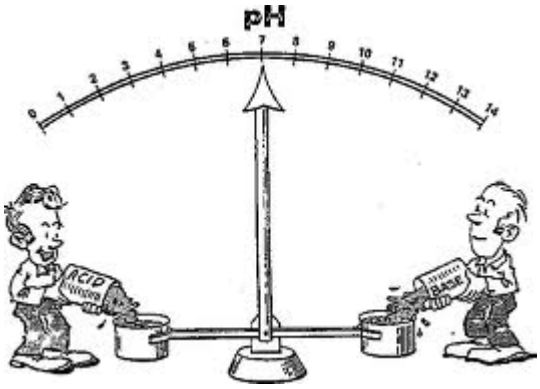

Acid Base Disorders

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New Edition Of

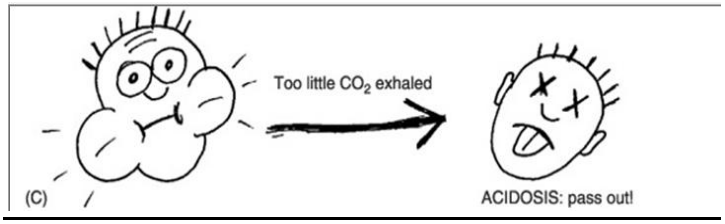
Done by

Medicine Team
429

Introduction

- **pH is maintained by two organs : Lungs (will take care of CO_2) and kidneys (by excretion or reabsorption of HCO_3^- and H^+ - take few days)**
- **Acid can come from inside or outside the body (mostly outside), balance mechanism will work to balance pH through bicarbonate (HCO_3^-), If the acid is too high and bicarbonate is too high the body will hyperventilate also to wash out the pCO_2 (compensatory mechanism)**

Respiratory Acidosis



Primary mechanism:

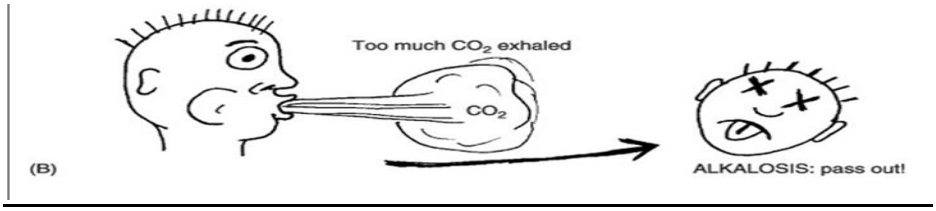
Any cause of Hypoventilation will lead to high pCO_2 (respiratory acidosis), like:

- **CNS (brain stem) :ex. brain stem stroke involves the respiratory center – Trauma – Tumor – medication (sedative drugs like benzodiazepine and lorazepam)**
- **Peripheral nerve : ex. Demylinating disease (Guillain Berre syndrome)**
- **Neuromuscular junction : ex. myasthenia gravis**
- **Chest wall : muscular or bone deformity (ex. kyphoscoliosis)**
- **Also there is a respiratory tree related cause of acidosis (not due to hypoventilation): Ex. restriction (COPD – Bronchial Asthma) - severe pneumonia - pneumothorax**

Chronic respiratory Acidosis:

- **Due to chronic causes like (CODP- Neuromuscular disease- Extreme obesity- Chest wall deformity)**
- **Hall mark : high pCO_2 – high HCO_3^- - pH in normal range (due to compensatory mechanism of HCO_3^-)**
- **NOTE: In Acute respiratory acidosis HCO3- is not high and pH will be low**
  In acute of top of chronic : pCO2 will be very high – pH will be low
- **Renal mechanisms increase the excretion of H^+ within 24 hours and may correct the resulting acidosis caused by chronic retention of CO_2 to a certain extent**

Respiratory Alkalosis



Primary mechanism:

Caused by hyperventilation so pCO_2 will go down and pH will go up and bicarbonate will decrease as a compensatory mechanism

Causes of hyperventilation:

- **Stroke if it affected the respiratory inhibitory center**
- **Drugs: Ex. Aspirin (NOTE: Aspirin has two effects: it causes hyperventilation and causes acidosis in overdose as it will be explained later)**
- **Sepsis**
- **Fever**
- **Thyrotoxicosis**
- **Pregnancy**
- **Hepatic failure**
- **Anxiety (treated by breathing in a bag)**
- **Hypoxemia (patient will hyperventilate and will develop alkalosis)**
- **Restrictive lung disease (Early phase of asthma)**
- **Severe congestive heart failure**
- **Pulmonary emboli (will cause hypoxia >> hyperventilation)**

Metabolic Acidosis

Primary mechanism:

Due to gaining of acid from inside (Ex. lactic acid) or outside the body (Ex. alcohol) or by loosing bicarbonate (HCO_3^-), Patient will have low pH and bicarbonate and low pCO_2 (due to hyperventilation as a compensatory mechanism)

Causes:

- **Increase acid intake:**

- **Alcohol**
- **Ethanol**
- **Aspirin overdose (aspirin is metabolized to salicylic acid) – NOTE: Aspirin also may lead to respiratory alkalosis as it has hyperventilation stimulatory effect**

- **Increase acid production:**

- **Ketone (Diabetic patient – Starvation)**
- **Lactic acid (Hypoxia)**
- **High creatinine**

- **Decrease acid excretion**

- **Sever renal failure**
- **Renal tubular acidosis (RTA)**

- **Loss of bicarbonate**

- **Either from kidney or GIT, ex. Diarrhea**

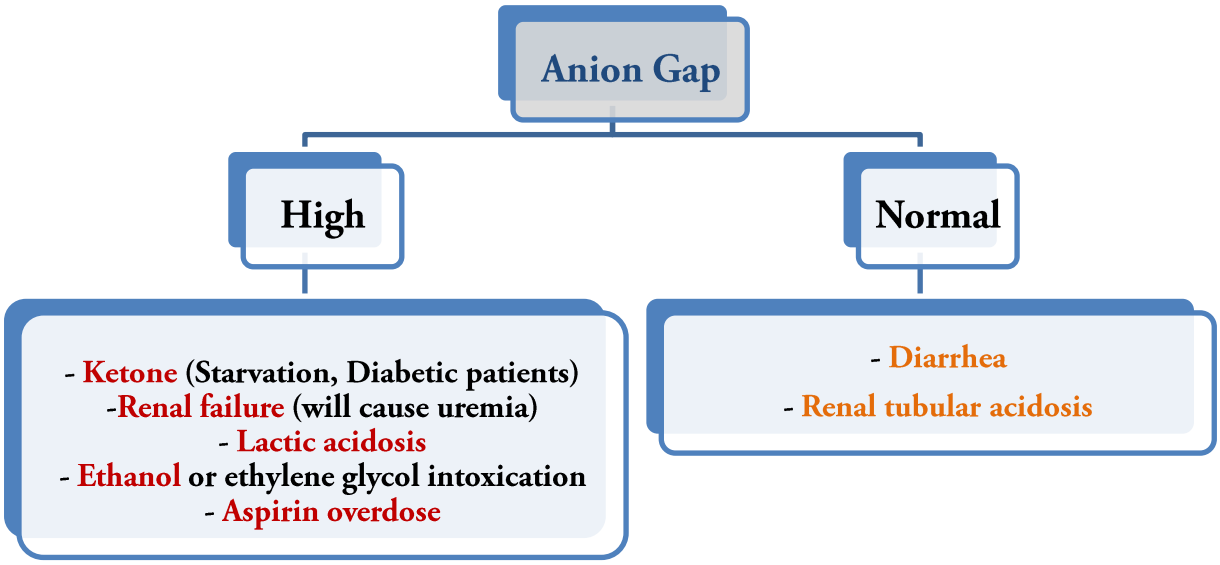
Anion Gap:

To exclude some possible causes of metabolic acidosis we calculate the anion gap :

- **It is the difference between the positive (cation) and the negative (Anion) charged particles in blood or plasma or urine**
- **Normally it is positive (8 - 12)**
- **In high anion gap : there is either increase in cation (positive charge) or decrease in Anion (negative charge)**
- **Anion gap= Cations - Anions**

  AG = $[Na^+]$ - ($[Cl^-]$ + $[HCO3^-]$) OR AG = ($[Na^+]$ + $[K^+]$) - ($[Cl^-]$ + $[HCO3^-]$)

- **Increasing AG is due to bicarbonate loss in buffering (HCO_3^- will buffer the acidity so it will not appear as a Anion in the blood)**
- **In normal AG there is still loss of bicarbonate in but chloride will be reabsorbed so we will have a net of normal anion gap**



Metabolic Alkalosis

Primary mechanism:

By gaining bicarbonate (HCO_3^-) or by loosing acids, patient will present with high pH and bicarbonate and high pCO_2 (as a compensatory mechanism)

Dehydrated patient will reabsorb bicarbonate and develop metabolic alkalosis (Dehydration is a very common cause of metabolic alkalosis)

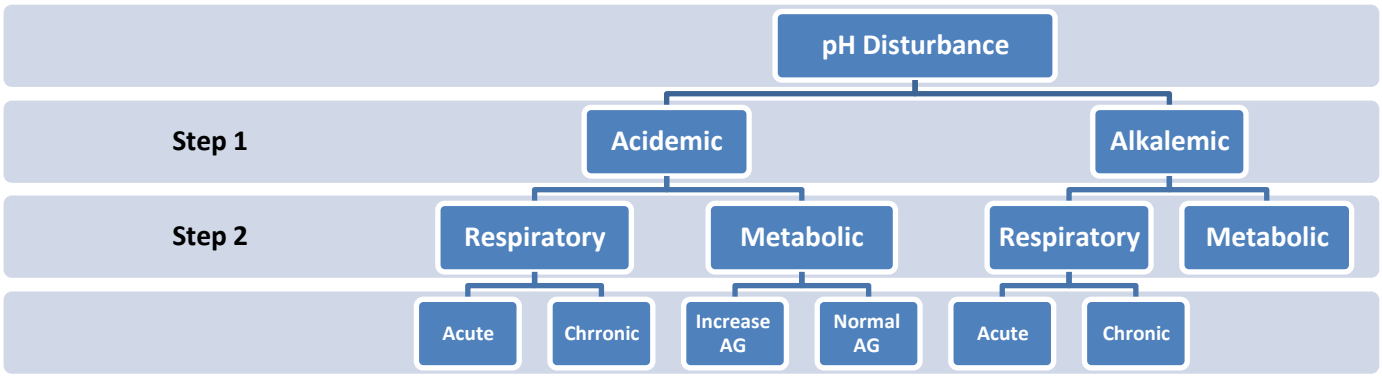
Etiology: (Saline Responsive or Saline Resistant)

Etiologies of Metabolic Alkalosis		
Catagory	**Saline Responsive** GI loss of H^+ and increase bicarbonate concentration due to volume loss	**Saline Resistant**
Seen in	- Dehydration - Vomiting - Diuretic usage - Nasogastric tube - Post-hypercapina	- Hypertensive (mineralocorticoid excess) - Hyperaldosteronism - Severe hypokalemia (remember K+/H+ exchange in kidney) - Exogenous alkali load
Urine CL^-	- Urine chloride will be less than 20	- Urine chloride will be more than 20

- **Ex. Patient with heart failure and he is on a long duration diuretic, what most likely he will develop?**

  Metabolic alkalosis

Acid-Base Analysis



Step 1: Acidemic or Alkalemic?

- **The pH of the arterial blood gas measurement identifies the disorder as alkalemic or acidemic**
- **Normal arterial blood pH = 7.35 – 7.45**
- **Acidemic: pH < 7.35**
- **Alkalemic: pH > 7.45**

Step 2: Is the primary disturbance respiratory or metabolic?

- **To determine the source of the disturbance we look at :**
  - **The arterial P_aCO_2**
  - **The serum HCO_3^-**
- **Respiratory disturbances alter the arterial P_aCO_2 (normal value 35-45)**
- **Metabolic disturbances alter the serum HCO_3^- (normal value 22-26)**

Primary Disorder	Problem	pH	HCO_3	$PaCO_2$
Metabolic acidosis	gain of H^+ or loss of HCO_3^-	↓	↓ (Primary Change)	↓ (To compensate)
Metabolic alkalosis	gain of HCO_3^- or loss of H^+	↑	↑ (Primary Change)	↑ (To compensate)
Respiratory acidosis	hypoventilation	↓	↑ (To compensate)	↑ (Primary Change)
Respiratory alkalosis	hyperventilation	↑	↓ (To compensate)	↓ (Primary Change)

Step 3: Is the respiratory disturbance acute or chronic?

- **Acute respiratory acidosis**: HCO_3^- increases by 1 mEq/l for every 10 mmHg increase in $PaCO_2$
- **Chronic respiratory acidosis**: HCO_3^- increases by 3-3.5 mEq/l for every 10 mmHg increase in $PaCO_2$
- **Acute respiratory alkalosis**: HCO_3^- decreases by 2 mEq/l for every 10 mmHg decrease in $PaCO_2$
- **Chronic respiratory alkalosis**: HCO_3^- decreases by 4-5 mEq/l for every 10 mmHg decrease in $PaCO_2$

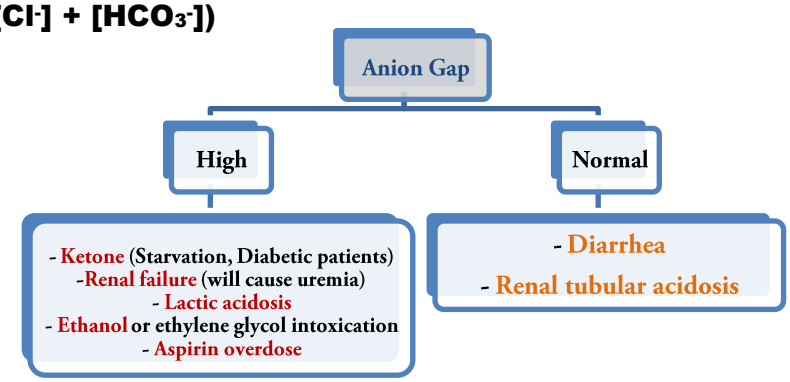
- **Ex. Patient with pH = 7, $PaCO_2$ = 70 what value of HCO_3^- do you expect ?**
  (We consider the normal values of $PaCO_2$ as 40 and HCO_3^- as 24)
  This patient has 30 increase in his $PaCO_2$ .. so we expect the bicarbonate to go up by 3 to be 27

  - If the patient has a HCO_3^- of 30 (high) so he has also metabolic alkalosis with his respiratory acidosis (we don't consider it as chronic respiratory acidosis in this case because his pH is still low)
  - If the patient has a HCO_3^- is 20 (too low) so he has also metabolic acidosis with his respiratory acidosis

Step 4: For metabolic acidosis, is there an increased anion gap ?

- Anion gap = $[Na^+] - ([Cl^-] + [HCO_3^-])$
- Normal AG 8-16

Remember:



Step 5: Are there other metabolic processes present in a patient with an increased anion gap metabolic acidosis?

Step 6: Is the respiratory system compensating adequately for a metabolic disturbance?

Short summery for Acid-Base analysis

Steps in Acid-Base Analysis:

- **First acidosis or an alkalosis?**
  - **Look at the pH**
- **Second what is the primary problem (metabolic or respiratory)?**
  - **Look at the pCO_2**
  - **If the pCO_2 change is in the opposite direction of the pH change, the primary problem is respiratory**
- **Third is there any compensation?**
  - **For a primary respiratory problem, is there a change in HCO_3^-?**
    - **if no, then there is no metabolic compensation**
    - **if yes, then there is either partial compensation or a metabolic problem**
- **Fourth calculate the compensation in a case of a respiratory problem or calculate the anion gap in a case of metabolic acidosis**

Normal values :

- **Normal arterial blood pH = 7.35 – 7.45**
- **P_aCO_2 = 35-45**
- **Serum HCO_3^-= 22-26**
- **Anion gap = 8-12**

Give an interpretation:

	pH	pCO2	HCO3	Interpretation
1	7.41	40	24	
2	7.5	42	35	
3	6.72	40	5	
4	7.26	63	25	
5	7.52	18	25	

Answers: 1- Normal
2- Metabolic alkalosis (dehydration is the most common)
3-Metabolic acidosis
4- Respiratory acidosis
5- Respiratory alkalosis

CASE STUDY-1

- **pH =7.2 pCO2 = 60 HCO_3^- = 24**
  pH = 7.2 indicates acidosis uncompensated
  pCO_2 = 60 = respiratory
  CO_2 = 24 = respiratory, uncompensated

- **What is the primary cause of the condition?**
  Respiratory acidosis is caused when carbon dioxide is not removed from the blood due to either:
  - **Hypoventilation**
  - **Obstructions in the exchange of gases, such conditions are caused by asthma, COPD, pneumonia and pulmonary edema**

- **Is it acute or chronic?**
  Note that the pH is abnormal
  Note the HCO_3^- is within normal (no compensation started)
  So it is Acute

- **Remember:**
  - **Acute respiratory acidosis: HCO_3^- increase by 1 mEq/l for every 10 mmHg increase in $PaCO_2$**
  - **Chronic respiratory acidosis: HCO_3^- increase by 3-3.5 mEq/l for every 10 mmHg increase in $PaCO_2$**

CASE STUDY-2

- **pH =7.1 pCO2 = 42 HCO_3^-= 12**
  pH =7.1 = acidosis, uncompensated
  HCO_3- = 12 = metabolic
  pCO_2 = 42 = uncompensated (it should go down to compensate)

CASE STUDY-3

- **What do you expect the ABG in the following patients to be:**
  24 years old male with acute SOB, and wheezes for 2days and has a history of a bronchial asthma?
  He may have Respiratory alkalosis or acidosis or normal depending on the severity
  (Asthma patient start with alkalosis "due hyperventilation" then will be normal "when his respiratory muscles starts to get tired" then acidosis finally)

- **67 years old women, HTN,DMII, COPD presenting with cough and SOB?**
  Acute Respiratory acidosis

CASE STUDY-4

- **pH: 7.25 HCO3-: 20 mEq/L PaCO2: 52 mmHg**
  What is the primary problem? Compensation? Differential diagnosis?
  Respiratory acidosis – primary cause
  And metabolic acidosis (As HCO_3^- should increase from normal "24" by 1.2 to be 25.2 but here it is too low)

CASE STUDY-5

- **56 years old male with Hx of COPD is admitted with 1-wk Hx of dyspnea, productive cough and diarrhea**
  (Na) 125, (CL) 103 , (BUN) 42, (Glucose) 100, (K) 3.5, (HCO_3-) 10, (Creat) 1.4
  ABG= 7.14 pCO2= 30 pO2= 50
  What is the predominant acid base disorder? diffrential diagnosis?
  Compensation ?

Metabolic acidosis
Anion gap =(125+3.5)-(103+10)=15.5 "normal"
So it may by diarrhea (most likely upon history - treated IV fluid) or renal tubular acidosis

Compensation: expected pCO_2 = 40 – (1.2 * (24-10)) = 23.2 this is not full compensation because pCO_2 is 30 and this indicates an underlying respiratory acidosis, suggested by the Hx of COPD, dyspnea, and productive cough (lungs are not able to appropriately compensate)

CASE STUDY-6

- **32 years old male present with 2 days history of intractable vomiting**
- **pH= 7.51 pCO_2= 41 HCO_3^-= 32**
  Na=132 CL= 90 K=3.4 creatinine=1.6
  What is the predominant acid-base disorder?
  Metabolic alkalosis
- **What pCO_2 is expected with normal respiratory compensation?**
  40 + {(32 – 24) * (~ 0.6 ⇔ 0.7)} = 44.8 ⇔ 45.6 mmHg; since the measured pCO2 < 44.8 ⇔ 45.6 there is also a primary respiratory alkalosis (inappropriate hyperventilation)
- **What is the appropriate treatment?**
  Isotonic saline to correct for volume depletion

CASE STUDY-7

- **A 58- year old man presents to the Emergency Department with abdominal pain and hypotension. Investigation reveal the following:**
  Na= 140 K= 4 CL^-= 90 HCO_3^- = 5 pH= 6.8 PCO_2=36
  PO_2= 7
  Analyze the acid-base disorder seen in the patient and differential diagnosis.
  Patient has metabolic acidosis
  Anion gap= (140+4)-(90+5)=49 "high anion gap"
  Differential diagnosis: Renal failure, lactic acidosis , ketoacidosis, alcohol or aspirin overdose
  He most likely has bowel ischemia (as his age is 58!)

Done