"He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all"
William Osler

Medicine

430

Acid Base Balance

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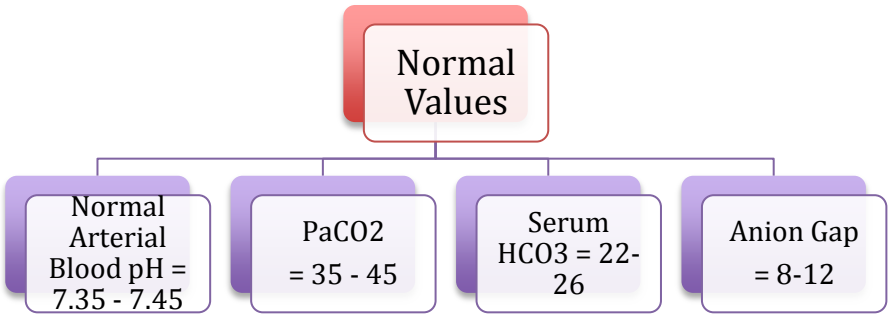
Acid base balance



Primary Disorders:

Primary Disorder	Problem	pH	HCO3	PaCO2
Metabolic Acidosis	Gain of H+ or loss of HCO3	Decreased	Decreased	Decreased
Metabolic Alkalosis	Gain of HCO3 or loss of H+	Increased	Increased	Increased
Respiratory Acidosis	Hypoventilation	Decreased	Increased	Increased
Respiratory Alkalosis	Hyperventilation	Increased	Decreased	Decreased

pH Decrease = Acidemia = Acidosis
pH Increase = Alkalemia = Alkalosis
When the increase/decrease is in PaCO2 → it's Respiratory and the change in HCO3 is Compensatory
When the increase/decrease is in HCO3 → it's Metabolic and the change in PaCO2 is Compensatory

Respiratory acidosis:

- Primary mechanism: <u>Hypoventilation</u>
- CNS (Damaged respiratory center → Trauma, Stroke, Ischemia, Hemorrhage…etc)
- Peripheral nerve (Guillain-Barré Syndrome)
- Neuro-muscular junction (Myesthenia Gravis)
- Chest wall (Congenital Muscular Dystrophy)
- Bronchial tree (COPD)

Causes of acute respiratory acidosis:

- Respiratory pathophysiology
  - Airway obstruction
  - Severe pneumonia
  - Chest trauma
  - Pneumothorax
- Acute drug intoxication (narcotics, sedatives)
- Residual neuromuscular blockade
- CNS disease (head trauma)

Chronic Respiratory Acidosis

- $PaCO_2$ is elevated with a pH in the acceptable range
- 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

Causes of chronic Respiratory Acidosis

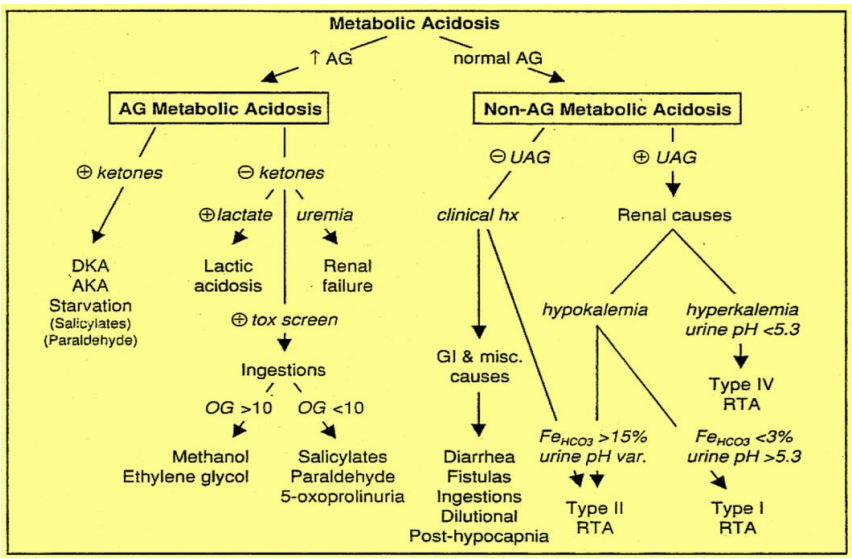
- Chronic lung disease (COPD)
- Neuromuscular disease
- Extreme obesity
- Chest wall deformity

Respiratory alkalosis (Hyperventilation)

- Pain
- Drugs
- Sepsis
- Fever
- Thyrotoxicosis
- Pregnancy
- Overaggressive mechanical ventilation
- Hepatic failure
- Anxiety
- Hypoxemia
- Restrictive lung disease
- Severe congestive heart failure
- Pulmonary emboli

Metabolic acidosis

- Increase acid production
- Decrease acid excretion
- Loss of bicarbonate



Metabolic Acidosis

Anion gap = Cations – Anions
(Positives – Negatives)

- Anion gap = [Sodium] - ([Chloride] + [Bicarbonate]) **Or** AG = $[Na^+]$ - ($[Cl^-]$ + $[HCO_3^-]$).
- Anion gap = ($[Na^+]$ + $[K^+]$) - ($[Cl^-]$ + $[HCO_3^-]$)

Increased anion gap metabolic acidosis

- Methanol other alcohols, and ethylene glycol intoxication
- Uremia (renal failure)
- Lactic acidosis
- Ethanol
- Paraldehyde and other drugs
- Aspirin
- Ketones (starvation, alcoholic and diabetic ketoacidosis)

The Anion Gap can guide you towards the cause of Metabolic Acidosis.

- High AG
  - Indicates an added acid, either by ingestion or a systemic cause.
- Normal AG:
  - Indicates loss of HCO3 due to a secondary problem. (Diarrhea, RTA)
  - Because it is a problem in the body itself (not an added cause), there is absorption of **Chloride** which leads to balancing the HCO3 loss → causing a normal AG.

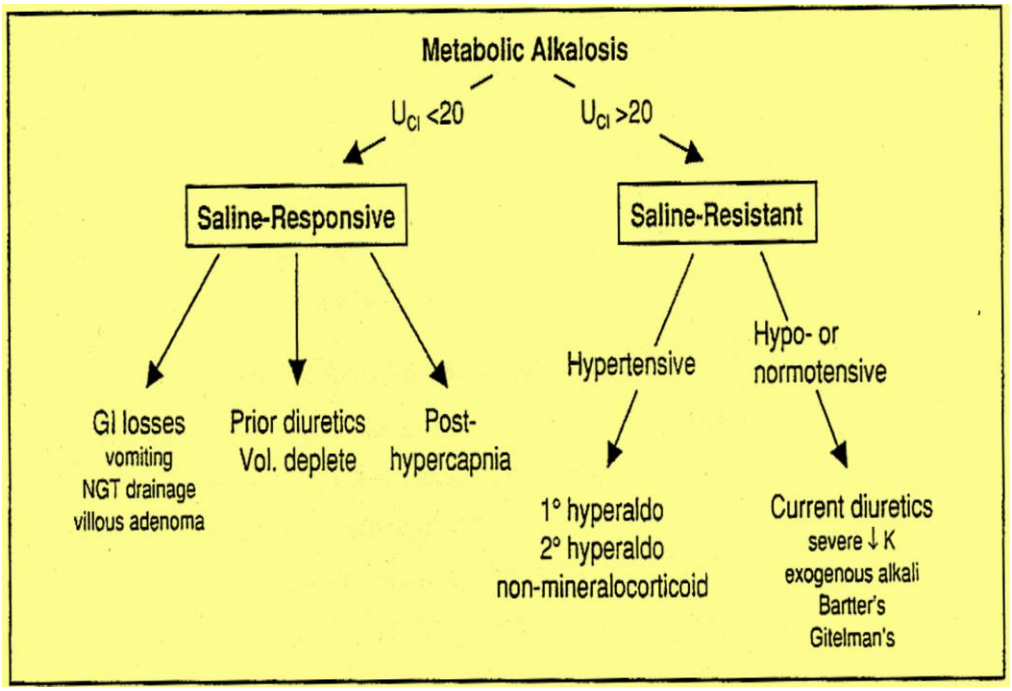
Etiologies of anion gap metabolic acidosis:

Ketoacidosis	• Diabetes mellitus**, alcoholism, starvation**
Lactic Acidosis	• Type A**: impairment in tissue oxygenation, eg.** Circulatory or Respiratory failure, sepsis, **ischemic bowel, carbon monoxide** • Type B: **no impairment in tissue oxygenation, eg. Malignancy, alcoholism, meds (metformin, NRTIs, salicylates)** • D-lactic acidosis: **short bowel syndrome glc metabolized by colonic bacteria** • **To D-lactate, which is absorbed; not detected by standard lactate assay**
Renal Failure	• **Accumulation of organic anions such as phosphates, sulfates, etc.**
Ingestions	• Methanol: **manifestations include blurred vision** • Ethylene glycol: **manifestations include ΔMS, cardiopulmonary failure, calcium oxalate crystals and renal failure** • Paraldehyde • Salicylates: **metabolic acidosis (from lactate, ketones) + respiratory alkalosis due to stimulation of CNS respiratory center** • Acetaminophen: **glutathione depletion, accumulation of the Endogenous organic acid 5-oxoproline in susceptible host**

Etiologies of non-anion gap metabolic acidosis:

GI losses of HCO_3	• **Diarrhea, intestinal or pancreatic fistulas or drainage**
RTAs	• **See section on renal tubular acidosis below**
Early renal failure	• **Impaired generation of ammonia**
Ingestions	• **Acetazolamide, sevelamer, cholestyramine, toluene**
Dilutional	• **Due to rapid infusion of bicarbonate-free intravenous fluids**
Post-hypocapnia	• **Respiratory alkalosis** • **Renal wasting of HCO_3** • **Rapid correction of resp. alk.** • **Transient acidosis until HCO_3; regenerated**
Ureteral diversion	• **Colonic Cl^-/HCO_3^- exchange, ammonium reabsorption**

Metabolic alkalosis



Etiologies of Metabolic Alkalosis:

Saline – responsive	• GI loss of H^+ : **vomiting, NGT drainage, villous adenoma** • Diuretic use • **posthypercapina**

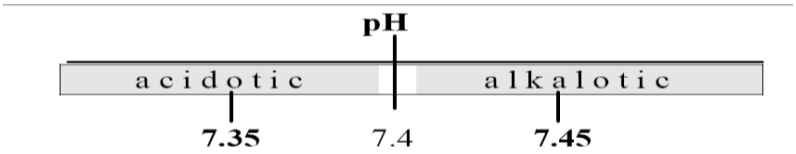
Saline – resistant	• **Hypertensive** (mineralocorticoid excess)
	  ○ **1^0 hyperaldosteronism (eg. Conn's)**
	  ○ **2^0 hyperaldosteronism (eg, renovascular dis. Rennin-secreting tumor)**
	  ○ **Non-aldo (eg. Cushing's, Liddle's, exogenousmineralocorticoids)**
	• **Normotensive**
	  ○ **Severe hypokalemia**
	  ○ **Exogenous alkali load**
	  ○ **Bartter's syndrome, Gitelman's syndrome**

Steps in Acid-Base Analysis

- **Step 1:** • Acidemic or Alkalemic?
- **Step 2:** • Is the primary disturbance respiratory or metabolic?
- **Step 3:** • Is the respiratory disturbance acute or chronic?
- **Step 4:** • For a metabolic acidosis, is there an increased anion gap?
- **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?

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 whether the disturbance affects primarily

- 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)

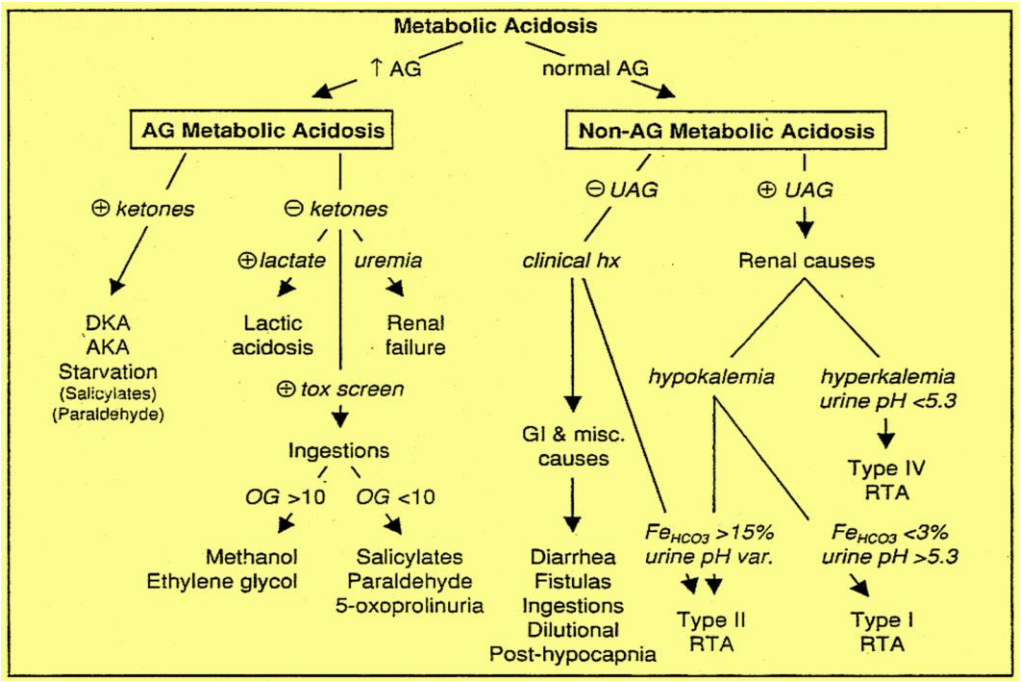
Step 3: Is the respiratory disturbance acute or chronic?

- Acute respiratory acidosis:
  - HCO_3^- increase by 1 mEq/l for every 10 mmHg increase in PaCO2
- Chronic respiratory acidosis:
  - HCO_3^- increase by 3-3.5 mEq/l for every 10 mmHg increase in PaCO2
- Acute respiratory alkalosis:
  - HCO_3^- decrease by 2 mEq/l for every 10 mmHg decrease in PaCO2
- Chronic respiratory alkalosis:
  - HCO_3^- decrease by 4-5 mEq/l for every 10 mmHg decrease in PaCO2

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

Anion gap = [Sodium] - ([Chloride] + [Bicarbonate]) Or
AG = [Na+] - ([Cl-] + [HCO3-]).
Normal AG 8-16

Serum Osmolality = (2 x (Na + K)) + (BUN) + (glucose)



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

- Metabolic acidosis:
  - PCO2 decreases by 1 mmHg for every 1 mEq/l decrease in HCO3
- Metabolic alkalosis:
  - PCO2 increases by 0.6 mmHg for every 1 mEq/l increases in HCO3

Cases

Case 1:

PH =7.2, pCO2 = 60, HCO3 = 24

Solution:

What it is the primary problem? Compensation?

PH (▼) = acidosis,

PCO2= (▲) → PH is in the opposite direction of PCO2 so it's **respiratory.**

Since there isn't any chronic lung disease then it's **acute Respiratory acidosis** (hypoventilation)

In respiratory acidosis, the CO2 is increased while the bicarbonate is either normal (uncompensated) or increased (compensated).

Now, we calculate the compensation by calculating the increases in PCO2 with increase in HCO3→ let's say that normal PCO2 is 40 and here it's 60 so the amount of increases is (20) so HCO3 should increase by 20 , here we have HCO3 (24) and the normal is 22-26 → it didn't increase so there's no compensation.

So the main problem is **Uncompensated Acute Respiratory Acidosis**

Differential diagnosis?

Airway obstruction, severe pneumonia, chest trauma/pneumothorax

Acute drug intoxication (narcotics, sedatives)

Residual neuromuscular blockade

CNS disease (head trauma)

Treatment?

Bronchodilator drugs to reverse some types of airway obstruction

Noninvasive positive-pressure ventilation (sometimes called CPAP or BiPAP) or mechanical ventilation if needed

Oxygen if the blood oxygen level is low

Treatment to stop smoking

Treatment:
http://www.umm.edu/ency/article/000092trt.htm#ixzz2CPg1BAmj

Case 2:

What do you expect the Arterial Blood Gas (ABG) in the following patients to be?

1-24 years old male with acute SOB (a dyspneic condition), and wheezes for 2days with past medical history of bronchial asthma.

Solution:

A patient like this can present with one of two problems. Initially he would have respiratory alkalosis due to hyperventilation, if left untreated he would then shift from respiratory alkalosis to respiratory acidosis due to hypoventilation (Chest muscles become exhausted) which one he presents with would depend on the time he's seen in.

The presentation in the case above is acute respiratory acidosis based on the numbers given

2-67 years old women, HTN,DMII, COPD presenting with cough and SOB

Solution:

Most probably it's Chronic respiratory acidosis (we said chronic due to chronic lung diseases), so ABG will be as follows: paCO2 is elevated with a pH in the acceptable range (PH is normal) due to long time compensation.

Renal mechanisms increase the excretion of H+ within 24 hours and may correct the resulting acidosis caused by chronic retention of CO2 to a certain extent.

Case 3:

PH: 7.25 (▼)
PaCO2: 52 mmHg (▲)
HCO3-: 20 mEq/L (▼)

Solution:

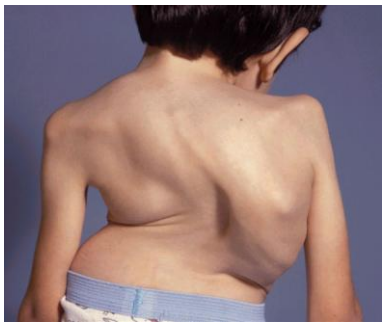
What it is the primary problem? Compensation?
1- Acidosis
2- Respiratory (Main Problem)
3- Not compensated
4-Metabolic Acidosis (Secondary Problem)

Differential diagnosis?

Airway obstruction, severe pneumonia, chest trauma/pneumothorax
Acute drug intoxication (narcotics, sedatives)
Residual neuromuscular blockade
CNS disease (head trauma)

Case 4:

PH: 7.32 (▼),PaCO2: 55 mmHg (▲), HCO3-: 19 mEq/L (▼).



Solution:

What it is the primary problem? Compensation?
1- Acidosis
2- Because he has kyphoscoliosis it is respiratory is the main problem.
3- There is metabolic acidosis on top of respiratory.
Chronic Respiratory acidosis due to Chest wall deformity – Severity of acid/bas problem depends on the severity of the deformity.

Differential diagnosis?

What other investigation you want to do?

Investigations:
http://orthoanswer.org/spine-neck/kyphosis/investigations.html

Case 5:

56 years old Man with History of COPD is admitted with 1-wk History of dyspnea, productive cough and diarrhea
(Na) 125, (Cl) 103 , (BUN) 42, (Glucose) 100, (K) 3.5, (Creat) 1.4
ABG: PH= 7.14
pCO2 30,
pO2 50,
(HCO3-) 10

Solution:

What is the predominant acid base disorder?
PH (▼) PCO2 (▼)
Metabolic acidosis (the main problem)
Calculate the AG?
125 – (103+10) = 12 → normal
AG; etiology is either diarrhea or RTA – most likely diarrhea because of the history

What pCO2 is expected with normal respiratory compensation ?
=40 – (22-10) = 28

This is not full compensation b/c pCO2 is 30 – indicates an underlying primary **respiratory acidosis**, suggested by the Hx of COPD, dyspnea, and productive cough (lungs not able to appropriately compensate) .. **The primary problrm is Metabolic acidosis with respiratory acidosis**

Case 6:

32 years old male presented with 2 days Hx of intractable vomiting.
pH 7.51
pCO2 41
HCo2 33
Na132, Cl 90 32, K3.4, creatinine1.6

Solution:

PH (▲), PCO2 (normal)
What is the predominant acid-base disorder?
Metabolic alkalosis (the main problem)

What pCO2 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 Treatment? Isotonic saline to correct for volume depletion.

Case 7:

A 58- year old man presents to the Emergency Department with abdominal pain and hypotension.
Investigation reveal the following:

PH 6.8,
PCO2 36,
HCO3 5
Na (140), K(4,)Cl (90), PO2(7)

Solution:

PH (▼) PCO2 (normal) HCO3 (▼)
Analyze the acid-base disorder(s) seen in the patient?
Metabolic acidosis (the main problem)

Calculate AG?

140-(90+5)= 45→ high anonion gap
There are 3 DDx for AG metabolic acidosis: DKA or AKA, lactic acidosis and renal failure.
Because hes's old it's unlikly to be DKA or AKA and we know nothing about his kidney the it's also unlikly to be renal failure.
So Most likly he has **lactic acidosis** (due to hypotention)
but we must ask for alcohol consumption.

What's the treatment? IV fluids to restore Bb then lactic acid production will stop.