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Analysis of Arterial Blood Gas & Its Interpretation in Critical Care Patients

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Abstract
Arterial blood gas is a basic investigative tool in monitoring acid base balance of critically ill patients. It is also indispensible in monitoring gas exchange, diagnosing and managing oxygenation. A thorough knowledge regarding ABG interpretation is an absolute necessity for the critical care physician. However, a complete and thorough understanding of arterial blood gases and its interpretation is an arduous task and the literature to guide the interpretation are aplenty and beyond the scope of this article. Thus, this article tries to portray a systematic, realistic and authentic approach for the arterial blood gas analysis and its interpretation right from preparation of the patient to sample collection and reading of ABG. This requires a proper application of the basic concepts of acid base interpretation to appreciate the progress of the management apart from aiding in the diagnosis.

Key Words: *Arterial blood gas (ABG)*

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I. Introduction

Arterial blood gas (ABG) is one of the most basic yet absolutely essential diagnostic tools for the diagnosis and management of critically ill patients. It is also quintessential in assessing oxygenation and acid base status. The usefulness of ABG relies solely on accurate interpretation of the results. Critical care patients include those with respiratory system disorders like COPD, asthma, cardiovascular disorders and more importantly renal and liver disorders. The renal system is of particular importance as it handles the body's acid base system, metabolism of drugs and their excretion. Also, an acid base disorder can occasionally be life threatening, which makes ABG an indispensable tool in critical care management. This article aims to simplify the analysis of ABG which includes part preparation, correct method of sample collection and interpretation[1].

Approaches for ABG interpretation:
There are many approaches for interpretation of an ABG. The most commonly used parameters include
1. Plasma bicarbonate (HCO3)
2. pCO2
3. Standard base excess (SBE)
4. Strong ion difference (SID)

Physiology of acid base balance:
The normal body pH ranges from 7.35-7.45
Maintaining the pH within this narrow range is absolutely essential for maintaining homeostasis.
If pH < 7.35→ the condition is acidosis
pH> 7.45→ the condition is alkalosis
Types of acid base disorders:
There are broadly 4 types of acid base disorders.
They are
1. Metabolic acidosis
2. Metabolic alkalosis
3. Respiratory acidosis
4. Respiratory alkalosis

They can be further divided as
i. Compensated
ii. Uncompensated

Most importantly, even in the presence of compensation, there will never be over correction.
A few basic points to note:

- CO2 is a respiratory component and HCO3 is metabolic component
- If CO2 moves in a direction opposite to pH (pH , PaCO2) it is respiratory acidosis
- If HCO3 moves in the same direction as pH (pH , HCO3) it is metabolic alkalosis
- In primary disorder HCO3 and pCO2 move in the same direction
- In mixed disorder HCO3 and pCO2 move in the opposite direction

Causes of acid base disorders:

1. Metabolic acidosis (pH , HCO3)
- DKA
- Acute/ chronic renal failure
- Lactic acidosis (sepsis , haemorrhage, shock)
- Sever diarrhoea
- Intestinal fistula

2. Metabolic alkalosis (pH, HCO3)
- Use of diuretics
- Prolonged vomiting
- Massive blood transfusions
- Antacid ingestion

3. Respiratory acidosis – any condition causing retention of CO2 like
- COAD
- Bronchial asthma
- Respiratory depression due to drug/ head injury
- Foreign body in bronchus

4. Respiratory alkalosis – any condition causing CO2 washout (hyperventilation) such as

-pain[6]
-fever
- pregnancy
- hypermetabolic state (sepsis)
- anxiety, fear

Blood gas analysis helps in:

- Establishing diagnosis
- Planning management and treatment
- Optional functioning of medications (some drugs work in acidic and some in alkaline medium)
- To maintain electrolyte balance
- Maintain of homeostasis

Limitations of ABG:

Though an invaluable tool in critical care management, ABG has its own limitations:

- It is not used for initial diagnosis, it helps in confirming an established diagnosis
- Degree of abnormality is not equal to actual degree of damage that has occurred in the body
- Prone for a legion of errors

Errors that can occur in ABG[2]

Various errors that can lead to misinterpretation of an ABG and more importantly missing a crucial diagnosis are

I. Pre analytical errors :
- missing or wrong patient or sample identification
- using inappropriate amount of anticoagulation
- using incorrect type of anticoagulation

II. Sampling errors:
- Venous sampling
- Mixed (arterial/ venous) sampling
- Insufficient mixing with heparin

III. **Post sampling errors**:

- Air bubbles in sample
- Cooling
- Storing sample for too long
- Wrong sample identification
- Clot in sample
- Inadequate cooling (>30 min delay is expected)
- Inadequate mixing of sample (the recommended procedure is to mix the blood sample thoroughly by rolling it in palms after inverting it 10 times to prevent rouleaux formation)

Anticoagulation used in ABG analysis:

Heparin is the most commonly used anticoagulant. Various types of heparin that can be used include:

- Liquid non balanced heparin
- Dry non balanced heparin
- Dry electrolyte balanced (Na+ K+)
- Dry Ca2+ balanced[3]

The correct procedure with liquid heparin:

Liquid heparin causes plasma dilution and hence pCO2 and electrolytes are affected. But HCO3 remains unaffected. Also, 0.05 ml heparin is adequate to anticoagulate 1 ml of blood. The standard dead space volume of a 5ml syringe with 1 inch 22 gauge needle is 0.2ml. So the amount of heparin in the dead space is adequate to anticoagulated 4ml of arterial blood sample. If the sample is less than 4ml, it results in overdilution and if more than 4ml, it results in underdilution. The importance of proper dilution is that plasma electrolytes vary linearly with dilution[4].

Also heparin binds to cations (Na+, K+, Ca2+) and lower their value significantly. The usage of electrolyte balanced heparin significantly reduces the effect of binding, thus preventing inaccuracies[5].

Where to obtain the sample:

The preferable site is the radial artery. It is superficial and has good collateral supply. Brachial and femoral arteries are also used but less commonly.

Tests done before obtaining the sample:

Modified Allen's test

- The patient is asked to make a fist using the middle and index fingers of both hands, pressure is applied on the wrist, compressing radial and ulnar arteries at the same time.
- Now ask the patient to open the hand slowly.
- The pressure over the ulnar artery is released
- In case of Allen's test positive: the hand flushes pink in less than 15 seconds
- In case of Allen's test negative: the hand does not get flushed or returns to normal colour
- Negative Allen's test indicates absence or atherosclerosis of collaterals and radial artery should not be used.

Sampling:

The arm of the patient is placed palm up and wrist is dorsiflexed 45 degrees on a flat surface. Regular aseptic precautions are taken. 2% lignocaine (local anaesthetic) is infiltrated. The pre heparinised syringe with 25 gauge needle is inserted at an angle of 45 degrees just distal to the radial pulse. Once adequate amount of sample is withdrawn, a sterile gauze should be placed firmly on the site and direct pressure is applied for atleast 5-8 minutes to achieve haemostasis.

Precautions before sampling:

- A steady state should always be allowed after initiation or a change in oxygen therapy.

Especially in patients with COAD/ asthma, it takes 20- 30 minutes and in patients without overt respiratory disease it takes 3-10 minutes.

- Heparin should be used accurately as sample may get over / under diluted.
- Delay in sample processing or transport should be avoided. If delayed, proper cooling protocol should be followed.
- Also, avoid air bubbles in the syringe.

Steps involved in interpretation of ABG values:

Step 1: The primary diagnosis should always be acted upon.

Step 2: pH

If pH <7.35 – acidosis
>7.45 – alkalosis
7.40 – Normal / mixed / fully compensated
Step 3: Check Sa02/ PaO2
PaO2 is partial pressure of oxygen in plasma and SaO2 is saturation of Hb in arterial blood
Step 4: Check for HCO3 and pCO2
Step 5: Check for base excess
Base excess is defined as the amount of base required to raise the pH to normal range
IF BE is positive – alkaline, BE is negative- acidosis[7]

Is pH authentic?
We know pH= -log [H] according to Henderson Hasselbach equation
pH= pKa+ log [HCO3]/ [pC02]
pH= 6.1 + log HCO3/ 0.03 x pCO2
HCO3 mentioned in ABG is actually using this equation from measured values of pCO2 and pH
Therefore, [H+]neq/l = 24x pCO2 / HCO3
If pH (expected) = pH measured, then the ABG is authentic
Alternatively, subtract the last two digits of pH.Eg: 20 in pH 7.20 from 80 ; this value is approximately equal to H+ concentration.
Eg: consider ABG: pH=7.42, pCO2=32, HCO3=20
Now H+= 24 x 30.8/ 19.3
H+ =38.4
Alternatively, 80 – last 2 digits of pH = 80- 42= 38 =approximately measured pH
Thus, the ABG is authentic

Interpretation of ABG
The most important aspect of ABG interpretation is obtaining a clear history which throws light on the etiology of the acid base disorder.
Any point with history of using diuretics, administration of soda bicarbonate, or on continuous nasogastric aspiration or vomiting is likely to have metabolic alkalosis.
Similarly, a history of hypotension, renal failure, diabetes (uncontrolled) and Metformin therapy indicates possible metabolic acidosis.
COAD/ Asthma, opioids, head surgery and respiratory depression would cause respiratory acidosis.
Any hypermetabolic state, pregnancy, fever, anxiety might cause respiratory alkalosis[8].

Table 1 Arterial versus venous blood gas

Value	Arterial blood	Mixed venous
pH	7.40 (7.35-7.45)	7.31-7.41
PaO2	80-100 mmHg	35-40 mmHg
O2 saturation	⩾95%	⩾70-75%
PaCO2	⩾35-45 mmHg	⩾41-45 mmHg
HCO3	⩾22-26 mEq/L	⩾22-26 mEq/L
BE	-2 to +2	-2 to +2

Table 2 Primary changes and compensatory mechanisms in acid–base disorders

Primary disturbance	Initial imbalance	Compensatory response	Compensatory mechanism	Expected level of compensation
Metabolic acidosis	↓ HCO3	↓ PCO2	Hyperventilation	1.2mmHg decrease in PCO2 for each 1 mmol/L decrease inHCO3(minimum PCO2 of 1.3–1.9 kPa in compensation)
Metabolic alkalosis	↑ HCO3	↑ PCO2	Hypoventilation	0.7mmHgincrease in PCO2 foreach 1 mmol/Lincrease in HCO3(PCO2 should not rise above 7–8 kPain compensation)
Respiratory acidosis	↑ PCO2	↑ HCO3		
• Acute			Intracellular buffering	1–2mmol/L increasein HCO3forevery 10 mmHg increase in PCO2
• Chronic			Renal: generation of bicarbonate via excretion of	3–4 mmol/L increase in HCO3for every 10 mmHg increase in PCO2

			ammonium	
Respiratory alkalosis	↓ PCO2	↓ HCO3		
• Acute			Intracellular buffering	1–2mmol/L decreasein HCO3 for every 10 mmHg decrease in PCO2
• Chronic			Renal: decreased reabsorption of HCO3, decreased excretion of ammonium	4–5 mmol/L decrease in HCO3for every 10 mmHg decrease in PCO2

Oxygenation status:

- Judged by PaO2
- Always consider FiO2 while commenting on oxygen status
- Based on expected PaO2, hypoxia can be classified as mild, moderate or severe[9]

Ventilatory status:

Value of PaCO2

Acid base status:

Primary disorder by pH

pH> 7.4 – alkalemia

pH< 7.4 – acidemia

If PaCO2 is increased (>40) or decreased (<40) then a respiratory disorder is the primary cause.

Else HCO3 (increased or decreased) indicating a metabolic disorder as the primary cause.

In the normal ABG

Direction of pH and PaCO2 is opposite

Direction of pH and HCO3 is same direction[10]

Mixed disorders:

If HCO3 and PaCO2 are in opposite direction, then it is a mixed disorder. In that case pH should be assessed for acidosis or alkalosis.

Eg. If pH= 7.23, HCO3=14, PaCO2=55

Here pH is acidotic, both HCO3 and PaCO2 are having features of acidosis. Then we look at the % difference.

HCO3% difference= 24-14/24 = 10/24 = 0.41

PaCO2 % difference = 55-40/40 = 15/40 = 0.37

Therefore, metabolic acidosis is the dominant disorder[11].

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