Interpretation of Mixed Acid Base Disorders-

BY

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Background

Arterial Blood Gasses (ABGs) analysis is usually an emergency test required in a critically ill patient. It is of immense help in the management of these patients.

Arterial Puncture

It requires considerable skill and should be carried out by a medical officer or a well trained nursing staff.

- The preferred sites are in the order: а
 - (1) Radial Artery at the wrist (best site because no vein adjacent to Radial Artery (2) Brachial Artery in the elbow
 - (3) Femoral Artery in the groin (Leakage from this artery tends to be greater than the sites in the arm, so it should be avoided especially in elderly.
 - (4) In Neonates, sample can be collected from umbilical artery
- b. Syringe should be rinsed thoroughly with Lithium Heparin
- c. Artery should be punctured at an angle of 90 after feeling the pulse and attaining proper sterilization.
- d. Just 1-2 ml blood is sufficient
- e. Remove air bubbles from the syringe immediately after the sample collection.
- Thoroughly mix the sample by vertical and rolling movements. f.
- g. Label the sample as per standard procedure.
- h. Arterialized Capillary Blood. In children (and in adults in whom it is impossible to perform an arterial puncture) a capillary puncture may be performed to obtain an arterialized capillary blood. The preferred site is earlobe in young child or adult and in infant it is heel. The skin should be first warmed by hot moist towels to improve the circulation. Then the blood should be collected in a capillary tube which should be sealed immediately after collection. Such a sample may not be appropriate for PO_2 .

Transportation of Sample:

- a. The arterial sample should be transported immediately to the lab for analysis.
- b. The samples that are measured within 15 minutes may be kept at room temperature.
- c. If unable to transport / measure sample within 15 minutes then place them temporarily in ice.
- d. Maximum transportation / measurement time is 30 minutes.

Reference Ranges (Adults):

• pH :	7.35 - 7.45
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- PCO_2 :
 35 48 mmHg

 HCO_3 22 28 mmol/I
- 22 28 mmol/L
- 83 108 mmHg PO_2
- Base Excess: -2.0----+3.0

Reference Ranges (Neonates)*:

•	Cord Blood:	рН :	7.18 - 7.38
•	Premature (48 h):	рН :	7.35-7.50
•	Full term		
	a. At Birth:	рН :	7.11 – 7.36
	b. 5-10 min	рН :	7.09 - 7.30
	c. 30 min	рН :	7.21-7.38
	d. 1 hr	рН :	7.26-7.49
	e. 1 Day	рН :	7.29–7.45
	f. Older Child	рН :	7.35-7.45

* Roberts WL, McMillin GA, Burtis CA, Burns DE. Reference Information for the Clinical Laboratory; In Burtis CA, Edward RA, (edi) Teitz Textbook of Clinical Chemistry. 4th ed. Saunders; 2006; 2289.

<u> ABG measurement</u> –

- •Samples to reach lab within 15 minutes
- •No incubation or reagent mixing
- •Instant results
- •Invalid if result delayed
- •"bedside use" with some precautions

Measured parameters:

- рН
- Partial Pressure of Carbon dioxide (PCO2)
- Partial Pressure of oxygen (PO2)

Calculated parameters:

- HCO3 using Henderson Hasselbalch equation)
- Base Excess (BE)

Types of Acid Base Disorders

- Single (simple) disorders
- Double (mixed) disorders
- Triple (mixed) disorders

Double Disorders

•Metabolic and Respiratory Acidosis

•Metabolic and Respiratory Alkalosis

•Metabolic Alkalosis and Respiratory Acidosis

•Metabolic Acidosis and Metabolic Alkalosis

Triple Disorders

Metabolic Acidosis, Metabolic Alkalosis and Respiratory Alkalosis:

Any high anion gap acidosis complicated by vomiting and hyperventilation can cause this disorder

Analysing ABG Reports

- Clinical features
- Results of other investigations e.g. electrolytes etc
- ABG result necessary for confirmation of diagnosis
- Biochemical opinion may be given even with meagre information

Interpretation Assistants

- Decoder
- Electronic Calculators (Skyscape for desktop, iPods etc.)
- Equations usually required for subtle changes in ABGs.
- Flenley's Graph (See Figure)

Decoding an ABG Report

1. Look at pH.

- i. Low pH-----Acidosis
- ii. <u>High pH</u>----Alkalosis
- iii. <u>Normal pH</u>--- A normal pH does not rule out existence of an acid base disorder (see below)

2. <u>If pH is abnormal Examine PCO₂ and HCO₃ relationship.</u>

• If PCO₂ and HCO₃ change in the same direction, it is a single disorder.

<u>Example:</u>

in Metabolic Acidosis (i.e. decreased HCO_3), PCO_2 should decrease. If it is decreased, consider Metabolic Acidosis with Respiratory Compensation (Single Disorder)

• If *P*CO₂ and HCO₃ change in the opposite direction, it is a **double disorder**

Example:

in Metabolic Acidosis (i.e. decreased HCO_3), PCO_2 should decrease. If it is increased, consider Metabolic Acidosis with Respiratory Acidosis (Double Disorder)

3. <u>Examine pH and HCO₃ relationship (For single disorders)</u>

a. If pH and HCO₃change in the same direction primary abnormality is metabolic

Examples:

- *in metabolic acidosis both pH and HCO*₃*decrease*
- in metabolic alkalosis both pH and HCO₃ increase
- b. If pH and HCO₃ change in the opposite direction primary abnormality is respiratory

Examples:

- in respiratory acidosis pH decreases and HCO₃ increases
- in respiratory alkalosis pH increases and HCO₃ decreases

4. If pH is normal Examine PCO₂ and HCO₃ relationship again

If *P*CO₂ and HCO₃ **grossly** change in the same direction, it is also a **double disorder**

<u>Examples:</u>

- in Metabolic Acidosis (i.e. decreased HCO₃), PCO₂ should decrease to a certain extent. If it is decreased too much, consider Metabolic Acidosis with Respiratory Alkalosis (Double Disorder)
- in Respiratory Acidosis (i.e. increased PCO₂), HCO₃ should increase to a certain extent. If it is increased too much, consider Respiratory Acidosis with Metabolic Alkalosis (Double Disorder)
- <u>If pH, PCO₂ and HCO₃ all are normal -----normal Acid Base Status</u> (Please be careful of values on the extreme of reference ranges, which may be due to a fully compensated disorder like Chronic Respiratory Alkalosis or mild double disorders)

The Equations

1. <u>Acute Respiratory Acidosis</u>

Expected [HCO₃] = 24 + {(Actual *P*CO₂ - 40) / 10}

Example: A patient with an acute respiratory acidosis (PCO₂ 60mmHg) has an actual [HCO₃] of 31mmol/l. The expected [HCO₃] for this acute elevation of PCO₂ is 24 + 2 = 26mmol/l. The actual measured value is higher than this indicating that a metabolic alkalosis must also be present.

2. <u>Chronic Respiratory Acidosis</u>

Expected [HCO₃] = 24 + 4 {(Actual *P*CO2 - 40) / 10}

Example: A patient with a chronic respiratory acidosis ($pCO2\ 60mmHg$) has an actual [HCO3] of 31mmol/l. The expected [HCO3] for this chronic elevation of pCO2 is 24 + 8 = 32mmol/l. The actual measured value is extremely close to this so renal compensation is maximal and there is no evidence indicating a second acid-base disorder.

3. <u>Acute Respiratory Alkalosis</u>

Expected [HCO₃] = 24 - 2 {(40 - Actual PCO₂) / 10}

Example: A patient with an acute respiratory alkalosis (PCO2 15 mmHg) has an actual [HCO₃] of 13 mmol/l. The expected [HCO₃] for this chronic elevation of PCO2 is 24 - 5 = 19 mmol/l. The actual measured value is lower than this indicating that a metabolic acidosis must also be present

4. <u>Chronic Respiratory Alkalosis</u>

Expected [HCO₃] = 24 - 5 {(40 - Actual *P*CO₂) / 10} (range: +/- 2)

- It takes 2 to 3 days to reach maximal renal compensation
- The **limit of compensation** is a [HCO₃] of about 12 to 15 mmol/l

Example: A patient with chronic respiratory alkalosis (PCO2 15 mmHg) has an actual [HCO₃] of 13 mmol/l. The expected [HCO₃] for this chronic elevation of PCO2 is 24 - 5 = 12 mmol/l. The actual measured value is close to this is so there is normal renal compensation and there is no evidence indicating a second acid-base disorder.

5. <u>Metabolic Acidosis</u>

Expected pCO2 (mmHg) = 1.5 x [HCO3] + 8 (range: +/- 2)

- Maximal compensation may take 12-24 hours to reach
- The **limit of compensation** is a *P*CO₂ of about 10 mmHg
- Hypoxia can increase the amount of peripheral chemoreceptor stimulation

Example: A patient with a metabolic acidosis ([HCO3] 14mmol/l) has an actual PCO_2 of 30mmHg. The expected PCO_2 is $(1.5 \times 14 + 8)$ which is 29mmHg. This basically matches the actual value of 30 so compensation is maximal and there is no evidence of another respiratory acid-base disorder

6. <u>Metabolic Alkalosis</u>

Expected PCO_2 (mmHg) = 0.9 x [HCO₃ (mmol/L) + 9

In metabolic alkalosis (alkalaemia plus high plasma [HC03) the compensatory response is hypoventilation and an increase in the blood PCO_2 , (respiratory acidosis). Unlike metabolic acidosis, the PCO_2 response in this condition is irregular, but the limit of compensation for PCO2 is about 60mmHg.

Example: A patient with a metabolic alkalosis ($[HCO_3]$ 30mmol/l) has an actual PCO₂ of 35mmHg. The expected PCO₂ is (0.9 x 30+ 9) which is 36mmHg. This basically matches the actual value of 36 so compensation is optimal and there is no evidence of another respiratory acid-base disorder

ANION GAP

It is a measure of anions othe	r than HCO ₃ and C	hlc	oride
	Always: CATIONS	=	ANIONS

	Major Cations	Major Anoins		
Na =	140 mmol/L	Cl	=	100 mmol/L
K =	4 mmol/L	HCO ₃	=	27 mmol/
Ca =	2.5 mmol/L	proteins	=	15 mmol/L
		PO ₃	=	2 mmol/L
Mg =	1.5 mmol/L	SO ₃	=	1 mmol/L
		Organic Acids	=	5 mmol/L
TOTAL CA	ATIONS = 150 mmol/L	TOTAL ANIO	NS	= 150 mmol/L

Calculating Anion Gap:

$$(Na + K) - (Cl + HCO_3)$$

Ref Range : 7 - 18 mmol/L

It is only a lab derived index

There can *Never* be an anion gap in any condition because electroneutrality is always maintained in the plasma

DELTA RATIO

The delta ratio is used for the determination of a mixed acid base disorder in an elevated anion gap metabolic acidosis

 $= \frac{\text{Measured anion gap} - \text{Normal anion gap}}{\text{Normal [HCO_3-]} - \text{Measured [HCO_3-]}} = \frac{(\text{anion gap} - 12)}{(24 - [HCO_3-])}$

Delta Ratio Assessment Guideline

< 0.4	Hyperchloraemic normal anion gap acidosis
0.4 - 0.8	 Renal failure (<1) Combined high anion gap and normal anion gap acidosis
1 to 2	Usual for uncomplicated high- anion gap acidosis Lactic acidosis: 1.6 (average value)
>2	A pre-existing elevated HCO ₃ level due to: • a concurrent metabolic alkalosis, or • a pre-existing compensated respiratory acidosis

