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Simple algorithm of arterial blood gas analysis to ensure consistent, correct and quick responses!

Abstract

Background: Arterial blood gas (ABG) analysis is an essential part of diagnosing and managing a patient's oxygenation, ventilation status as well as acid-base balance. The usefulness of this diagnostic tool is dependent on being able to correctly interpret the results. The body operates efficiently within a fairly narrow range of blood P^H (acid-base balance). Even relatively small changes can be detrimental to cellular function. Disorders of acid-base balance can create complications in many disease states, and occasionally the abnormality may be so severe so as to become a life-threatening risk factor. A thorough understanding of acid-base balance is mandatory for physicians, intensivists, and anesthesiologists are not exception! We must always interpret them in light of the patient's history, clinical presentation and laboratory information's.

Objectives: ABG is not merely a tracing paper! So many variables right at the tracing paper as well as clinical variables of the patients hatch fearfulness among young physicians. So the effort was to make ABG EASY and to develop an algorithm which will conduct navigating diagnosis!

Conclusion: Arterial blood gases help assess three vital physiologic processes in the critically ill patient: acid-base balance, ventilation and oxygenation. Initial blood gas analysis helps diagnose underlying disease processes as well as guide therapeutic interventions. Serial measurements can be utilized to assess proper response to therapy. Blood gas analysis takes a step-by-step approach and practice. Blood gas data should always be integrated in light of the full clinical and laboratory information.

Keywords: oxygenation, ventilation, acid-base, metabolic, saturation, bicarbonate

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Introduction

Arterial blood gas (ABG) analysis is a crucial part of diagnosing and managing a patient's state of oxygenation, ventilation as well as acid-base balance. The practicability of this diagnostic tool is dependent on being able to correctly interpret the results. Disorders of acid-base balance can create complications in many disease processes, and occasionally underlying disorders may be so severe that might cause life-threatening risk. So, thorough understanding of acid-base balance is vital for any physician, intensivist, and anesthesiologists are not exception.

ABG analysis is a diagnostic tool that allows the objective evaluation of a patient's oxygenation, ventilation and acid-base balance. The results from an ABG will indicate not only patient's respiratory status but also indicate how well a patient's kidneys and other internal organs (metabolic system) are functioning. Although all of the data in an ABG analysis can be useful, it is possible to interpret the results without all variables. Essentials of interpreting ABG need maximum of six values: - Oxygen concentration (PO_2), - Oxygen saturation (SaO_2), - Bicarbonate ion concentration (HCO_3-), - Base excess, - Carbon dioxide concentration (PCO_2); - Hydrogen ion concentration (P^H).[1–5]

Basic terminology[6–8]

a. P^H: signifies free hydrogen ion concentration. P^H is inversely related to H+ ion concentration.

b. Acid: a substance that can donate H+ ion, i.e. lowers P^H.

c. Base: a substance that can accept H+ ion, i.e. raises P^H.

d. Anion: an ion with negative charge.

e. Cation: an ion with positive charge.

f. Acidemia: blood P^H< 7.35 with increased H+ concentration.

g. Alkalemia: blood P^H>7.45 with decreased H+ concentration.

h. Acidosis: Abnormal process or disease which reduces P^H due to increase in acid or decrease in alkali.

i. Alkalosis: Abnormal process or disease which increases P^H due to decrease in acid or increase in alkali.

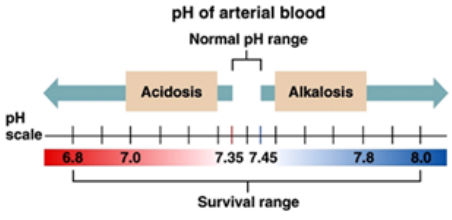
Requirement of acid-base balance[7,8]

Acid-base balance is important for metabolic activity of the body:

P^H of arterial blood = 7.35 – 7.45.

Alteration of P^H value out of the range 7.35-7.45 will have effects on normal cell function.

P^H< 6.8 or > 8.0 death occurs.



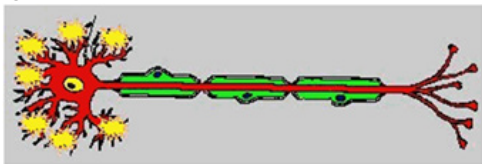
Changes in excitability of nerve and muscle cells

↓P^H→ depresses the CNS

Can lead to loss of consciousness.

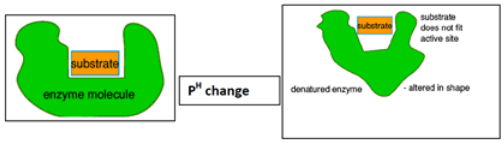
↑P^H → over-excitability of CNS

Tingling sensations, nervousness, muscle twitches.



Alteration of enzymatic activity:

P^H change out of normal range can alter the shape of the enzyme rendering it non-functional.



Alteration of K^+ levels

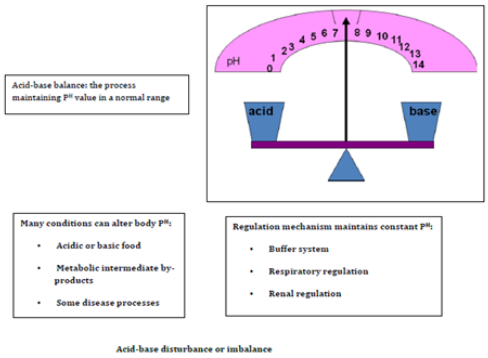
Acid-base state of ECF influence:

K^+ distribution in ECF and ICF

Renal excretion of K^+

Acid-base disturbance or imbalance

I. Acid-base balance: the process maintaining P^H value in a normal range



Acid-base disturbances:

i. Secondary alterations to some diseases or pathologic processes

ii. Can aggravate and complicate the original disease

iii. Concept of acids and bases:

iv. Acids are molecules that can release H+ in solution. (H+ donors)

v. Bases are molecules that can accept H+ or give up OH- in solution. (H+ acceptors)

vi. Acids and bases can be:

a. Strong – dissociate completely in solution. HCl, NaOH

b. Weak – dissociate only partially in solution. Lactic acid, carbonic acid

Regulation of acid-base balance

a. Blood buffering

React very rapidly (less than a second)

b. Respiratory regulation

Reacts rapidly (seconds to minutes)

c. Ion exchange between intracellular and extracellular compartment and intracellular buffering

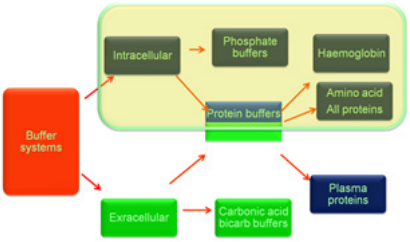
Reacts slowly (2 ~ 4 hours)

d. Renal regulation

Reacts very slowly (12 ~ 24 hours)

table 2.5 Hydrogen Ion Concentrations and pH

Grams of H^+ per Liter	pH	
0.00000000000001	14	↑ Increasingly basic
0.0000000000001	13	
0.000000000001	12	
0.00000000001	11	
0.0000000001	10	
0.000000001	9	
0.00000001	8	Neutral–neither acidic nor basic
0.0000001	7	
0.000001	6	
0.00001	5	
0.0001	4	
0.001	3	Increasingly acidic
0.01	2	
0.1	1	
1.0	0	↓



Respiratory regulation

The lung regulates the ratio of $[HCO_3^-]/[H_2CO_3]$ to approach 20/1 by controlling the alveolar ventilation and further elimination of CO_2, so as to maintain constant P^H value.

Regulation of alveolar ventilation (V_A)

a. V_A is controlled by respiratory center (at medulla oblongata).

b. Respiratory center senses stimulus coming from:

Central chemoreceptor (located at medulla oblongata)

c. Alteration of [H+] in Cerebrospinal fluid

↑[H+] in Cerebrospinal fluid→ respiratory center exciting→ ↑V_A

d. Alteration of $PaCO_2$

$PaCO_2$> 60mmHg→ V_A increase 10 times

$PaCO_2$> 80mmHg→ respiratory center inhibited

Peripheral chemoreceptor (carotid and aortic body)

e. ↓PaO_2 or ↑$PaCO_2$ or ↑[H+]

↓PaO_2< 60mmHg→ respiratory center exciting→ ↑V_A

↓PaO_2< 30mmHg→ respiratory center inhibited

How does alteration of alveolar ventilation regulate P^H value?

↑[H+] in Blood→ rapidly buffered by buffer system such as HCO_3^-/$H2CO_3$→ ↓ $[HCO_3^-]$ and ↑ $[H2CO_3]$ → $[HCO_3^-]/[H2CO_3]$ tend to decrease, while ↑[H+] can stimulate peripheral chemoreceptor

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→respiratory center exciting →↑alveolar ventilation →↑CO_2 elimination →↓$PaCO_2$ → [HCO_3-] / [$H2CO_3$] tends to 20/1 → P^H is maintained.

Renal regulation

The kidney regulates [HCO_3-] through changing acid excretion and bicarbonate conservation, so that the ratio of [HCO_3-]/[$H2CO_3$] approach 20/1 and P^H value is constant.

Bicarbonate conservation

a. Bicarbonate regeneration by distal tubule and collecting duct.

b. Bicarbonate reclamation by proximal tubule.

How does the renal regulation maintain the constant PH value?

↑[H+] in Blood→ rapidly buffered by buffer system such as HCO_3-/ $H2CO_3$→ ↓ [HCO_3-] and ↑ [$H2CO_3$] → [HCO_3-]/[$H2CO_3$] tend to decrease, while ↑[H+] can stimulate the activity of CA, H+-ATPase and glutaminase→↑secretion of H+ and ammonia, ↑reabsorption of HCO_3- → [HCO_3-] / [$H2CO_3$] tends to 20/1 → P^H is maintained.

Ion exchange between intra- and extracellular compartment & intracellular buffering:

A. Intracellular buffer system

a. Phosphate buffer system (HPO_4^{2-} /H_2PO^{4-})

b. Hemoglobin (Hb-/HHb) and oxyhemoglobin buffer system (HbO_2-/$HHbO_2$)

A. Ion exchange between intra- and extracellular compartment

i.e. ↑Extracellular [H+] → H+ shift into cells and K^+ shift out of cells

a. acidosis→ hyperkalemia

b. alkalosis→ hypokalemia

Base excess & base deficit[9,10]

In human physiology **base excess** and **base deficit** refer to an excess or deficit, respectively, in the amount of base present in the blood. The value is usually reported as a concentration in units of mEq/L, with positive numbers indicating an excess of base and negative a deficit. A typical reference range for base excess is −2 to +2mEq/L. Comparison of the base excess with the reference range assists in determining whether an acid/base disturbance is caused by a respiratory, metabolic, or mixed metabolic/respiratory problem.

The base excess of blood does not truly indicate the base excess of the total extracellular fluid (ECF). Because of different protein content and the absence of hemoglobin, ECF has a different buffering capacity. What's more, each extracellular fluid (for example CSF vs interstitial fluid) has a different buffer status. The clinical determination of the amount of bicarbonate required for treatment of severe acidosis is usually based on the base excess of the blood.

There is an unavoidable inaccuracy, however, due to several factors:

i. The time course of the acidosis makes the blood acid poorly reflect the total body acid burden in many cases.

ii. Depending on the state of hydration, body fluid distribution varies.

iii. ECF as a percent of body weight varies with age and fat content.

In general, however, recommendations for bicarbonate therapy are in the range of 0.1 to 0.2 mEq times the body weight times the base excess (ignoring the minus sign).

Bicarb = 0.1 x (-B.E.) x Wt in Kg

Tips for determining primary and mixed acid base disorder[11,12]

Tip-1: Only a process of acidosis can make the P^H acidic and only a process of alkalosis can make P^H alkaline

Tip-2: In primary disorder P^H 7.35 ─ 7.40 is indicative of primary acidosis, when compensation is complete

Tip-3: In primary disorder P^H 7.40 ─ 7.45 is indicative of primary alkalosis, when compensation is complete

Tip-4: Keeps in mind that three states of compensation are possible:

a. Non-compensation- alteration of only PCO_2 or HCO_3-

b. Partial-compensation- When all three variables like P^H, $PaCO_2$ and HCO_3- are abnormal.

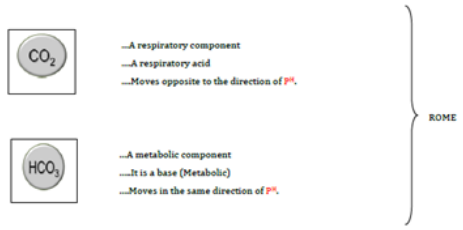
c. Complete-compensation- P^H is normal but both $PaCO_2$ & HCO_3- are abnormal.

Tip-5: Don't interpret any blood gas data without examining corresponding serum electrolytes.

Tip-6: Truly normal P^H with distinctly abnormal HCO_3- and $PaCO_2$ invariably suggests two or more disorders.

Tip-7: Whenever the PCO_2 and [HCO_3] are abnormal in *opposite* directions, *ie*, one above normal while the other is reduced, a mixed respiratory and metabolic acid-base disorder exists.

Facts about Acid-Base balance……[13]



Compensation of primary & mixed disorder

Compensation for simple acid-base disturbances always drives the compensating parameter (*ie*, the PCO_2, or [HCO_3-]) in the same direction as the primary abnormal parameter (ie, the [HCO_3-] or PCO_2) & compensation for mixed disorder always drives compensating parameters in the opposite direction as the primary abnormal parameters.

Description of superscripts inside algorithm box[15–29]

a. Increase or decrease of P^H in relation with HCO_3- indicate metabolic disorder

b. Increase or decrease of P^H in relation with PCO_2 indicate respiratory disorder

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c. Step to look at compensation, noncompensation means alteration of only PCO_2 or HCO_3-

d. Partial compensation means P^H, PCO_2 and HCO_3- all variables are abnormal

e. Full compensation means only P^H is normal but PCO_2 and HCO_3- are abnormal

f. Anion Gap (AG) = Na+ -(Cl + HCO_3-), it represents unmeasured anions in the plasma which primarily includes Sulphate, Organic acids, Albumin and Phosphate (SOAP). The normal value of AG is 12 ± 4, an increase AG almost always indicates metabolic acidosis

g. HAGMA(High Anion Gap Metabolic Acidosis)- Increase anion gap means an acid has been added to the blood, causes are KULT means Ketoacidosis,Uraemia,Lactic acidosis, Toxins

h. NAGMA(Normal Anion Gap Metabolic Acidosis)- when HCO_3 is lost to maintain electroneutrality Cl is conserved by Kidney's, so anion gap is normal, causes are DURHAM means Diarrhoea, Ureterosigmoid fistula, RTA, hyperalimentation, Acetazolamide, Misc

i. Delta Gap=$\Delta AG/\Delta HCO_3$

Delta ratio is a formula that can be used to assess elevated anion gap metabolic acidosis and to evaluate whether mixed acid base disorder is present.

In High anion gap metabolic acidosis (HAGMA) Delta ratio will be 1-2

If the ratio is greater than 2 in a HAGMA it is due to concurrent metabolic alkalosis.

In Nonanion gap metabolic acidosis (NAGMA) delta ratio will be =0.4

If the ratio is between 0.4-1 then it is due to Mixed (HAGMA+NAGMA) disorser

Bedside Rules for Assessment of Compensation[14]

Rule 1: The 1 for 10 Rule for Acute Respiratory Acidosis

The [HCO_3] will increase by 1 mmol/l for every 10 mmHg elevation in pCO_2 above 40 mmHg.

Expected [HCO_3] = 24 + {(Actual pCO_2 - 40) / 10}

Rule 2: The 4 for 10 Rule for Chronic Respiratory Acidosis

The [HCO_3] will increase by 4 mmol/l for every 10 mmHg elevation in pCO_2 above 40mmHg.

Expected [HCO_3] = 24 + 4 {(Actual pCO_2 - 40) / 10}

Rule 3: The 2 for 10 Rule for Acute Respiratory Alkalosis

The [HCO3] will decrease by 2 mmol/l for every 10 mmHg decrease in pCO_2 below 40 mmHg.

Expected [HCO_3] = 24 - 2 {(40 - Actual pCO_2) / 10}

Rule 4: The 5 for 10 Rule for a Chronic Respiratory Alkalosis

The [HCO3] will decrease by 5 mmol/l for every 10 mmHg decrease in pCO_2 below 40 mmHg.

Expected [HCO_3] = 24 - 5 {(40 - Actual pCO_2) / 10} (range: +/- 2)

Rule 5: The One & a Half plus 8 Rule - for a Metabolic Acidosis

The expected pCO_2 (in mmHg) is calculated from the following formula:

Expected pCO_2 = 1.5 x [HCO_3] + 8 (range: +/- 2)

Rule 6: The Point Seven plus Twenty Rule - for a Metabolic Alkalosis

The expected pCO_2 (in mmHg) is calculated from the following formula:

Expected pCO_2 = 0.7 [HCO_3] + 20 (range: +/- 5)

Conclusion[30]

Predicting difficult tracheal intubation is still a matter of debate among anesthesiologists, with each individual score or sign having a poor predictive value.[1] In the present case report we present a case of difficult tracheal intubation that could not have been predicted with any score or clinical sign used for preoperative airway assessment.

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None.

Conflicts of interest

The authors declare there is no conflict of interests.

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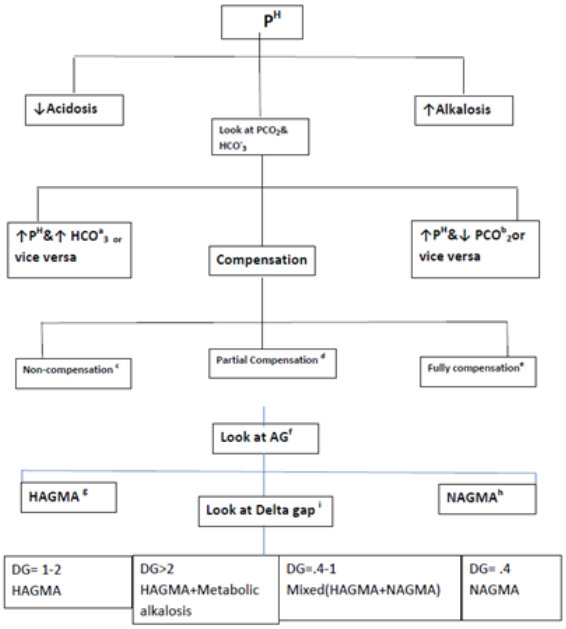
None.

Algorithm for interpreting arterial blood gas analysis: (Annexure-1)



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