Basics of interpretation of ABG

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The structural integrity of intracellular enzymes is essential for survival. Proton activity at enzymatic sites of action in cytosol and organelles must be tightly controlled.

In critically ill patients it is impractical to directly monitor the intra cellular sites. So the clinicians track the extracellular data from tests on arterial blood. We know that plasma pH exceeds intra cellular pH by 0.6 pH units.

Systemic arterial pH is maintained between 7.35 to 7.45 by intra & extracellular chemical buffering together with respiratory & renal regulatory mechanisms. The arterial $\rm CO_2$ tension is controlled by CNS & respiratory system while plasma $\rm HCO_3$ - is controlled is controlled by kidneys so as to stabilize arterial pH.

Henderson-Hasselbatch equation –

pH =
$$6.1 + log$$
 HCO₃-
Pa CO₂ x 0.0301

This equation tells us about metabolic & respiratory components that regulate systemic pH. Usually the CO_2 production & excretion are exactly matched. The $PaCO_2$ is not regulated by CO_2 production but is primarily controlled by neutral respiratory factors in normal circumstances. It means hypercapnia is due to hypoventilation & not due to increased CO_2 production. In simple words, rise or fall in $PaCO_2$ means either of two problems:

- Hypoventilation due to neurological or lung problems
- 2) Compensatory changes in response to primary change in HCO₃-.

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Physician and Intensivist Director of ICU CARE Hospital, Nagpur Kidneys regulate HCO₃- in plasma.

In this review report we will see how to approach an ABG report. We will see how to interpret the values and numbers. Detail discussion on etiology, clinical features & management is out of scope of this article.

Validity of ABG report

Always note that ABG report may not be correct. This is due to improper calibration of machine. If the report is not valid, then the interpretation may be wrong.

Example

A 54 year old male, diabetic presented with respiratory distress. pH 7.20, $PaCO_2 - 30$, $PaO_2 - 108$, HCO_3 - 15. Apparently it looks like metabolic acidosis.

But if we put the values in simplified Henderson's equation:

$$[H+][HCO_3-] = 24$$
$$[PaCO_2]$$

If you put values in above equation, you will come to know that values in the ABG report are wrong. H+ ion concentration is measured by many machines directly. Otherwise one approximate mono gram is given below. From pH value you can calculate [H+] value and can use in this formula.

<u>[H+][HCO₃-]</u> = 24 [PaCO₂]	рН	Substract from	H+
e.g. In DKA	6.8		160
	6.9		130
pH = 7.20	7.0	100	100
PaCO _{2 = 30}	7.1	90	90
HCO ₃ - = 15	7.2	80	60
3	7.3	80	50
60x15 = 30	7.4	80	40
30	7.5	80	30
ABG is Incorrect	7.6	85	25
	7.7	90	20
	7.8	95	15

ABG gives us important informations about :-

- 1) oxygenation status
- 2) ventilation status
- 3) acid-base status

Oxygenation status:

Look at FiO,

Before interpreting, check FiO₂. PaO₂ & O2 saturation is dependent on FiO₂.

When there is normal PaCO₂, normal gas exchange & ventilation & perfusion, then PaO₂ is usually 4-5 times of FiO₂.

Means FiO₂ of 21% gives PaO₂ of 80-100 mm Hg. Normal PaO₂ at higher FiO₂ indicates hypoxemia.

Is the PaO₂ lower than expected?

Calculate (A-a)pO, gradient.

 $PaO_2 = FiO_2(PB-PH_2O) - PaCO_2/R$

Where, PaO₃ is alveolar arterial tension.

FiO, is concentration of O, in inspired air.

PB is barometric pressure which 760 at sea level PH₂O is pressure of water vapor in inspired gas (usually 47)

PaCO, is arterial CO,

R is respiratory coetient, for normal balanced diet it is usually 0.8

Normally the gradient is less than 10- 15 mm Hg. This gradient can tell us the reason for hypoxemia in a particular patient.

ACIDOSIS OR ALKALOSIS

The pH of arterial blood gas measurement identifies the disorder. Normal arterial blood pH is = 7.40 ± 0.05 . Acidemic – pH < 7.35

Alkalemic-pH>7.45

IS THE PRIMARY DISTURBANCE RESPIRATORY OR METABOLIC?

- 1) Respiratory disturbance primarily alters arterial PaCO₂(normal value 40, range 35-45)
- 2) Metabolic disturbance alters the serum HCO₃- (normal value 24, range 22-26)
- 3) In simple acid base disorder, PaCO₂ & HCO₃- move in same direction.
- 4) In simple metabolic disorder , PaCO₂ & HCO₃-moves in same direction of pH
- 5) In simple respiratory disorder, PaCO₂ & HCO₃-moves in opposite direction of pH.

6) In mixed disorder, PaCO₂ & HCO₃- can move in any direction, depending on severity of individual disorder.

DISORDER	рН	PaCO ₂	HCO₃-
METABOLIC ACIDOSIS	+	\	↓
METABOLIC ALKALOSIS	1	<u> </u>	↑
RESPIRATORY ACIDOSIS	\	↑	↑
RESPIRATORY ALKALOSIS	↑	+	↓

RESPIRATORY ACID BASE DISTURBANCES

Primary respiratory disturbances (primary changes in PaCO₂) invokes compensatory metabolic response (secondary changes in HCO₃-).

Compensation in Simple Respiratory Disturbances:-

A) Respiratory acidosis

Acute: HCO₃- increases by 1mEq/L for every 10 mmHg increase in PaCO₃.

Chronic: HCO₃- increases by 4mEq/L for for every 10 mmHg decreases in PaCO₃

B) Respiratory alkalosis

Acute: HCO₃- decreases by 2mEq/L for every 10 mmHg increase in PaCO₃.

Chronic: HCO₃- decreases by 4mEq/L for every 10 mmHg increase in PaCO₃.

HOW TO KNOW IF RESPIRATORY ACIDOSIS IS ACUTE OR CHRONIC?

Calculate Δ H+ & Δ PaCO₂

If $\Delta H+/\Delta PaCO_2$ < 0.3 - chronic

If $\Delta H+/\Delta PaCO_2$ 0.3 to 0.8 - acute or chronic

If $\Delta H+/\Delta PaCO_2$ >0.8 - acute

METABOLIC ACIDOSIS

The primary change is in HCO_3 -, lungs compensate by exhaling CO_3 .

Expected PaCO₂ = $(1.5 \times HCO_{3}) + (8 \pm 2)$

if actual PaCO₂ is less than expected, additional respiratory alkalosis

- ■I f actual PaCO₂ is more than expected, additional respiratory acidosis
- PaCO, is rarely <10mm Hg
- If actual PaCO₂ is <10, consider additional respiratory alkalosis.

Types of metabolic acidosis

- High anion gap metabolic acidosis
- Normal anion gap metabolic acidosis

Anion Gap (AG)

It is difference between actual & calculated anion gap. It is because of few unmeasured anions.

The AG quantifies [unmeasured anions]-[unmeasured cations]

$$AG = Na + - [HCO_3 - + Cl -]$$

Normal AG = 8-12 (average 10 mEq/L)

Albumin Corrected Anion Gap:

For every 1 gm increase or decrease of serum albumin beyond normal(4 gm/dl),

AG increases or decreases by 1.

Causes of increased AG metabolic acidosis

K – ketoacidosis – diabetic, starvation, alcoholic

L - lactic acidosis

U-uremia

M-methanol

P-paraldehayde

E-ethanol, ethylene glycol

S-salicylates

Determine whether other metabolic disturbences coexist with high AG metabolic acidosis – Determine ΔAG & ΔHCO₃- & ΔAG/ΔHCO₃-

- 1) High AG metabolic acidosis, ratio is one.
- 2) High AG metabolic acidosis + metabolic alkalosis -> 1
- 3) High AG metabolic acidosis + normal AG metabolic acidosis <1

The different types of normal anion gap metabolic acidosis, clinical features & management of metabolic acidosis is out of the scope of this review article.

Metabolic alkalosis

The relationship of PaCO₂ & HCO₃ is is difficult to predict & its nonlinear.

Expected PaCO₂= PaCO₂+ $[0.6 \times \Delta HCO_3]$

ΔHCO₃ is change in HCO₃

A patient will increase PaCO₂ above 40 but not greater than 50-55.

If the PaCO₂ is > 55, consider additional respiratory acidosis.

In metabolic alkalosis, if pH is less than expected, consider additional metabolic acidosis.

To summarize,

Cleck validity of report before interpreting the ABG values.

Check FiO2 before interpreting oxygenation status. Check anion gap always.

Interpret ABG in context with clinical condition only.

Treat the patient and not the ABG.