processing. The sample should be shaken before putting in machine (Fig. 1).

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Figure 1. Arterial blood gas analysis machine.

Table 2.

Changes in ABG every 10 minutes *in vitro*

	37°C	4°C
pH	0.01	0.001
PaCO ₂	0.1 mmHg	0.01 mmHg
PaO ₂	0.1 mmHg	0.01 mmHg

For minimal error, blood sample should be stored at 4°C, if it cannot be processed immediately for minimal error.

Terminology and Normal ABGs

Terminology

Acidosis pH <7.35

Alkalosis pH >7.45

Acidemia and alkalemia refer to blood while acidosis, alkalosis to tissue pH.

THb

THb is total hemoglobin of patient. Few machines measure hemoglobin, others need this information to be fed. Hemoglobin is required to calculate oxygen content (O₂CT) of blood.

Temp

Patient temperature has to be fed into machine because the machine measures all values at 37°C. Temperature

affects pH, PaCO₂ and PaO₂. Hence, it is desirable to have values corrected for patient temperature.

BE, St BE(SBE), BB

Base excess (BE) refers to actual base excess in variance from (above or below) total buffer base (BB). Normal BB is 48–49 mmol/l. If BB is 40, it means buffer base is reduced by nearly 8 mmol/l, or BE is –8 (also called base deficit). If BB is 60, it means buffer base is increased by nearly 12 mmol/l, or BE is +12.

Standard base excess (SBE) is the BE adjusted for temperature of 30°C and PaCO₂ of 40 mmHg.

BB is dependent on hemoglobin, as 25% of BB is constituted by hemoglobin buffer. Fifty percent of BB is contributed by bicarbonate and 25% by other buffers (proteins, phosphate, sulfate).

HCO₃⁻; St HCO₃⁻ (SBC); TCO₂

TCO₂ is sum of HCO₃⁻ and amount of CO₂ dissolved in plasma. For each mmHg PaCO₂, 0.03 ml CO₂ is dissolved per 100 ml of plasma. As HCO₃⁻ values change with CO₂ levels, standard bicarbonate (st HCO₃⁻) is used to denote value of HCO₃⁻, independent of CO₂ changes (i.e., at PaCO₂ of 40 and temperature of 37°C).

St. pH

Standard pH (st. pH) is the pH adjusted for temperature of 37°C and PaCO₂ of 40 mmHg. This would represent pH value purely due to metabolic status.

[H⁺]

It is the concentration of hydrogen ions in nmol/l at 37°C patient's temperature.

$$[H^+]_{nEq/l} = 24 \times (PaCO_2 / HCO_3^-)$$

O₂CT

It is the sum of oxygen bound to hemoglobin and oxygen dissolved in plasma. For each gram saturated Hb, 1.34 ml O₂ is bound to hemoglobin and for each mmHg PaO₂ 0.003 ml oxygen is dissolved per 100 ml of plasma.

$$O_2CT = 1.34 \times Hb \times SaO_2 + 0.003 PaO_2$$

SaO₂ sat

It is the proportion/percentage of arterial hemoglobin which is saturated with oxygen.

AaDO₂

This refers to the alveolar-to-arterial oxygen gradient. Normal value is 5–15 mmHg.

RQ

Respiratory quotient (RQ) is the amount of CO₂ liberated per minute divided by amount of O₂ utilized per minute. Normal values are 200 ml/250 ml = 0.8.

FiO₂

It is inspired oxygen fraction (FiO₂) concentration. This value has to be fed to machine; it is required for calculation of alveolar oxygen concentration.

Details about pH

$pH = pK + \log_{10} (HCO_3^- / H_2CO_3)$ (Henderson–Hasselbach equation)

Normal pH = 7.35 – 7.45

Types of Acid-base Disorder

- Metabolic acidosis
- Metabolic alkalosis
- Acute respiratory acidosis
- Chronic Respiratory acidosis
- Acute respiratory alkalosis
- Chronic Respiratory alkalosis

Metabolic acidosis

Metabolic acidosis is a process that causes primary decrease in plasma bicarbonate concentration. This can be due to gain in acid or loss of bicarbonate (Table 3).

Types of metabolic acidosis are as follows:

- A. High anion gap (AG)
- B. Non-AG acidosis or normal AG acidosis

High-AG acidosis

High-AG acidosis results from production of endogenous acid. It results from addition of hydrogen ion and an unmeasured anion in the blood. The hydrogen ions are buffered by bicarbonate causing decrease in its concentration.

Anion gap = Unmeasured anions – Unmeasured cations

$AG = Na - (Cl + HCO_3^-)$

Normal AG = 12 ± 2 mEq/l

Reference AG is influenced by albumin concentration in blood.

Adjusted AG = Observed AG + 2.5 [4.0 – measured albumin (g/dl)]

If an elevated AG metabolic acidosis is present, the “delta–delta ratio” should be calculated to determine if a second metabolic disorder is present.

The delta–delta calculation compares the deviation from normal of the AG with that of HCO₃⁻ (normal [HCO₃⁻] ≈ 22–26 mEq/l). In a simple AG acidosis, these values would be expected to roughly equal one another (HCO₃⁻ decreasing by one unit for every unit the AG increases); however because not all H⁺ is buffered by HCO₃⁻, the Δ in the AG usually exceeds the Δ in HCO₃⁻.

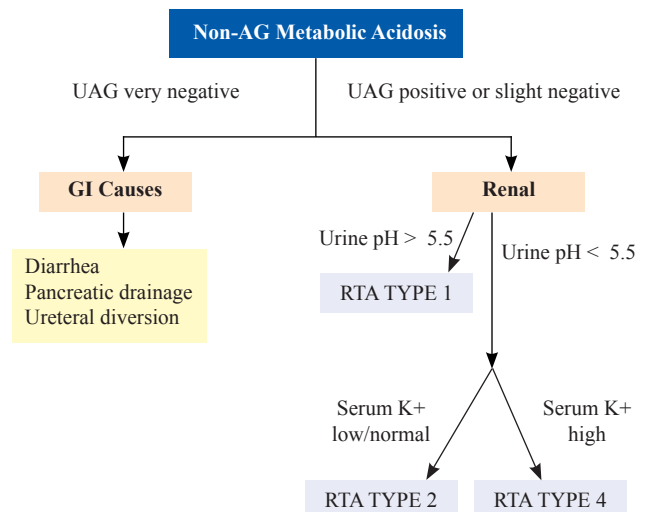
If ΔAG/Δ HCO₃⁻ <1, an elevated gap metabolic acidosis and a normal gap acidosis are both present.

If ΔAG/Δ HCO₃⁻ = 1–2, a simple elevated gap metabolic acidosis is present.

If ΔAG/Δ HCO₃⁻ >2, an elevated gap metabolic acidosis and a metabolic alkalosis are both present.

Non-AG acidosis or normal AG acidosis

It is also known as hyperchloremic acidosis and drop in bicarbonate is the primary pathology. Sometime increased chloride can cause non-AG acidosis.



Urinary AG = (Urine Na⁺ + Urine K⁺) – Urine Cl⁻

Table 3.

Causes of metabolic acidosis

High-AG acidosis	Normal AG acidosis
M - Methanol	F - Fistula
U - Uremia	U - Uretero-enterostomy
D - Diabetic ketoacidosis	S - Saline administration (large volume)
P - Paraldehyde/propylene glycol	E - Endocrine (hyperparathyroidism)
I - Isoniazide	D - Diarrhea
L - Lactic acidosis	C - Carbonic anhydrase inhibitors (acetazolamide)
E - Ethylene glycol	A - Ammonium chloride
S - Salicylate intoxication	R - Renal tubular acidosis (type 1,2,4)
R - Renal failure (MUDPILES)	S - Spironolactone (FUSED CARS)

In compensated metabolic acidosis, there are PaCO₂ less than 30, low HCO₃⁻, with a pH of 7.3–7.4.

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

In uncompensated metabolic acidosis, the following conditions prevail:

- Normal PaCO₂, low HCO₃⁻ and a pH less than 7.30
- Occurs as a result of increased production of acids and/or failure to eliminate these acids

Respiratory system is not compensating by increasing alveolar ventilation (hyperventilation).

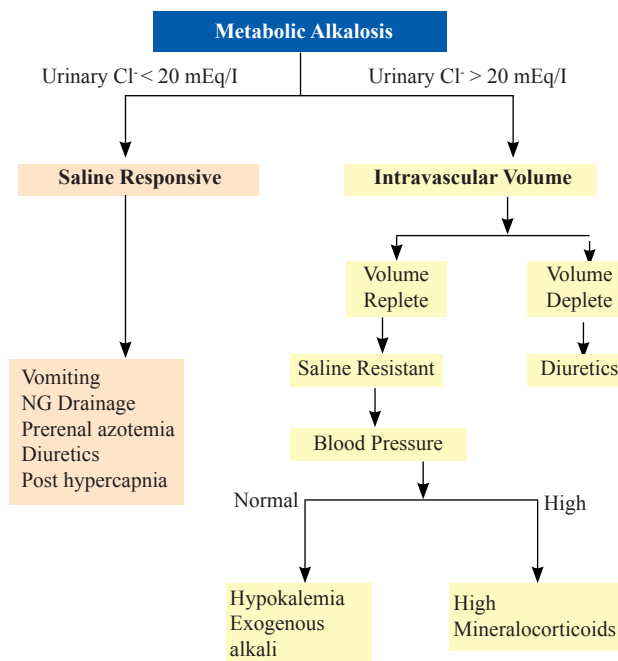
Metabolic alkalosis

Metabolic alkalosis is a process that causes primarily increase in HCO₃⁻ concentration. It can be either generated by loss of hydrogen ion or gain of HCO₃⁻ and compensated by respiratory system.

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

Causes of metabolic alkalosis are as follows:

- Extracellular fluid volume depletion
- Severe potassium depletion
- Mineralocorticoid excess syndrome



Acute respiratory acidosis

- PaCO₂ is elevated and pH is acidotic
- The decrease in pH is accounted for entirely by the increase in PaCO₂
- Bicarbonate and base excess will be in the normal range because the kidneys have not had adequate time to establish effective compensatory mechanisms

Causes are as follows:

- Respiratory pathophysiology (airway obstruction, severe pneumonia, chest trauma/pneumothorax)
- Acute drug intoxication (narcotics, sedatives)
- Residual neuromuscular blockade
- Central nervous system disease (head trauma)

Chronic respiratory acidosis

- PaCO₂ is elevated with a pH in the acceptable range
- Renal mechanisms increase the excretion of H⁺ within 24 hours and may correct the resulting acidosis caused by chronic retention of CO₂ to a certain extent

Causes are as follows:

- Chronic lung disease (e.g., chronic obstructive pulmonary disease)
- Neuromuscular disease
- Extreme obesity
- Chest wall deformity

Acute respiratory alkalosis

- PaCO₂ is low and the pH is alkalotic
- The increase in pH is accounted for entirely by the decrease in PaCO₂
- Bicarbonate and base excess will be in the normal range because the kidneys have not had sufficient time to establish effective compensatory mechanisms

Causes are as follows:

- Pain
- Anxiety
- Hypoxemia
- Restrictive lung disease
- Severe congestive heart failure
- Pulmonary emboli
- Drugs
- Sepsis
- Fever
- Thyrotoxicosis
- Pregnancy
- Overaggressive mechanical ventilation
- Hepatic failure

Chronic respiratory alkalosis

In chronic respiratory alkalosis, metabolic component compensates almost completely. Therefore there will be little change in pH.

Oxygenation Assessment

A–a gradient. The alveolar–arterial oxygenation gradient

is the difference between P_AO₂ and P_aO₂ [a normal A–a gradient ≈ (age in years + 10)/4]. The source of this normal gradient is a physiological shunt due to bronchial blood flow (which bypasses the alveoli and is therefore not oxygenated) and a small portion of coronary venous blood that drains directly into the left ventricle via the thebesian veins.

$$A-a \text{ oxygen ratio} = P_aO_2 / P_AO_2$$

Normal range > 0.77

$$P_AO_2 = FIO_2 \times 713 - (P_aCO_2/RQ)$$

It is used to approximate the change expected in P_aO₂ for a given increase in F_IO₂.

P_aO₂/F_IO₂ ratio: Normal range = 300–500;

Gas exchange derangement = 200–300;

Severe hypoxia < 200.

ABG Interpretation – Steps

First, does the patient have an acidosis or an alkalosis?

Second, what is the primary problem – metabolic or respiratory?

Third, is there any compensation by the patient?

Respiratory compensation is immediate while renal compensation takes time.

Normal values

pH= 7.35–7.45	(Reference value 7.4)
PaCO ₂ = 35–45 mmHg	(Reference value 40)
HCO ₃ ⁻ = 22–26 mEq/l	(Reference value 24)

Abnormal values

pH < 7.35	Acidosis (metabolic and/or respiratory)
pH > 7.45	Alkalosis (metabolic and/or respiratory)
PaCO ₂ > 45 mmHg	Respiratory acidosis (alveolar hypoventilation)
PaCO ₂ < 35 mmHg	Respiratory alkalosis (alveolar hyperventilation)
HCO ₃ ⁻ < 22 mEq/l	Metabolic acidosis
HCO ₃ ⁻ > 26 mEq/l	Metabolic alkalosis

Putting it together:

Respiratory

- PaCO₂ > 45 with a pH < 7.35 represents a respiratory acidosis

- PaCO₂ < 35 with a pH > 7.45 represents a respiratory alkalosis
- For a primary respiratory problem, pH and PaCO₂ move in the opposite direction
 - For each deviation in PaCO₂ of 10 mmHg in either direction, 0.08 pH units change in the opposite direction

Metabolic

- HCO₃ < 22 with a pH < 7.35 represents a metabolic acidosis
- HCO₃ > 26 with a pH > 7.45 represents a metabolic alkalosis
- For a primary metabolic problem, pH and HCO₃- are in the same direction, and PaCO₂ is also in the same direction
- Fourth, look for compensation
 - The body's attempt to return the acid/base status to normal (i.e., pH closer to 7.4)

Primary Problem	Compensation
Respiratory acidosis	Metabolic alkalosis
Respiratory alkalosis	Metabolic acidosis
Metabolic acidosis	Respiratory alkalosis
Metabolic alkalosis	Respiratory acidosis

Expected Compensation

Respiratory acidosis (1/4)

- Acute – the pH decreases 0.008 units for every 1 mm Hg increase in PaCO₂; HCO₃-1 mEq/l per 10 mm Hg PaCO₂
- Chronic – the pH decreases 0.003 units for every 1 mm Hg increase in PaCO₂; HCO₃-4 mEq/l per 10 mm Hg PaCO₂

Expected compensation

Respiratory alkalosis (2/5)

- Acute – the pH increases 0.008 units for every 1 mmHg decrease in PaCO₂; HCO₃- 2mEq/l per 10 mmHg PaCO₂
- Chronic – the pH increases 0.0017 units for every 1 mmHg decrease in PaCO₂; HCO₃- 5 mEq/l per 10 mmHg PaCO₂

Expected Compensation

Metabolic acidosis

- Expected PaCO₂ = 1.5(HCO₃) + 8 (±2)
- PaCO₂ 1-1.5 per 1 mEq/l HCO₃

Metabolic alkalosis

- Expected PaCO₂ = 0.7(HCO₃) + 21 (±2)
- PaCO₂ 0.5–1.0 per 1 mEq/l HCO₃

RADIOMETER ABL800 BASIC			
ABL800 Basic		07:59 AM	11/25/2013
Patient Report	Syringe - S 195 uL	Sample #	77208
Identifications			
Patient ID			
Patient Last Name			
Sex	Male		
Sample type	Arterial		
FO ₂ (I)	21.0%		
T	37.0% ^c		
pH	7.407		[7.350 - 7.450]
pCO ₂	41.1	mmHg	[35.0 - 45.0]
pO ₂	104	mmHg	[83.0 - 108]
cHCO ₃ ⁻ (P.st) ^c	25.5	g/dL	
↓ ctHb	9.2	g/dL	[12.0 - 16.0]
sO ₂	98.1	%	[95.0 - 99.0]
cK ⁺	4.1	mmol/L	[3.5 - 4.5]
↑ ctHb	139	mg/dL	[70.0 - 105.0]
↓ cLac	0.5	mmol/L	[0.5 - 1.6]
↓ cNa ⁺	124	mmol/L	[135 - 145]
↓ cCl ⁻	95	mmol/L	[98 - 106]
↓ cCa ²⁺	1.06	mmol/L	[1.15 - 1.29]
ABEc	1.2	mmol/L	
Calculated Values			
cCa ²⁺ (7.4) ^c	1.07	mmol/L	
ctCo ₂ (P) ^c	59.7	vol%	
Anion Gap ^c	3.1	mmol/L	
Anion Gap, K ⁺ ^c	7.1	mmol/L	
Hct ^c	28.6	%	
pH(st) ^c	7.416		
p50 ^e	26.44	mmHg	
pO ₂ (A-a) ^e	---	mmHg	
pO ₂ (a/A) ^e	104.6	%	
Rl ^e	-4	%	
mOsm ^c	255.2	mmol/kg	
Temperature Corrected Values			
pH(T)	7.407		
pCO ₂ (T)	41.1	mmHg	
pO ₂ (T)	104	mmHg	
Notes			
↑	Value(s) above reference range		
↓	Value(s) below reference range		
c	Calculated value(s)		
e	Estimated value(s)		

Figure 2. A typical arterial blood gas analysis report.

References

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Picture courtesy: Rachna Kasliwal