# Acid-Base Balance Workshop

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## Objectives

- Normal Acid-Base Physiology
- Simple Acid-Base Disorders
- Compensations and Disorders
- The Anion Gap
- Mixed Acid-Base disorders
- The General Approach
- Common pitfalls and Practical Issues

### Don't forget about the patients



Mastering ABG interpretation will happen only if you practice



### **Role of Acid-Base Balance**

- Maintains stable pH at 7.40 (7.35 7.45)
- Physiologic pH "necessary to prevent enzyme inactivation and denaturing"



### **Role of Acid-Base Balance**

- Clinical consequences of deregulation of acid-base balance:
  - Poor vascular tone
  - Myocardial pump failure
  - Increased risk of arrhythmias
  - Skeletal muscle weakness
  - Electrolyte abnormalities
  - Delirium/Coma
  - Impaired cellular respiration



## **Role of Acid-Base Balance**

• Net Acid production = Net Acid Elimination

#### **Three Stages**

- 1. Acid is produced as a consequence of normal metabolism
- 2. Acid is transport via blood
- 3. Acid is eliminated via lungs and kidneys

## 1. Acid Production (Physiologic)



## 1. Acid Production (Pathological)

#### **Accumulated Acids**



### 2. Intravascular Transport of Acid

- To prevent sudden and large swings in pH, buffers are necessary
- Buffers have two major characteristics:
  - Consists of a weak acid and its anion
  - Resists changes in pH

- Three major buffering systems:
  - Protein buffer system
    - Amino acid
  - Hemoglobin buffer system
    - H<sup>+</sup> are buffered by hemoglobin
    - Carbonic acid-bicarbonate
      - Buffers changes caused by organic and fixed acids
  - Minor buffering system
    - Phosphate
      - Buffer pH in the ICF

#### The Basic Relationship between P<sub>CO2</sub> and Plasma pH Increased Increased pH PcO2 pH HOMEOSTASIS (more basic) Pco2 40-45 mm Hg 7.35-7.45 Decreased pH Decreased (more acidic) Pco2 if if Pco<sub>2</sub> Pco2 falls rises pH falls, Pcoz acidosis pH P<sub>CO2</sub> falls develops pH rises, PH PCO2 P<sub>CO2</sub>rises alkalosis develops Relationship Between P<sub>CO2</sub> and Plasma pH

## Buffer Systems in Body Fluids



### **Amino Acid Buffers**







In acidic medium, amino acid acts as a base and absorbs H<sup>+</sup>

In alkaline medium, amino acid acts as an acid and releases H<sup>+</sup>

### The Carbonic Acid-Bicarbonate Buffer System



### 3. Elimination of Acid

<u>Site</u>		<u>Mechanism</u>
Lungs		Expiration of CO <sub>2</sub>
<section-header></section-header>	Proximal Tubule	Reabsorption of HCO <sub>3</sub>
	Distal Tubule/Collecting ducts	Excretion of H <sup>+</sup> as titratable acid
		Excretion of NH <sub>4</sub> <sup>+</sup>

Primary Acid-Base Disorders

### Acidemia vs Alkalemia



### **Respiratory vs Metabolic Disorders**











pH /  $P_aCO_2$  /  $P_a\Theta_2$  /  $HCO_3$  /  $\Theta_2$ -Sat

### Step 1: Always start with the pH

#### $pH / P_aCO_2 / HCO_3$

If pH <7.35 >> Acidemia

If pH >7.45 >> Alkalemia

## Step 2: Check the PCO<sub>2</sub>

#### pH 7.25 / P<sub>a</sub>CO<sub>2</sub> 60 / HCO<sub>3</sub> 26

pH is  $\checkmark$  than normal, while  $P_aCO_2$  is than normal

Since these are deranged is different directions the disorder is reparatory

**Respiratory Acidosis** 

## Compensation

- A primary metabolic disorder will result in respiratory compensation
- A primary respiratory disorder will result in metabolic compensation

### Compensation

Compensation does not return the pH to normal

Patients <u>never</u> "overcompensate"

### Compensation



#### Remember.....

#### Respiratory compensation is always

**FAST**...12-24 hrs



Metabolic compensation always **SLOW**...5 -7 days



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#### **Acid-Base Disorders**

#### **Blood Gas Norms**

	рН	pCO <sub>2</sub>	pO <sub>2</sub>	HCO <sub>3</sub>	BE
Arterial	7.35-7.45	35-45	80-100	22-26	-2 to +2
Venous	7.30-7.40	43-50	~45	22-26	-2 to +2



Note: if PaCO2, PH & HCO3 in same direction----→Metabolic if opposite →Respiratory

**HCO**<sub>3</sub>

PaCO<sub>2</sub>

### **Practical Approach**

#### First:

- Detailed history/exam
- Medication
- Complementary lab test (Na/K/Cl , UEG,etc)

#### Second: Check if data consistence

$$\left[H^{+}\right] = 24 \times \frac{PaCO_{2}}{HCO_{3}^{-}}$$

<u>pH</u>	$[\mathbf{H}^+]$	<u>pH</u>	$[\mathrm{H}^+]$
7.80	16	7.30	50
7.75	18	7.25	56
7.70	20	7.20	63
7.65	22	7.15	71
7.60	25	7.10	79
7.55	28	7.00	89
7.50	32	6.95	100
7.45	35	6.90	112
7.40	40	6.85	141
7.35	45	6.80	159

#### Third: What does the patient has



#### Fourth: Determine is it respiratory or metabolic?



### Fifth- Evaluate Compensatory Response:

#### If there is primary metabolic:

Acidosis:

-Expected PACO2=1.5X(HCO3)+8+/-2

#### Alkalosis:

-PaCO2 increase by 7mmhg for each 10meq/l increase in [HCO3]

If expected = measured  $-\rightarrow$  fully compensated.

If expected > measured --→ respiratory Alkalosis

If expected < measured---→ respiratory acidosis

#### If there is respiratory :

#### Acidosis:

-[HCO<sub>3</sub><sup>-</sup>] increases by 1 (<u>acute</u>) & 3.5-4 (<u>chronic</u>) for each 10-mm Hg increase in PCO<sub>2</sub>.

#### **Alkalosis:**

-[HCO<sub>3</sub><sup>-</sup>] falls by 2 (<u>acute</u>) & 4 (chronic) for each 10-mm Hg decrease in  $PCO_2$ 



DISORDER	EXPECTED COMPENSATION
Metabolic acidosis	$PCO_2 = 1.5 \times [HCO_3^{-1}] + 8 \pm 2$
Metabolic alkalosis	$PCO_2$ increases by 7 mm Hg for each 10 mEq/L increase in s. [ $HCO_3^{-}$ ] or $PaCO_2 = 0.7 \times (HCO3^{-}) + 20 \pm 5$
Respiratory acidosis	
Acute	[HCO <sub>3</sub> <sup>-</sup> ] increases by 1 for each 10-mm Hg increase in PCO <sub>2</sub>
Chronic	[HCO <sub>3</sub> <sup>-</sup> ] increases by 3.5 for each 10-mm Hg increase in PCO <sub>2</sub>
Respiratory alkalosis	
Acute	[HCO <sub>3<sup>-</sup>] falls by 2 for each 10-mm Hg decrease in <math>PCO_2</math></sub>
Chronic	$[HCO_3^{-}]$ falls by 4 for each 10-mm Hg decrease in PCO <sub>2</sub>

### Sixth: Calculate Anion Gap

- SAG-Represents the level of unmeasured anions in extracellular fluid
- Helps differentiate acidotic conditions
   UA-UC=Na-(Cl+HCO3)
- SAG=Na-(Cl+HCO3)
  - Normal=10+/-2
- Corrected SAG for Hypoalbumenia = measured SAG+2.5(4-Albumin {g/dl})



### **Urine & Osmolar gap**

- UAG=Na + K- Cl
- NeGUTive----GUT loss
- Positive----RTA
  - -<u>Type 1-(</u>urine PH>5.5 & S.K low) -<u>Type 2-(</u>urine PH <5.5 & S.K low) -<u>Type 4-(</u>urine PH <5.5 & S.K high)

#### > For high osmolar gap calculate:

**Osmolar Gap=**measured serum osmollarity – calculated serum osmolality .

(Normal < 15mosmol/kg)

If high consider :ME DIE

-Methanol

-Ethanol

-Diuretic

- -Isopropyl Alcohol
- -Ethylene glycol

#### Seventh: Calculate the excess anion gap

Compare change of AG to change in HCO3
 Can uncover mixed metabolic disorder (Metabolic acidosis & Metabolic Alkalosis).

- **Excess AG(G-G ratio)=** {Calculated AG-12}+measured HCO3 If the Sum > 30----Concomitant Metabolic Alkalosis.
- If the Sum < 23----concomitant non Anion Gap Metabolic Acidosis



3 yo boy with diarrhea i ph – 7.23 HCO3 – 10 pCO2 – 23 AG - 13

Expected pCO2 = (1.5 \* HCO3) + 8 +/-2So, Expected pCO2 = (1.5 \* 10) + 8 +/-2=(15) + 8 +/-2=23 +/-2So, we have a metabolic acidosis with respiratory compensation (Use Winter's formula) 5 yo boy presents to ED with dyspnea for 3 days. ABG shows the following:



#### **Case study**

15 years; old; Female pregnant with insulin dependent diabetes is admitted to the ICU after stopping insulin . She has had severe nausea and vomiting for several days & she start to be tachypnea.

ABG - PH 7.55/ PaCO2- 21/ HCO3 18/ PaO2-70

Na-136 BUN-32

Cr-1.2 K - 3.5

Cl-70 CO2-19

-What is/are the Acid – base imbalance she had?

A 4 year old moderately dehydrated was admitted with a two day history of acute severe diarrhea.

Electrolyte results: Na+ 134, K+ 2.9, Cl- 108, HCO3- 16, BUN 31,

Cr 1.5. -ABG: pH 7.31 pCO2 33 HCO3 16 pO2 93 mmHg What is the acid base disorder

A 12 year old female with type I DM, presents to ER with a 1 day history of nausea, vomiting, polyuria, polydypsia and vague abdominal pain. P.E. noted for deep sighing breathing, orthostatic hypotension, and dry mucous membranes.

Labs:

Na 132 , K 6.0, Cl 93, HCO3- 11, glucose 720, BUN 38, Cr 2.6. UA: pH 5, SG 1.010, ketones negative, glucose positive . Plasma ketones trace. ABG: pH 7.27 HCO3- 10 PCO2 23

-What is the acid base disorder?

A previously well 5 year old male is admitted with a complaint of severe vomiting for 5 days. Physical examination reveals postural hypotension, tachycardia, and diminished skin turgor. The laboratory finding include the following:

> Na=140,K=3.4,Cl=77,HCO3-=9,Cr=2.1 ABG: pH=7.23 , PCO2= 22mmHg

A 16 year old women with history of CHF presents with increased shortness of breath and leg swelling.

ABG: pH 7.24, PCO2 60 mmHg, PO2 52 HCO3- 27 -What is the acid base disorder? A 20 year old female presents with nausea, vomiting and poor oral intake 2 days prior to admission. The patient reports a 3 day history of binge drinking prior to symptoms.

Labs : Serum chemistry: Na 132, K 5.0, Cl 104, HCO3- 16 , BUN 25, Cr 1.3, Glu 75 ABG: pH 7.30, PCO2 29, HCO3- 16, PO2 92 Serum albumin 1.0

6years old with Muscle dystrophy & progressive distress ABG-PH-7.30/PaCO275/PaO2-45 & SPO2 80%

1 month old male presents with projectile emesis . ABG - 7.49 / 40 / 98 / 30 Na- 140 / K- 2.9 / Cl- 92 / HCO3- 32.

A 3 year old brought to the ER at 3 am after being found unarousable on his bedroom floor, with urinary incontinence. EMS monitoring at the scene revealed sinus bradycardia. One amp of D50W and 5 mg of naloxone were given IV without response. Vital signs are stable; respiratory effort is regular, but tachypneic. He is acyanotic

Na+=154, K=5.6, Cl=106, HCO3=5, BUN=6 creatinine=1.7, glucose=804, PO4=12.3, Ca++=9.8, NH4=160, serum osms=517
PH=6.80, PaCO2=33, PaO2=298.

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